Volume-01
Number-02
July-2018
ISSN: 2616-6178

The BEACON Medical Journal

Journal of Current Medical Practice

Original Article

Clinico-pathological Evaluation of Acute Confusional State Among Elderly Patients

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ABSTRACT

Background: *Acute confusional states are most common problems in general medicine. They account for a substantial proportion of admissions to emergency wards and are a frequent cause of distress in all hospital services. It is an acute or subacute brain failure in which impairment of attention is accompanied by abnormalities of perception and mood. During the acute phase, thought and speech are incoherent, memory is impaired and misperceptions occur. Aetiology of acute confusional state are numerous and presentations are also variable, so many time create confusion regarding the actual diagnosis and thus delay the prompt management which may result in fatal outcome.*

Objectives: *Purpose of this study was to find out the causes and short-term outcome of acute confusional state among elderly patients.*

Materials & method: *A descriptive type of cross-sectional study was conducted in a tertiary care hospital amongst seventy eight patients of acute confusional state from March 2016 onwards. Sample was selected by purposive sampling technique. All patients were in acute confusional state, age above 50 years (diagnosed on the basis of clinical parameter and supportive laboratory profile). This included persons residing in urban and semi-urban areas, as well as persons transferred from hospitals in rural areas.*

Result: *Maximum numbers of patients (48.71%) were between 50-59 years age group, mean age of the patient were 78.38 ± 11.23 years. Male & female ratio was 1.9:1. Common aetiology for acute confusional state patients were cerebrovascular disease (41.02%), followed by meningitis (11.53%), electrolyte imbalance (14.10%), and pneumonia (7.69%). Electrolyte imbalance commonly associated with other major physical illness e.g severe diarrhoea, advance cancer, renal failure. According to evaluation by Modified Rankin Scale (MRS), 73% of the patients recovered without sequele, 23% recovery with sequele and three cases (4%) expired during hospital stay (MRS score 6).*

Conclusion: *Patients who develop acute confusional state have high disability, complication rates, and have longer lengths of stay than other patients. Knowing the nature and timing of disease, together with the identification of high-risk patients, may be useful to those planning ACS services.*

Key words: *Acute confusional state, Stroke.*

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Introduction:

Acute confusional state (ACS) is a common accompanied physical illness particularly in old age that may complicate different type of disorders. It is associated with significant morbidity & mortality. It is an acute or subacute brain failure in which impairment of attention is accompanied by abnormalities of perception and mood. During the acute phase, thought and speech are incoherent, memory is impaired and misperceptions occur. The prevalence in hospital ranges from 10–20% in medicine wards and could become higher as the elderly population in hospital increases. About 25% of people over 70 years old admitted to hospital have delirium[1].

Confusion and delirium always signify a disorder of the nervous system. They may be the major manifestation of a head injury; a seizure; drug toxicity (or drug withdrawal); a metabolic disorder resulting from hepatic, renal, pulmonary or cardiac failure; a systemic infection; meningitis or encephalitis[2].

In inflammatory disease, infection, trauma, or a surgical procedure in brain, a systemic inflammatory response leads to the increased production of cytokines. Aside from this harmful effect on neurons, cytokines can also impair the synthesis and release of neurotransmitters. It appears that inflammatory processes play a role in causing acute confusional state with simultaneous febrile illness[3]. Another hypothesis that stress, factors that induce the sympathetic nervous system to release more noradrenaline, and the hypothalamic-pituitary-adrenocortical axis to release more glucocorticoids, can also activate glia and thereby damage neurons[4].

Elderly patients more commonly present with atypical or non-specific symptoms of UTI, and this may contribute to delayed diagnosis and treatment. They may have much more vague presentations such as an acute confusional state[5]. Other common aetiology are pneumonia, hepatic encephalopathy, electrolyte imbalance. Hyponatraemia due to water intoxication

the possible need for rescue PCI[8].

Resolution of ST elevation represents reperfusion. In patients receiving fibrinolysis, this reflects both epicardial recanalization and tissue reperfusion. With primary PCI and angiographically documented restored epicardial flow, resolution of ST elevation serves to differentiate those with from those without tissue reperfusion[2].

Studies using ST segment resolution demonstrated that patients with rapid ST resolution had smaller infarcts than those with persistent ST elevation7. Schroder, et al. has demonstrated a strong, stepwise correlation between the degree of ST resolution at 180 min and subsequent mortality. They found that patients who develop complete ST resolution by 60 min were at lower risk for death and heart failure than those who develop complete ST resolution by 90 min[9].

Several studies demonstrated that ST resolution, not TIMI flow grade, was an independent predictor of mortality and CHF. These studies support the hypothesis that ST resolution is a surrogate for tissue-level reperfusion. When "complete" ST resolution is seen 90 min after fibrinolysis, successful reperfusion has occurred at both the epicardial and microvascular level, and the prognosis is excellent[9].

Methods:

This Observational study was conducted in the Department of Cardiology, National Heart Foundation Hospital and Research Institute, Dhaka, Bangladesh from 22 October 2013 to 21 October 2014. Purposive sampling was done. All the patients of acute ST-segment elevation myocardial infarction admitted in coronary care unit (CCU) of National Heart Foundation Hospital and Research Institute, Dhaka and who fulfill the inclusion and exclusion criteria. Studied of a continuous response variable such as score of ST segment resolution from independent Fibrinolytic and Primary PCI group with equal number of subjects in each group. In a previous study the response within each subject group was normally distributed with standard deviation 0.2. If the true difference between these two group' s mean ST resolution score is 0.15, which we think clinically important difference to detect, we need to study 29 Fibrinolytic group subjects and 29 PPCI subjects to be able to reject the null hypothesis that the population means of the two groups are equal with probability (power) 0.8. The Type I error probability associated with this test of this null hypothesis is 0.05[10].

Group I: Consisted of 33 patients with acute STEMI treated with Primary PCI with complete ST segment resolution.

Group II: Consisted of 33 patients with acute STEMI treated with Fibrinolytic (with streptokinase) therapy with complete ST segment resolution.

Inclusion criteria-

• Patients with acute STEMI who admitted in National Heart Foundation Hospital and Research Institute, Dhaka during the study period.

• For Primary PCI(Group I) after complete ST segment resolution:

Patients with acute STEMI , those who presented within 12 hours of onset of symptoms and were agreed for Primary PCI.

• For Fibrinolysis with Streptokinase (Group II) after complete ST segment resolution.

Patients with acute STEMI those who presented within 12 hours of onset of symptoms, were not agreed for Primary PCI and had no contraindications for Streptokinase (thrombolytic) therapy.

Exclusion criteria:

• Patients age >75 years.

• Patients have any other co-morbid conditions like malignancy, LV dysfunction, CCF, known CKD (S.Creatinine > 2 mg/dl), coagulation or bleeding disorder.

• Patients with pre existing valvular heart disease and acute STEMI.

• Causes of ST-segment elevation in ECG other than acute MI and new onset of bundle branch block.

• Unwilling to participate.

Ethical approval: This study was approved by the ethics committee of National Heart Foundation Hospital and Research Institute, Dhaka,

Result:

This Observational study was conducted in the Department of Cardiology, National Heart Foundation Hospital and Research Institute, Dhaka, Bangladesh. Total 66 patients were studied and they were grouped on the basis of their treatment modality. Group I underwent primary PCI and group II received fibrinolytic therapy as reperfusion therapy for acute ST-segment elevation myocardial infarction (STEMI) with complete ST segment resolution. Comparison of mortality and morbidity benefits after complete ST segment resolution in both groups were studied.

Regarding the aetiology of different cases of acute confusional state, cerebrovascular disease was the commonest cause of ACS present in 41.02% of patients, followed by meningitis (11.53%), electrolyte imbalance (14.10%), and pneumonia (7.69%). On CT scan of brain, findings shows 23 (71.87%) of cases infarctive stroke and 9 (28.12%) hemorrhagic. Electrolyte imbalance commonly associated with other major physical illness which aggravate the condition. In our study we enrolled patients of severe diarrhoea, advanced cancer, end-stage renal failure patients had association of electrolytes imbalance.

Table-I: Distribution and comparison of patients by cardiovascular risk factors (n=66)

Risk factors	Group I (n=33) f (%)	Group II (n=33) f (%)	P value
Smoking			
Current	17(51.5)	14(42.4)	$^{a}0.805^{NS}$
Former	5(15.2)	8(24.2)	
Never	6(18.2)	6(18.2)	
Recent	5(15.2)	5(15.2)	
Hypertension			
Present	23(69.7)	18(54.5)	$^{a}0.205^{NS}$
Absent	10(30.3)	15(45.5)	
Diabetes Mellitus			
Present	19(57.6)	23(69.7)	$^{a}0.306^{NS}$
Absent	14(42.4)	10(30.3)	
Family history of premature CAD			
Present	17(51.5)	11(33.3)	$^{a}0.135^{NS}$
Absent	16(48.5)	22(66.7)	
Dyslipidaemia			
Present	21(63.6)	12(36.4)	$^{a}0.027^{S}$
Absent	12(36.4)	21(63.6)	
Obesity			
Present	6(18.2)	7(21.2)	$^{a}0.757^{NS}$
Absent	27(81.8)	26(78.8)	

3.2 Alternative treatment with other drugs:

a. Corticosteroids[21,22]

Oral corticosteroids, such as prednisolone, should be considered in cases of severe AU, severe serum sickness, urticarial vasculitis, and delayed pressure urticaria that are not responsive to other treatment. In CU patients, prednisolone should not be prescribed regularly or continued for a long period of time. It should be used only in recalcitrant disease or in disease exacerbation for a short period of time.

b. Combination of H_1 and H_2 antihistamines

Treatment using H_2 antihistamines in combination with H_1 -antihistamines has low quality of evidence and its efficacy is still unclear. Combined H_1 and H_2 antihistamine therapy may be considered in some recalcitrant CSU patients who do not respond well to H_1 antihistamines alone[23,24].

c. Leukotriene receptor antagonists

Patients not responding to antihistamines alone should be offered a 1-4 week trial of the addition of a montelukast 4-10 mg once.[25]

d. Ciclosporin

The optimal dose is 2.5-5 mg/kg/day.[26,27] Ciclosporin should not be used for longer than 3-6 months due to its adverse effects. .

e. Omalizumab

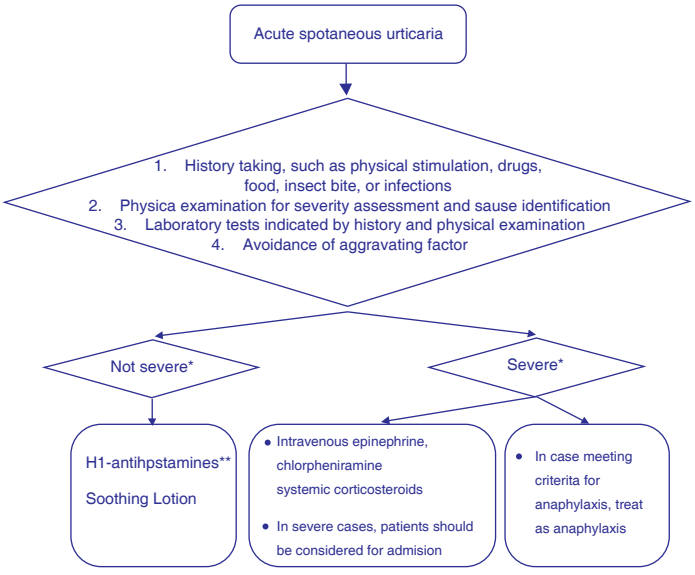
The reported findings from many studies support the effectiveness of omalizumab in CSU patients.[28,29]

4. Other treatment modalities:

4.1 Calamine lotion application.

4.2 Patient education regarding etiology, process of disease, prognosis, and psychosocial support.

Management algorithm for children and adult patients with acute spontaneous urticaria



(Figure 7)

Algorithm for treatment of chronic spontaneous urticaria

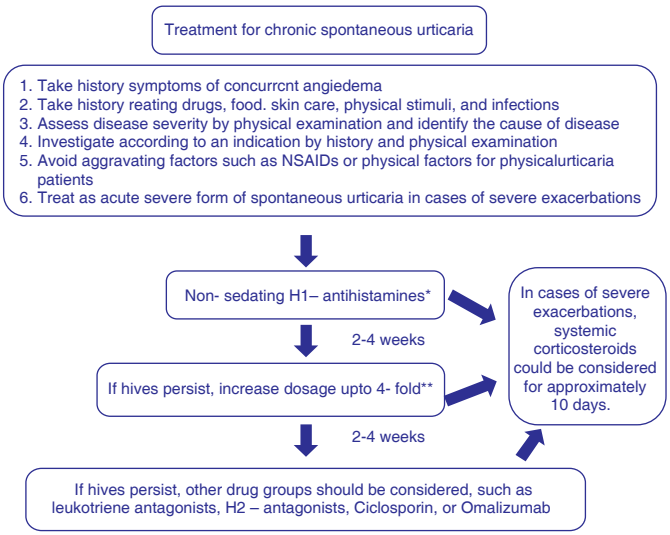


Figure 8: Algorithm-1

Time	Treatment
Week-1	Start second-generation antihistamine (H1 blocker)
Week-3	Titrate second-generation antihistamine to two to four times normal dose [If insufficient control after two weeks]
Week-7	Switch to different second-generation antihistamine or Consider adding one of the following: • H_2 blocker • First-generation antihistamine at night • Leukotriene receptor antagonist • Brief burst of oral corticosteroids (3 to 10 days in tapered dose) [If insufficient control after four weeks]
Week-11	Try another option listed above or Consider referral for second-line therapies such as hydroxychloroquine (Plaquenil) or tacrolimus (Prograf) [If insufficient control after four weeks]

(Algorithm: 2)

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Editor's choice

This is a great pleasure for us that we are going to publish "The Beacon Medical Journal" second issue in July 2018. Next issue will be published in January 2019. The journal will publish 2 issues/-year as regular basis. Ten thousands copy/issue will be distributed to graduate doctors throughout the country by our field colleagues. Already we had form a strong advisory and review board to attract the attention of it's authors and readers nationally and internationally. Editorial of this issue is 'Renal Disorder in Pre-eclampsia' (P-01). At present this is a very commmon problem in Bangladesh. Here pathogenesis of renal disorder in pre-eclampsia, diagnostic procedure, treatment protocol and preventive measures are discussed. Apart from that this issue also contains 6 original articles, 1 review article and 2 case reports.

Your opinion and suggestions will highly encourage us for the development of the journal. The journal is freely available at www.beaconpharma.com.bd for contributing the advancement of public health and medical research.

I do believe this journal will scientifically help doctors in their daily practice.

Dr. G.M. Raihanul Islam
Executive Editor
The Beacon Medical Journal

("diluting delirium") may present with acute confusional states and is seen as paraneoplastic syndrome (due to SIADH), after head trauma, or secondary to drugs (antiepileptics, antidepressants, etc.)[6]. An extrapontine (supratentorial) variant of this syndrome presents with mental changes (including acute confusional states) and movement disorders. Hypernatraemia can lead to seizures and mental changes including acute confusional states[7]. AIDS, leukaemia, lymphoma and other malignant neoplasms cause development of immune suppression and brain metastasis, also causes ACS.

As the etiology, presentations of acute confusional state are variable, appropriate evaluation is mandatory. Acute confusional state is thus associated with high rate of mortality and morbidity, specially if undiagnosed. Therefore an awareness regarding the clinico-pathological situation of acute confusional state is essential for all level of medical practitioners for its early diagnosis, proper treatment and prevention as well.

Methods:

A prospective study was conducted over the period of twelve months from March 2016 to February 2017 in Gazi Medical College Hospital. A total number of 78 cases with acute confusional state, age above 50 years were purposefully collected for conducting the study. Patients were assessed on the basis of the definition of acute confusional state given by the Diagnostic and Statistical Manual, 4th Edition (DSM-IV) of American Psychiatric Association (APA) and level of consciousness was assessed according to Glasgow Coma Scale. It means disturbed consciousness, cognitive function or perception which has an acute onset and fluctuating course. It usually develops in less than 2 weeks. A patient must show each of the following features:[8]

a) Disturbed consciousness with reduced ability to focus, sustain or shift attention.

b) A change in cognition (e.g. memory, language, orientation) or the development of a perceptual disturbance (hallucinations) that is not accounted for by a pre-existing or developing dementia.

c) Development of the disturbance over a short period (hours or days) and a tendency to fluctuate over the course of the day.

d) Evidence from the history, physical examination or laboratory findings that the disturbance is caused by the direct physiological consequences of a general medical condition, or by substance intoxication or withdrawal.

Detailed neurological and systemic examination was done. All the relevant investigations like complete blood count, random blood sugar, blood urea, serum creatinine, serum electrolytes, urine R/E, chest X-Ray were done immediately as per clinical context. Other important investigations like lumbar puncture, CT scan of head, MRI, etc were done where indicated. Patients admitted with some of the following features of acute confusional state in medicine unit in hospital were included in the study -

Inclusion criteria:

1. Patients who clinically present with features of acute confusional state (according to DSM IV) within 2 weeks and have a proper attendant who can give the proper history.
2. Any sex > 50 years.

Exclusion criteria:

1. Patient's attendant who are not interested to participate in this study.
2. Investigations could not be done within 2 days.
3. Patients with preexisting illnesses such as dementia, psychiatric disorders.

Data were obtained from all participants by the use of a predesigned and easily understandable questionnaire. After collection of all information, these data were checked, verified for consistency and edited for finalized result. Data cleaning validation and analysis were performed using the SPSS/PC software and graph and chart by MS Excel. The result was presented in tables, figures, graphs, diagrams & charts etc.

Literature Review:

Acute confusional state called delirium has been described in various ways since ancient times; the Latin word and the related verb delirare ("to be out of one's mind") are said to derive from the figurative expression de lira ire ("to go off the ploughed furrow")[9]. The term is imprecise, as are its more or less synonymous equivalents "acute brain syndrome," "organic brain syndrome," "acute cerebral insufficiency," "disorders of consciousness," "transitional syndrome," and "confusional syndrome."[10]

Common causes include central nervous system infection, cerebrovascular accident, head injury, psychotropic drugs, pulmonary or urinary tract infections, electrolyte disturbances, hypoglycaemia, hypothermia, and alcohol abuse11. Traumatic brain injury is another common neurological causes of ACS. A variety of cognitive and behavioural disturbances including acute confusional states and post traumatic amnesia can be observed in patients with acute traumatic brain injuries[12,13]. The presence of acute confusional states is predictive of a poorer outcome after acute traumatic brain injury[14].

Neurological causes are potential causes of an acute confusional state. An acute confusional state is found in about 10–20% of stroke patients. Both hypovigilant/hypoactive and hypervigilant/hyperactive forms of acute confusional states are possible. In some patients hallucinations, behavioral changes, neuropsychiatric symptoms may additionally be observed[15,16]. Associated metabolic disturbances, fever, sleep-wake disturbances, emotional distress and epileptic seizures may also play a role. Metabolic/septic encephalopathies are probably the most common cause of acute confusional states. Hypoglycaemia and hyperglycaemia causes metabolic encephalopathies. Hypoglycaemia can arise in the fasting state or as a reactive phenomenon (after eating, alcohol, etc.). Hyperglycaemia usually presents with polyuria, thirst, fatigue, rapid (Kussmaul) breathing (if ketoacidotic) and mental changes including acute confusional states and coma. Hypercalcaemia, often as paraneoplastic manifestation, presents with mental changes including acute confusional states, fatigue, nausea and polydipsia/polyuria[17]. Hypocalcaemia due to hypoparathyroidism can also lead to acute confusional states, sometimes in association with papilloedema[18].

Bacterial meningitis is an acute purulent infection within the subarachnoid space. It is associated with a CNS inflammatory reaction that may result in decreased consciousness[19]. Patients with sepsis syndrome, usually in association with Streptococcus pneumoniae, Legionella pneumophila or Klebsiella pneumonia infections, often suffer significant morbidity[20].

Table-I: Shows that 17(51.5%) patients of group I and 14(42.4%) patients of group II were current smoker, 5(15.2%) patients of group I and 8(24.2%) patients of group II were former smoker and recent smoker were similar in both the groups (15.2%). There was no significant difference in group I and group II (p=0.805).

Twenty-three (69.7%) patients of group I and 18 (54.5%) patients of group II had hypertension. There was no significant difference in hypertension between group I and group II (p=0.205).

Nineteen (57.6%) patients of group I and 23(69.7%) patients of group II had diabetes mellitus. There was no significant difference in diabetes mellitus between group I and group II (p=0.306).

Seventeen (51.5%) patients of group I and 11(33.3%) patients of group II had family history of premature CAD. There was no significant difference in family history of premature CAD between group I and group II (p=0.135).

Twenty-one (63.6%) patients of group I and 12 (36.4%) patients of group II had dyslipidaemia. Prevalence of dyslipidaemia was significant high in group I patients than group II (p=0.027).

Six (18.2%) patients of group I and 7 (21.2%) patients of group II had history of obesity. There was no significant difference in obesity between group I and group II (p=0.757).

Table-II: Distribution and comparison of patients by past medical history (n=66)

Past medical history	Group I (n=33) f (%)	Group II (n=33) f (%)	P value
Previous revascularization			
PCI done	4(12.1)	4(12.1)	[a]0.100[NS]
None	29(87.9)	29(87.9)	
Chronic heart failure			
Present	1(3.0)	0(0.0%)	[a]0.314[NS]
Absent	32(97%)	33(100%)	
Cerebrovascular disease			
Present	2(6.1)	1(3.0)	[a]0.555[NS]
Absent	31(93.9)	32(97.0)	
Chronic kidney disease (CKD)			
Present	3(9.1)	2(6.1)	[a]0.642[NS]
Absent	30(90.9)	31(93.9)	

Table-II: Shows that PCI was done in 4(12.1%) patients in both groups for revascularization (p=1.000). One (3.0%) patient of group I and none of group II suffered from chronic heart failure (p=0.314). Two (6.1%) patients of group I and 1(3.0%) patient of group II had cerebro vascular disease (p=0.555). Three (9.1%) patients of group I and 2(6.1%) patient of group II had Chronic kidney disease (CKD) (p=0.642).

Table-III: Comparison of post procedural mean left ventricular ejection fraction between group I and group II (n=66)

Left Ventricular Ejection Fraction	Group I (n=33)	Group II (n=33)	P value
	Mean±SD	Mean±SD	
LVEF%	47.03±4.92	43.45±4.83	[b]0.004[S]

Table-III: Shows that the mean post procedural LVEF% was significantly more in group I than group II (47.03±4.92% vs 43.45±4.83%, p=0.004) after complete ST segments resolution in both the groups.

Table-IV: Distribution and comparison of adverse outcome after 60, 90 and 120 minutes of procedure between two groups (n=66)

Adverse outcome after procedure	Group I (n=33) f(%)	Group II (n=33) f(%)	P value
Acute LVF			
Present	2(6.1)	11(33.3)	[a]0.005[S]
Absent	31(93.9)	22(66.7)	
Cardiogenic shock			
Present	1(3.0)	6(18.2)	[a]0.046[S]
Absent	32(97.0)		
Death			
Occurred	1(3.0)	2(6.1)	[a]0.555[NS]
Not Occurred	32(97.0)	31(93.9)	

Table-IV: Shows that Acute LVF developed in 2(6.1%) patients of group I and 11(33.3%) patients of group II. Significantly higher number of patients of group I developed acute LVF than group II (p=0.005). Cardiogenic shock developed in 1(3.0%) patients of group I and 6(18.2%) patients of group II. Patients of group I developed significantly more cardiogenic shock than group II (p=0.046). Death occurred in 1(3.0%) patients of group I and 2(6.1%) patients of group II with no significant difference (p=0.555) after complete ST segments resolution in both the groups.

Discussion:

This observational comparative study was conducted in the Department of Cardiology, National Heart Foundation Hospital and Research Institute, Dhaka, Bangladesh for a period of one year. Total 66 patients were studied and they were grouped on the basis of their treatment modality. Group I underwent primary PCI and group II received fibrinolytic therapy as reperfusion therapy for acute ST segment elevation myocardial infarction (STEMI) and their benefits were compared after complete ST segment resolution in both the groups.

Except for the higher prevalence of dyslipidaemia in group I (p=0.027), other cardiovascular risk factors like smoking (p=0.805), hypertension (p=0.205), diabetes mellitus (p=0.306), family history of premature CAD (p=0.135) and obesity (p=0.757) were similarly in both group I and group II. The prevalence of risk factors in the present study was comparable with other studies.

During admission the most frequent complaints of group I and II patients was chest pain (84.8% vs 75.8%) and chest discomfort (15.2% vs 24.2%) with a mean duration of 5.06±2.72 and 4.77±2.54 hours respectively. But the difference was not statistically significant (p 0.05).

The mean heart rate was 75.88±17.44 and 80.97±17.36 beats/min for group I and group II respectively and the result was not statistically significant (p>0.05). The mean systolic blood pressure was 114.39±26.68 and 123.48±23.53 mmHg in group I and group II respectively. The mean diastolic blood pressure was 76.81±14.45 and 80.91±15.63 mmHg in group I and group II respectively. Similar hemodynamic parameters were also reported by Salim[11].

Regarding left ventricular ejection fraction, it is considered to be

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Result:

Table-1: Age and gender distribution of study population (n=78)

Age (Years)	Frequancy		Total
	Male(n=51) (65.38%)	Female(n=27) (34.61%)	78 (100%)
50-59	28(54.90%)	10(37.03%)	38
50-59	16(31.37%)	13(48.18)	29
>70	7(13.72%)	4(14.81)	11

Table-1 shows that out of 78 cases 51(65.38%) cases were male and 27(34.61%) were female. Male and female ratio was 1.92:1. Maximum numbers of patients were between 50-59 years age group.

Study also demonstrated that female patients comparatively older than male, majority 13(48.18%) were in the age group 60-69 years.

Table-2: Status of consciousness of study population (n=78)

Status of consciousness	Conscious	Semiconscious
78(100%)	32%(25)	68%(53)

Table-2: Shows that among 78 cases 32% (25) were alert or conscious and 68% (53) were semiconscious.

Residence	Urban Area	Rural Area
100%(78)	28.21% (22)	71.79%(56)

Table-3: Shows that among 78 cases 28.21% (22) patients were come from urban area and 71.79% (56) patients were come from rural area.

Table-4: History of Hypertension (n=78)

Hypertension	Present	Absent
100%(78)	58.97% (46)	41.03%(32)

Table-4: Shows that among 78 cases hypertension present in 58.97% (46) patients and hypertension absent in 41.03% (32) patients.

Table-5: History of obesity, old CVD and smoking (n=78)

Parameters	Present	Absent
Obesity	28.20%(22)	71.8%(56)
Old CVD	20.51%(16)	79.49(62)
History of smoking	24.35%(19)	75.65(59)

Table-5: Shows that Past medical history shows that 28.20% had obesity, 20.51% had old CVD and 24.35% had history of smoking.

Table-6: Clinical findings of study population (n=78)

Clinical Findings	Present	Absent
Fever	78% (61)	22% (17)
Disorientation	64% (50)	36% (28)
Slurred speech	48% (37)	52% (41)
Sphincter problem	46% (36)	54% (42)
Tachycardia	34% (27)	66 %(51)
Oranial nerve palsy	22 % (17)	78 % (61)
Planter extensor	20 % (16)	80 % (62)

Table-6 shows that fever had present in 78% of cases, disorientation 64% cases, slurred speech 48% cases, sphincter problem 46%) cases, tachycardia 34% cases, cranial nerve palsy 22% cases and planter extensor 20% cases.

Table-7: Aetiology of acute confusional state (n=78)

Aetiology	Frequency	Percentage (%)
Cerebrovascular disease	32	41.02
Electrolyte imbalance	11	14.10
Meningitis	9	11.53
Urinary tract infection	7	8.97
Hypoglycemic coma	7	8.97
Pneumonia	6	7.69
Diabetic ketoacidosis	3	3.84
Encephalitis	2	2.56
Lung cancer	1	1.28
Total	**78**	**100**

Table-7: Shows that the aetiology of different cases of acute confusional state, cerebrovascular disease was the commonest cause of ACS present in 41.02% of patients, followed by meningitis (11.53%), electrolyte imbalance (14.10%), and pneumonia (7.69%). On CT scan of brain, findings shows 23 (71.87%) of cases infarctive stroke and 9 (28.12%) hemorrhagic. Electrolyte imbalance commonly associated with other major physical illness which aggravate the condition. In our study we enrolled patients of severe diarrhoea, advanced cancer, end-stage renal failure patients had association of electrolytes imbalance.

Study shows that 73% of the patients recovered without sequele (MRS score 0 to 2), 23% recovered with sequele (MRS score 3 to 5). Three cases (4%) expired during hospital stay (MRS score 6).

Discussion:

This prospective cross sectional study was carried out to evaluate the aetiology and short-term outcome of acute confusional state among elderly patients in a secondary level hospital of Bangladesh. Present study demonstrates that maximum numbers of patients (48.71%) were between 50-59 years age group, mean age of the patient were 78.38 ± 11.23 years. Out of 78 cases 51 (65.38%) cases were male and 27 (34.61%) were female. Male & female ratio was 1.92:1. Study demonstrated that female patients were comparatively older than male, majority 13 (48.18%) were in the age group 60-69 years.

All findings correlate with the results of similar studies at home and abroad, e.g. with the results of Hossain H[2], a prospective study in tertiary centre of Bangladesh showed majority of cases belongs to 56 to 65 age. Study by Naemee A, Taleb NM, Mahdawi AA[21] revealed that majority of cases were 65 to 70 years. Present study also shows that frequency of disease gradually decreases. It may be due to family and social burden, lack of feelings for better outcome after treatment of elderly subjects. In fact in our country life expectancy is lower than those of the developed countries.

At the time of admission thorough physical examination, history

one of the major endpoints after treatment of myocardial infarction and is strongly related to long term survival. The mean post procedural left ventricular ejection fraction was significantly more in group I than group II (47.03±4.92% vs 43.45±4.83%, p=0.004). Salim[11] also showed that mean post procedural left ventricular ejection fraction was significantly lower in patients treated with IV streptokinase than those treated with primary PCI.

In our study we observed that significantly higher number of patients of group II developed acute LVF (33.3% vs 6.1%, p=0.005) and cardiogenic shock (18.2% vs 3.0%, p=0.046) than group I and Rescue PCI was needed in 5 (15.2%% vs 0%, p=.020) patients of group II than group I. No significant difference was observed between two groups in the development of death (3.0% vs 6.1%, p=0.555)[12].

Conclusion:

Although after the complete resolution of ST segment occurred similarly in group I and group II at 90 minutes after the procedure, significantly higher number of patients of group II developed acute LVF and cardiogenic shock and needed more rescue PCI than group I. But surprise that there was no significant difference in mortality benefits between the two groups. So only some morbidity benefit not the mortality benefits was observed in this study.

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Case Report

Leukemoid Reaction in a Patient with Squamous Cell Carcinoma of Lung: A Case Report

Khan AAM[1], Akhtar PS[2], Khatun N[3], Islam MR[4], Alam MJ[5], Islam MR[6]

ABSTRACT

Introduction: *Lung cancers are known by its high incidence, prevalence and mortality. They may be associated with numerous paraneoplastic syndromes. Mild leukocytosis is not rare.*

Case presentation: *A 65 years old male Bangladeshi, who was diagnosed with squamous cell carcinoma without any evidence of metastasis. Before diagnosis, our patient developed leukocytosis and increasing day by day, for which, despite extensive diagnostic tests, no infection-related or hematological cause could be identified. He developed extreme leukocytosis at over 60620 cells/µL.Descriptions of such leukemic forms of lung cancer are few in the literature. In our case, the complete hematological diagnostic investigation, which included cytological, immunocytological, histological and examination of the bone marrow. The tumor proved to be highly resistant to treatment. Our patient died only seven months after the initial diagnosis.*

Conclusion: *A raising leucocyte count, regarded as poor prognostic factor, can most likely be interpreted as the paraneoplastic production of hematopoietic growth factors. Despite the absence of a verified primary hematological origin or infection, possibility should always be investigated in all patients in a comparable situation.*

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Introduction:

The presence of leukocytosis associated with solid tumors has been documented for many decades[1]. The first demonstration that it was the tumor itself producing a colony-stimulating agent came in 1977 when Asano et al. demonstrated that serial transplantation of human lung cancer tissue from a patient with neutrophilia into mice caused neutrophilia in recipient mice as well[2]. The formal definition of a leukemoid reaction is a white blood cell (WBC) count 50,000/mm^3 or more with a predominance of neutrophil precursors. When this elevation in the WBC count is associated with malignancy it is termed a paraneoplastic leukemoid reaction (PLR)[3]. The differential diagnosis of leukemoid reaction includes infection, hematologic malignancy, iatrogenic origin (e.g. steroids, growth factors), solid tumor spread to bone and PLR. It is thought to occur approximately 10%-15% of cancers. PLR is known to be predictive of poor prognosis. Recent work has documented that there may be relationship between PLR activated by intra-tumoral production of granulocyte colony stimulating factor (G-CSF), the RAS/RAF/MEK pathway & tumourogenesis, specifically, activation of the RAS/RAF/MEK pathway is thought to regulate G-CSF production which in turn, mediates expansion & mobilization of cells that produce factors that promote tumour metastasis[4].

Case presentation:

A 65 years old male, farmer, Smoker,normotensive, non-diabetic with low socio-economic status, from Sujanagor, Pabna, Bangladesh with the complain of fever for about one month since 15 February 2016. The fever was low grade, continuous without chills & rigor. The highest recorded temperature was 102^{0}F. He gave no H/O headache, vomiting, abdominal pain, haemoptysis, hematemesis, abnormal bleeding or collection of pus any where in body. After investigations, his WBC count was found raised & the causes were searched by physical examinations and blood and urine routine and culture, chest x-ray, sonography of whole abdomen and testes. He was attended in Pabna to several doctors & treated symptomatically including various types of antibiotics without any document of infection but WBC count was raising gradually.

Investigation Profile: CBC/cmm

Date 2016	WBC	N %	L %	M %	E %	B %	ESR	Hb	RBC	Platelet
17/4	23400	82	13	3	1	1	91	8.8		N
27/4	27000	84	11	3	2	0	90	8.8		N
11/5	20000	94	4	2	0	0	120	7.8	2.96x10^{12} /L	600x10^9 /L
26/5	17000	90	6	3	1	0	100	8.4		N
18/9	27000	87	9	3	1	0	45	12.2		N
9/9	15000	91	7	1	1	0	87	12.3		N
20/10	38430	92	6	1	1	0	83	12.1	4.13x10^6 /uL	231x10^3 /L
14/11	53920	91	5	2	1	1	42	12.3	4.16x10^6 /uL	103x10^3 /L
19/11	60620	94	4	1	1	0	58	9.8	3.33x10^6 /uL	153x10^3 /L
24/11	54000	93	5	1	1	0	not	Done	3.87x10^{12} /L	193x10^9 /L
03/12	19760	97	2	8	1	1	48	9		N

Editorial

Renal Disorder in Pre-eclampsia

Worldwide, about 2–8% of pregnancies are complicated by pre-eclampsia, a disorder that is characterized by new-onset hypertension and proteinuria after 20 weeks of pregnancy[1]. The excessive soluble fms-like tyrosine kinase (sFlt)-1 or endoglin and the reduced free placental growth factor (PlGF) are responsible for pre-eclampsia[2]. When soluble fms-like tyrosine kinase (sFlt)-1 levels are increased there is an inactivation or decrease of placental growth factor (PlGF) and vascular endothelial growth factor (VEGF) concentration, resulting in endothelial dysfunction[2]. In the case of endoglin, which is a surface coreceptor for the transforming growth factor(TGF), soluble endoglin (sEng) binds to endothelial receptors and inhibits several TGF isoforms,resulting in a decreased endothelial nitric oxide (NO)-dependent vasodilatation[3].Vascular endothelial cells collected from pre-eclamptic women or exposed to serum from pre-eclamptic pregnancies produce less NO than endothelial cells from normal pregnancies[4]. Pre-eclampsia affects kidney function during pregnancy and also increases the risk of future chronic hypertension, chronic kidney disease (CKD) and cardiovascular disease[5]. Pre-eclampsia is associated with a fourfold increased risk of developing end-stage renal disease (ESRD) within 10 years after pregnancy[5]. Pre-eclampsia can be subdivided into mild and severe, with severe forms exhibiting more prominent signs and symptoms of end-organ damage that may result in life-threatening disease. Multiple organ systems may be affected in severe preeclampsia including dysfunction of the central nervous system,liver,cardiovascular system,lungs and kidneys (i.e. proteinuria of $\geq$5 grams in 24hours, oliguria of <500 mL in 24 hours)[6]

Several mechanisms have been proposed to account for the association between preeclampsia and later development of kidney disease. One possibility is that preeclampsia causes direct kidney injury resulting in proteinuria or hypertension that continues to mediate subsequent injury[7,8] In support of this theory, studies have found that 20% to 40% of women with preeclampsia have microalbuminuria 3 to 5 years after pregnancy, compared to just 2% of women without preeclampsia17.However, because pregnancy is typically the first health care encounter for young women, many women have not had kidney function tests prior to pregnancy and pre pregnancy kidney disease may be underappreciated. Preeclampsia and kidney disease may be becaused by factors that mediate both pathophysiologic processes, such as obesity, hypertension, insulin resistance, or endothelial dysfunction[9-12]. Antiangiogenic factors have been proposed to be key elements in the pathogenesis of preeclampsia and in the progression of CKD[13,14].

Typically, women with preeclampsia display mild proteinuria; however, nephrotic range proteinuria and slight hematuria may be seen in severe preeclampsia and represents a significantly increased risk for complications[15]. Although edema can be present in preeclamptic patients, normal pregnancies often will induce edema, making this finding unreliable for the diagnosis of preeclampsia. A dramatic decrease in glomerular filtration rate may occur in preeclampsia although serum creatinine is generally close to baseline levels or may be slightly elevated. Acute renal failure is highly unusual. Other potential clinical features include hyperuricemia and hypercalciuria. Since renal diseases, especially those of glomerular origin, often present with hypertension and proteinuria, the clinical differential diagnosis of preeclampsia is broad and includes various glomerular diseases. Chronic glomerulonephritis, minimal change nephrotic syndrome, focal segmental glomerulosclerosis, membranous nephropathy, post infectious glomerulonephritis, diabetic nephropathy, and sickle cell nephropathy should be considered.

In severe cases of preeclampsia with significant microangiopathic hemolytic anemia and thrombocytopenia, hemolytic-uremic syndrome (HUS) and thrombotic thrombocytopenic purpura (TTP)

should also be included in the differential diagnosis[16]. Although preeclampsia is a clinical diagnosis based on new onset hypertension and proteinuria, as mentioned earlier, the specificity of these features is low and a renal biopsy may be helpful to confirm the suspicion of preeclampsia. Since many other forms of renal diseases may arise during pregnancy, the utility of the renal biopsy is also to exclude (or include) other pathologic processes of the kidney that may mimic preeclampsia clinically. Typically, preeclampsia manifests morphologically as thrombotic microangiopathy (TMA) on renal biopsy, a pattern of renal injury commonly seen in association with endothelial cell injury. It should be stressed that TMA is a histologic and ultrastructural pattern that develops in response to renal injury and is not a specific disease. Many etiologies of TMA exist, including but not limited to thrombotic thrombocytopenic purpura (TTP), hemolytic-uremic syndrome (HUS), malignant hypertension, scleroderma/systemic sclerosis, drugs/medications, antibody-mediated rejection (in allografts), and preeclampsia/eclampsia. Although a few subtle morphologic features appear to be seen more often in renal biopsies from patients with preeclampsia;overall, these findings remain relatively non-specific and are not entirely reliable as morphologic indicators to distinguish preeclampsia from other etiologies of thrombotic microangiopathy (TMA).

At this time, delivery of the neonate and placenta is the only definitive cure of preeclampsia; however, fetal morbidity and mortality is highly dependent on the gestational age at the time of delivery. Therefore, a crucial decision must be made to weigh the benefits of delivery for the mother against the risks of prematurity for the neonate. Generally, quick resolution of both hypertension and proteinuria follows delivery. If delivery is not a viable option, management with antihypertensive medications and prophylactic seizure treatment with intravenous magnesium sulfate may be initiated[17-19]. If preterm delivery prior to 34 weeks is considered, betamethasone is commonly given to the mother to hasten fetal lung development[20]. In severe cases of preeclampsia/eclampsia where preterm delivery is not a viable option, maternal symptoms of significant microangiopathic hemolytic anemia, thrombocytopenia, renal failure, and/or neurologic abnormalities may be treated with plasma exchange with variable benefit[21]. Currently, treatment for preeclampsia is largely supportive, requiring careful obstetric management. As we make significant progress in elucidating the molecular mechanisms of preeclampsia, the hope of future targeted therapies to ameliorate or even prevent the development of preeclampsia appears to inch closer to reality. Given the immense health impact of preeclampsia due to its high world-

of case, clinical evaluation were done meticulously and relevant investigations were sent accordingly. Among the 78 cases 32% were alert or conscious, 68% were semiconscious. Common symptoms and signs include hemiplegia, acute onset of fever, headache, vomiting, neck stiffness, altered mental status (confusion, lethargy, agitation, irritability, inability to talk), abdominal pain and sphincter problem. We observed that fever was (78%) of cases, then disorientation (64%), slurred speech (48%) and sphincter problem (46%) of cases. Among the clinical signs majority of patients are found to have tachycardia (34%), hypertension (30%), cranial nerve palsy (22%) and plantar extensor (20%). It is almost consistent with international data, which says, headache, vomiting and cranial nerve palsy are common presentation in acute confusional state patients[22]. In a study by TW Wong[23] demonstrated that disorientation, behavioral changes and cognitive disturbance are common presentations in ACS.

Past medical history shows, 58.97% of patients had history of hypertension, (28.20%) obesity, (20.51%) old CVD and (24.35%) smoking. Other research support that main risk factors for acute confusional are: 1) Age >65, 2) Pre-existing dementia or depression, 3) Malnutrition/dehydration, 4) Alcohol abuse, 5) Pre- and coexisting medical conditions[24].

Regarding the aetiology of different cases of acute confusional state, cerebrovascular disease was the commonest cause of ACS present in (41.02%) of patients, followed by meningitis (11.53%), electrolyte imbalance (14.10%), and pneumonia (7.69%). On CT scan of brain, findings shows 23(71.87%) of cases infarctive stroke and 9 (28.12%) hemorrhagic. Among the hemorrhagic stroke, 9 (23%) were intracerebral haemorrhage (ICH) and 2 (6%) was subarachnoid hemorrhage (SAH). On background we found that most of the attendants of the patients had no idea about stroke, its management and consequences. Stroke patients developed confusional state due to structural damage by disease process. Then pathophysiology is detoriated by associated nutritional or metabolic abnormalities. Prolong immobilization and inappropriate I/V fluid also had contributed to the sufferings. In a Bangladeshi study by Das S[22] revealed that 22.52% ischemic stroke, 12.29% hemorrhagic stroke and 6.14% subarachnoid hemorrhage patients faced crisis of confusional state.

Data on electrolyte disorders in acute confusional state are somewhat scanty and clinically not easy to distinguish solely without involvement of other comorbid condition. Electrolyte imbalance commonly associated with other major physical illness which aggravates the condition. In our study we enrolled patients of severe diarrhoea, advanced cancer, end-stage renal failure patients who had association of electrolytes imbalance. Among the total 78 cases of acute confusional state patients, 11(14.10%) cases found electrolyte imbalance. Hyponatremia was detected in majority of patients 8 (72%), others imbalance are hypokalaemia 5 (45%) and Hypochloraemia 3 (27%). No cases were found to have hypernatremia, hyperkalaemia, hyperchloraemia. Electrolyte imbalance plays crucial factors for acute confusional state. After any chronic illness or advance cancer, hepato-renal failure or stroke, there are some range of change in body milieu due to nutritional maintenance, immobilization, long time intravenous administration and neuro-hormonal instability.

All these finding correlate with other study[22]; findings showed that the highest incidence of electrolyte imbalance was in 56 to 65 age group. Another study Kusuda K, Saku Y, Sadoshima S, Kozo I, Fujishima M. "Disturbances of fluid and electrolyte balance in patients with acute stroke"[25] demonstrated that the incidences of hypernatremia, hyponatremia, hyperkalemia and hypokalemia were higher in patients with hemorrhage than infarction. In elderly patients, electrolyte disturbances were more common than in young or middle-aged patients. Renal insufficiency and diabetes mellitus were frequent complications in stroke patients, of which majority died within one month of admission. Findings are quite different due to study design & methodology in their study with our hospital management system, inadequate patients care, indiscriminate drug use and poor nutritional support in our country.

Patients symptoms, degree of disability or dependence in the daily activities and clinical outcome had been evaluated and measured by Modified Rankin Scale (MRS). Study shows that 73% of the patients recovery without sequele (MRS score 0 to 2), (23%) recovered with sequele (MRS score 3 to 5). Three cases (4%) expired during hospital stay (MRS score 6).

Conclusion:

Acute confusional states are among the most common problems in general medicine. In this prospective hospital based study with acute confusional state reflects the exact or almost nearest clinical situation of the disease in the secondary level hospital. Maximum effort is given to find out the cause, outcome of acute confusional state by thorough evaluation of its clinical aspects and correlating them with the laboratory findings. As the etiology of acute confusional state are numerous and presentation also variable, so many time create confusion regarding the actual diagnosis. Present study demonstrated that aetiology for acute confusional state patients, 41.02% of patients were cerebrovascular disease, followed by meningitis 11.53%, electrolyte imbalance 14.10%, and pneumonia 7.69%. Hospital prevalence rates and presentation for acute confusional state vary widely because of different patient characteristics, socioeconomical status, hospitalization time, association with multiple comorbid condition and concomitant complication. The highest rates are seen in older patients in critical care settings.

This finding emphasize the great importance of early accurate diagnosis of acute confusional state, as correct diagnosis can lead to judicious management and save many valuable lives.

Limitation of our study was, it was a small study; only patients of ACS admitted in secondary level Hospital, were taken for the study. So this will not reflect the overall picture of the country. A large scale study needs to be conducted to reach to a definitive conclusion.

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Original Article

Anoproctoscopic Distribution of Common Anorectal Diseases among Patients Attending at Sadar Hospital, Jhenidah.

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ABSTRACT

Introduction: Anorectal disorders include a diverse group of pathologic disorders that generate significant patient discomfort and disability. Although these are frequently encountered in general medical practice, they often receive only casual attention and temporary relief.

Objectives: *To explore the epidemiological distribution of anorectal diseases among patients attending at Sadar Hospital, Jhenidah.*

Methods: *A prospective analytical study was conducted on patients attending at Sadar Hospital, Jhenidah to assess and determine the anorectal disease distribution among the population of Jhenidah, Bangladesh.*

Results: *Among those 316 patients, 189 (59.81%) patients were female and 127 (40.18%) patients were male. Among those registered patients the lowest age of the patients was 6 years and highest was 76 years. The study showed that majority of the cases were Anal Fissure 48.41% (n=153), second major cases were Hemorrhoids 19.93% (n=63), 3rd were with perianal abscess 9.49% (n=30), Among the other major cases, there were Anal Fistula 8.22% (n=26), Carcinoma Rectum 3.48% (n=11) and normal findings 3.48 (n=11).*

Conclusion: *Anoproctoscopy in anorectal diseases increases the accuracy of diagnosis.*

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Introduction:

Diseases of the rectum and anus are common phenomena. Their prevalence in the general population is probably much higher than that seen in our clinical practices .The most patients with symptoms referable to the anus and rectum usually do not seek medical attention .The examination and diagnosis of certain anorectal disorders can be challenging, it is a matter of concern that clinical examination of anorectum is infrequently performed in general medical practice.

The diagnosis and management of hemorrhoids, fissure and pruritus ani, account on rough estimate, for more than 81% of the complaints centering around this part of the human anatomy. A systemic approach to patients with anorectal complaints allows for an accurate and efficient diagnosis of the underlying problem. The process can be interviewing the client, clinical examination and co- relation of information. Throughout the process, the patient should be reassured and made as comfortable as possible. The key to diagnosis remains the patient history and confirmation by anoscopy and proctoscopy.

There are few studies carried out on the epidemiologic pattern of anorectal disorders in Bangladesh, specially on anoproctoscopic prevalence of anorectal disease. As anoproctoscopic examination provides more accurate diagnosis, the prevalence of common anorectal disease can be more accurately determined.

Methods:

A prospective analytical study was conducted from January 2014 to July 2015 among patients attending at Jhenidah Sadar Hospital to assess and determine the anorectal disease distribution among the population of Jhenidah, Bangladesh. Jhenidah is a south-western district of Bangladesh comprising 20 lakh people. There are 6 Upazilla Health Complexes and one District Hospital. Huge number of patients come to Sadar Hospital from different parts of the district. Anoproctoscopic examinations were done in operation theatre for relevant patients and findings were recorded in anoproctoscopy register book.

The data were collected from the operating room “Anoproctoscopy Register Book” and with the prior permission from the hospital authority. Patient with same registration number and name was excluded from the study (follow- up cases) to avoid duplication of data. Informed consent was taken from each patient regarding the ano proctoscopic examination. Collected data were managed and analyzed by using SPSS- 16 and proper statistical modeling was used to minimize biases.

Result:

The main objective of the study was to explore the epidemiological pattern of anorectal diseases revealed through anoproctoscopy at Jhenidah Sadar Hospital.

Infection were ruled out by

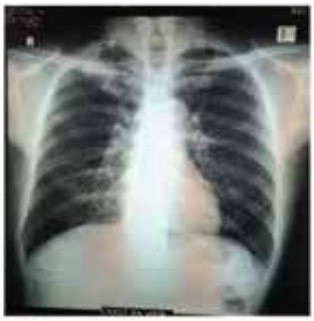
Specimen & Test	Data (2016)	Result
Urine RME	17 & 27 April	Normal
rk39/ICT for kala-azer	17 April	Positive
Rickettial	17 April	Negative
ICT for Malaria	17 April	Negative
Urine C/S	27 April	No growth
Blood C/S	27 April	No growth
Widal	17 April	Non Spnificant
Bone marrow C/S	11 May	No growth
Anti HIV 1,2	11 May	Negative

TB ruled out by

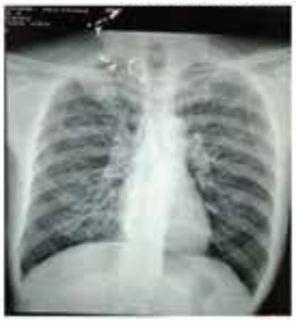
Specimen for AFB	27 April	Not detected
MT	30 April	Negative
Bronchoscopy	08 May	Normal
Sputum & Bloob for	11 May	
Gram stain		Negative
Z-N stain		Negative
GeneXpert & RIF		Not detected

Then he was referred to higher centre where he was investigated by routine & special investigations but no sources of infections were found for raised WBC count. Again, he was treated by empirical antibiotics like ceftriaxone, cefuroxime with clavulanic acid, clindamycin, antifungals & antivirals (acyclovir) under a medicine specialist. Suddenly he developed painful swelling of right testes during treatment in higher centre . Then he was advised to sonogram of testes and it was revealed acute Epididymoorchitis & treated accordingly & the painful swelling was subsided within a short time. There after his testes were normal but WBC count was raised like previous report and he developed cough and mild chest pain. On 03 May 2016, chest x-ray revealed a consolidation in his right lung & he was diagnosed as a patient of SQCC right lung by CT chest & CT guided FNAC on 18 May 2016 & referred to oncologist & he was advised to take CCRT on 31 May 2016 at National Institute of Cancer Research and Hospital (NICRH), Dhaka, Bangladesh.

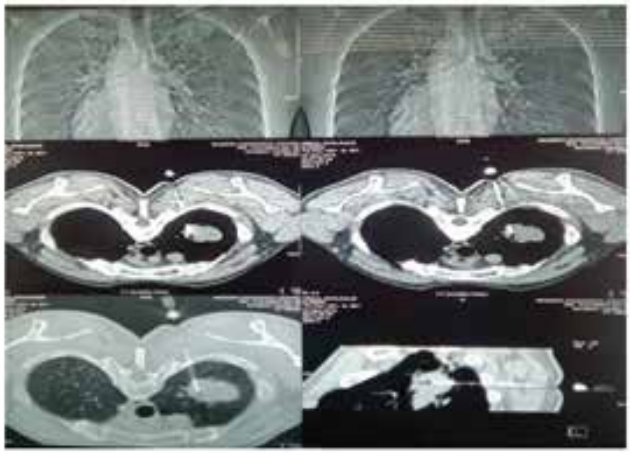
17 April 2016 (normal)



03 May 2016 (RT sideed consolidation)

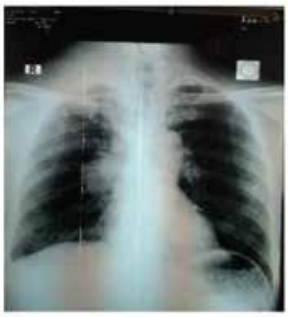


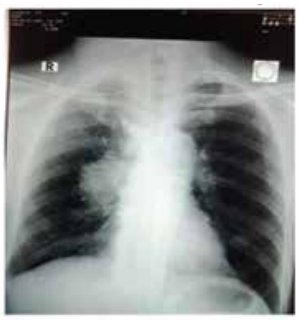
18/5/2016 CT Guided FNAC



Then he had been taken 25 fractions (5000cGy, 2 field daily, 100/fraction) radiotherapy & inj. cisplatin upto 26 July 2016. He felt better for a month. Then he felt respiratory distress, right sided neck swelling, cough and chest pain & thats why he was admitted again at NICRH. He was treated by Inj. Paclitaxel & Inj. cisplatin from 24 September 2016. After 2 cycle chemotherapy, he felt no improvement & his WBC count was raising and symptoms were deteriorating. Then chemotherapy drugs were changed to inj. Gemcitabine & carboplatin. After 1 cycle, WBC count improved but physical conditions were becoming worst.

20 October 2016 14 November 2016
result of treatments were not satisfactory





Our patient died on 15 December, 2016, two weeks after starting the second line chemotherapy and seven months after initial diagnosis.

Discussion:

Leukemoid courses of various tumor entities, especially gastrointestinal, urogenital, head-neck and lung cancers, have been described in the literature[5]. The development can be affected by a variety of factors. On the other hand, this may involve the presence of an infection or abscess formation. Equally, leukocytosis may be induced in the short term by the administration of high-dose corticosteroids. Another possible cause that needs to be considered is an existing or secondary hematological neoplasia, which develops following previous therapy[6]. The cause most discussed in the literature, however, is paraneoplastic production of hematopoietic growth factors. Asano et al. published the first report of CSF-producing lung cancers, characterized by the development of extreme neutrophilia. The neutrophilia was transferred to nude mice by the transplantation

wide prevalence, progress in this field has the potential to greatly improve global women's health in a relatively short period of time.

The uniqueness of medical care in pregnancy lies in two areas: the body's dramatic alterations caused by gestation and the need to treat two patients simultaneously, only one directly accessible. Renal failure in pregnancy presents particular challenges, in that it occurs in a system physiologically altered from baseline and can occur due to disease processes that are specific to pregnancy and as yet incompletely understood. It is crucial for physicians caring for these patients to have a broad knowledge of physiologic alterations in the renal system in pregnancy, to apply the best evidence based diagnostic and therapeutic strategies for these disease processes, and to consider both maternal and fetal effects of disease and therapy.

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Table –1: Anoproctoscopic distribution of common anorectal diseases attending at Sadar Hospital, Jhenidah (N=316)

Name of the Disease	Frequency	Percent
Anal fissure	153	48.41
Anal fistula	26	8.22
Haemorrhoids	63	19.93
Perianal abscess	30	9.49
Anal stenosis	3	1.12
BEP	1	0.37
Carcinoma Rectum	7	2.61
Fungal infection in perianal region	1	0.31
Anal growth	2	0.63
Normal findings	11	3.48
Anal papilloma	1	0.31
Perianal ulceration	1	0.31
Perianal sinus	1	0.31
Pinonidal sinus	1	0.31
Prostatitis	1	0.31
Rectal Papilloma	1	0.31
Rectal ulcer	1	0.31
Rectocele	2	0.63
Sentinel piles	6	1.89
teniasis	1	0.31
Ulcerative Collitis	3	0.94
Total	**316**	**100.00**

Table-1: Shows that total 316 patients attended the operating room for anoproctoscopy from January 2014 to July 2015. Among these 316 patients, 189 (59.81%) patients were female and 127 (40.18%) patients were male. Among the registered patients the lowest age of the patients was 6 years and highest was 76 years. The study showed that majority of the cases were anal fissure 48.41% (n=153), second major cases were hemorrhoids 19.93% (n= 63), third were with perianal abscess 9.49% (n=30). Among the other major cases there were anal fistula 8.22% (n=26), carcinoma rectum 2.61(n=7) and sentinel piles 1.89% (n=6). The rest of the cases were illustrated in table -1.

Discussion:

The main objective of the study was to explore the anoproctoscopic pattern of common anorectal diseases among the patients attending at Jhenidah Sadar Hospital. The study revealed that majority of the cases were anal fissure 48.41%, in comparison to a study done at Rajshahi Medical Collage Hospital, showed 30.42% cases were anal fissure[2]. According to that study the majority cases were hemorrhoids (60.22%)[2], whereas the present study showed hemorrhoids was 19.93% and it was the second major anorectal disorder. The differences between data of these two studies were mainly due to the fact that this study was a prospective analytical study and the other one was perspective study, this study involves in direct evaluation of the patients, other one used the structured questionnaire.

The present study showed the prevalence of anorectal cancer was 3.49% (n=11), however other study showed the incidence of colonic and rectal cancer is 0.72/100,00 in male and 0.69/100,000 in female in India and highest is Philippines with incidence of 0.64/100,000 in male and 0.65/100,000 in female 3.

A study on prevalence of benign anorectal diseases showed most of the patients were suffering from hemorrhoids4. The study showed prevalence is more in females than males. Our present study also showed similar findings that majority of the cases were females suffering from anal fissure. A North American study also showed that most of the cases of anorectal disease were hemorrhoids and most common cause of hematochezia was also hemorrhoids, the study also mention most of the case later diagnosed by endoscopy and biopsy[5].

Conclusions:

Most of the time, the omission of anorectal examination has been a cause of missed diagnosis and many anorectal disorders are wrongly diagnosed and treated. For this, there are increased sufferings of the patient. Anoproctoscopy can increase the accuracy of diagnosis. In any symptoms suggestive of anorectal disorders, the physician must perform anoproctoscopic examination.

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of tumor cells[7]. Likewise, several subsequent investigations demonstrated elevated serum concentrations of the hematopoietic growth factors G-CSF, granulocytemonocyte (GM)-CSF and also interleukin-6 in patients with lung cancer and extreme neutrophilia[8-10].

In a single case report on a patient with a leukemic course of a lung cancer, differentiation from CML was provided by a bone marrow biopsy; but no conclusions were reached on the precise histology of the primary tumor or with regard to molecular genetics[11].

In the case described here, extensive diagnostic tests ruled out infection-related and primary hematological causes for the leukocytosis. A paraneoplastic origin seems likely. It is known that, to a high degree, epithelial tumors can express various forms of the G-CSF receptor and that cell proliferation may occur after ligand binding[12]. Paraneoplastic production of growth factors by the tumor itself would mean permanent stimulation of these tumor cells and explain the high aggressiveness and the uncontrollable progression. It is impossible to judge at this time if potential G-CSF and GM-CSF-stimulated signal transduction can be influenced by medication.

Conclusions:

In rare cases, lung cancer can exhibit a leukemoid course. Where there is autonomous production of hematopoietic growth factors by the tumor cells themselves and concurrent, uncontrollable proliferation, the prognosis for such lung cancers is very poor. To distinguish between this paraneoplastic phenomenon and primary hematological or therapy-related secondary neoplasia, extensive hematological diagnostic tests should always be carried out.

Consent:

Written informed consent was obtained from the patient's son for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Competing interests:

The authors declare that they have no competing interests.

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Original Article

Headache in Acute Stroke

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ABSTRACT

Background: Headache is a common accompanying symptom in cerebrovascular diseases but many aspects of stroke related headache is still unknown, this might be due to the difficulties encountered in headache assessment in the emergency situation of stroke.

Methodology: *A six months prospective observational study was carried out in Chittagong Medical College Hospital, Chittagong. A total of 100 patients with stroke were included in the study to describe headache in acute stroke. We have prospectively studied headache features in patients with first-ever acute stroke and assessed the relationship between headache, stroke location and distribution among Bangladeshi admitted in Chittagong Medical College Hospital. The results of the study were compared with the results of similar type of studies carried out at home and abroad.*

Results: *One hundred patients were randomly selected for this study, of which majority (57%) were males and maximum number (55%) of patients were between 50 to 69 years of age. Computed tomographic (CT) scan revealed ischemic stroke (69%), intracerebral haemorrhage (29%) and subarachnoid haemorrhage (2%). Within the study subjects nearly one third (29%) had headache; while 23.2% among ischemic stroke, 37.9% among intracerebral haemorrhage and 100% among subarachnoid haemorrhage had headache. It was also seen that among the ischemic stroke patients, headache were more common in posterior circulation stroke (25%) than in anterior circulation stroke (22%). In this study, 19 patients had bilateral (66%) and 10 patients had unilateral headache (39%), among which 6 cases were ipsilesional. This study also described the severity and quality of headache. It was seen that among 29 stroke patients with headache, majority (41%) had moderate headache and maximum patients described their headache as pressing headache (41%).*

Conclusions: *This study showed that the headache, a relatively common phenomenon in stroke, was present in one third of the patients. Like other prospective studies, this study also showed that headache is more common and more severe in intracerebral haemorrhage. Majority of the patients described headache as bilateral, pressing headache. When unilateral, headache was more often ipsilesional.*

Keywords: *Headache, Stroke, Acute, Ischemic, Hemorrhagic.*

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Introduction:

The frequency of headache varies widely among different studies, ranging from 18% to 41% depending on data sources[1-3] few reports[4] have prospectively analyzed the incidence, nature, and type of headache in stroke of diverse pathogenesis and location. We had prospectively studied headache features in patients with first-ever ischemic acute stroke and assessed the relationship between headache, stroke location, and etiology. The findings were compared with those in patients without headache.

Methods:

Patients with stroke admitted in the department of Neuromedicine and three other medicine units of Chittagong Medical College Hospital over the study period were selected for this study. The patients were diagnosed clinically as a case of stroke, confirmed by Computed Tomographic (CT) Scan of head. All patients were interviewed regarding headache complaints appearing within 7 days of stroke symptoms, using a standardized questionnaire and were questioned about headache characteristics such as time of onset, duration, location, quality and severity, assessed with Visual Analogue Scale (VAS). Data were also collected from the hospital records on various risk factors such as previous hypertension, diabetes and heart disease. All data were meticulously checked, verified, and compiled in frequency distribution tables.

Results:

The study included 100 consecutive patients with stroke (57 male, 43 female; mean age 61.98±10 years, range 22–86 years, majority of the patients aged between 50 and 59 years), Baseline characteristics of the patients with and without headache were summarized in Table 1. Computed Tomographic (CT) scan revealed ischemic stroke (69%), intracerebral haemorrhage (29%) and subarachnoid haemorrhage (2%). Within the study subjects nearly one third (29%) had headache; while 23.2% among ischemic stroke, 37.9% among intracerebral haemorrhage and 100% among subarachnoid haemorrhage had headache (Figure-1).

Original Article

Renal Failure of Eclampsia Patients

Gani MO[1], Khatun R[2], Ghosh L[3], Hossain MZ[4]

ABSTRACT

Background: *Preeclampsia (PE), an endothelial disease that affects kidney function during pregnancy, is correlated to an increased future risk of chronic kidney disease. Maternal death due to preeclampsia occurs in 3.5 of 100,000 live births, accounting for 39 % of total maternal deaths. Preeclampsia affects kidney function during pregnancy and also increases the risk of future chronic kidney disease (CKD) and associated with a fourfold increased risk of developing end-stage renal disease*

Objectives: *To determine the renal failure in eclampsia.*

Materials & method: *This cross sectional study was carried out on 100 patients with eclampsia in Department of Obstetrics and Gynecology in Dhaka Medical College and Hospital, during the period of October 2012 to March 2013. Study group comprised of 31 diagnosed cases of renal failure and 61 patients with normal renal function. Statistical analyses of the results were obtained by using window based computer software devised with Statistical Packages for Social Sciences (SPSS-20).*

Result: *: Patients with eclampsia having renal failure were found to be 31%. The mean serum creatinine was 1.21±0.18 mg/dl in renal failure and 0.94±0.27 mg/dl in normal group. Blood urea was 9.93±3.77 and 8.26±3.12 mmol/L in renal failure and normal group respectively. Serum uric acid 9.93±3.77 mg/dl in renal failure and 7.94±3.27 mg/dl in normal group. Total urinary protein was 5.93±3.51 g/24 hour in renal failure and 3.88±3.68 g/24 hour in normal group. Urinary volume was 1737.95±1036.48 ml/24 hour in renal failure and 2094.78±897.61 ml/24 hour in normal. The difference were statistically significant (P<0.05) between renal failure and normal renal function. Platelet count was significantly lower in renal failure group compared to the normal group. Blood pressure was significantly higher among the renal failure group than the normal group.*

Conclusion: *Impairment of renal function is common among the patients with eclampsia. So a particular attention should be given to treatments that could worsen the kidney function in that situation.*

Key words: *Pregnancy, Renal failure, Eclampsia.*

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Introduction:

Pre-eclampsia is a syndrome of two distinct disease types; it may be purely idiopathic, restricted to and caused by pregnancy, or it may be due to an underlying hypertensive disorder. Chronic renal disease has long been recognized as an important precursor of pre-eclampsia. In 1982 Fairley and Birch showed that finding dysmorphic ''Glomerular'' erythrocytes in the urine is a highly specific (93%) and sensitive (99%) indicator of Glomerulonephritis. Patients with ante partum eclampsia have significantly higher incidences of acute renal failure (14%). In contrast, women with post partum eclampsia were more unlikely to have acute renal failure (7%)[1].

Renal involvement varies from mild changes to severe damage and characteristic lesion range from simple ischaemia to patchy or completes tubular or cortical necrosis. From renal biopsy studies, tubular necrosis was identified as the commonest cause of renal failure and in 25% cases patchy cortical necrosis was reported[2]. In severe pre-eclampsia and eclampsia, there are elevation in serum creatinine, blood urea and uric acid levels. A significant increase in proteinuria indicates that the disease has worsened. So, only simple renal function test i.e. urinary albumin, serum creatinine and blood urea level may give important clue for prediction of maternal and fetal outcome. In a study, the renal failure was the cause of 7.5% of all maternal deaths due to eclampsia in DMCH[3]. In another study the incidence of eclampsia patients having renal failure was found to be 30% in DMCH[4]. Acute Renal Failure (ARF) is regarded as relatively uncommon in preeclampsia-eclampsia (PE-E) and, in any event, of moderate degree or reversible. Cortical necrosis is reported as rare, event in fatal cases. Little light has as yet been shed on the mechanisms responsible for ARF in PE-E.

Abbate et al.5 documented in their study that the severity of renal impairment did not appear to be related to chronological age, party, period of pregnancy in which PE-E commenced and its duration prior to delivery, presence of frank eclamptic crises or the concomitance of earlier vascular or renal disease (p>0.05). The superimposition of Abruptio Placentae (AP) was the only clinical factor significantly correlated with cortical necrosis (p>0.05). The association PE-E + AP seems to be a particularly unfavorable prognostic sign for the kidney owing to the contribution of additional damage mechanisms (vaso-

Original Article

Treatment of Bipolar Mania with Paliperidone Extended-Release

Bhuiyan MSI[1], Shimu BNS[2], Biswas M[3], Aktar D[4], Ghose TC[5]

ABSTRACT

Introduction:

Bipolar disorder is a life-long condition associated with frequent relapses of symptoms and clinicians often use combinations of psychotropic agents to treat their patients. Second generation antipsychotics are a frequent choice in anti manic pharmacologic treatment. The evidence suggests that for acute mania a combination of lithium or valproate and an atypical antipsychotic is the most effective approach. Paliperidone extended-release (ER) is approved for treatment of acute schizophrenia and for maintenance treatment of schizophrenia. It has -probably as a result of its pharmacokinetic profile shown robust efficacy, and a favorable tolerability in multiple trials for the treatment of schizophrenia.

Objective:

Our goal was to assess its efficacy and tolerability as acute and maintenance of effect therapy in patients with bipolar I disorder experiencing manic or mixed episodes while on a mood stabilizer.

Material & method:

A Cross-sectional observational study was conducted in a tertiary care hospital. Ten hospitalized patients with average age 35.6 years and average duration of illness 2.2 years. They were put on paliperidone (4 patients on 6 mg and 6 patients on 9 mg). They were all receiving a mood stabilizer. The primary outcome measure was the mean change in the Young Mania Rating Scale and secondary measures included the 21-item Hamilton Rating Scale for Depression.

Result:

Paliperidone ER provided improvement of acute mania within 4 days, continuing over 4 weeks and sustained over 16 weeks in 9 of our 10 patients. Paliperidone was generally well tolerated and helped patients achieve and maintain remission without occurrence of depressive symptoms.

Conclusion:

It is well known that patients with bipolar disorder appear more sensitive to antipsychotics. Paliperidone provided in our small sample significant improvement of acute mania and maintained its effect for 4 months. Paliperidone ER may be a safe and effective treatment option for acute mania and provide additional benefit over monotherapy for the management of the manic phase but also for control of mood symptoms in the long run, particularly in preventing manic relapses.

Keywords: *Paliperidone ER, Bipolar disorder, Clinical efficacy, Safety*

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Introduction:

Bipolar disorder (BD) is a chronic, episodic mental illness characterized by periods of mania, depression, and mixed episodes with an estimated lifetime prevalence of approximately 1%[1]. The mean age of onset is 18–20 years, that is, in the late teens to early adulthood[2]. According to the Diagnostic and Statistical Manual of Mental Disorders, 5th edition, text revision (DSM-5-TR), patients presenting with a manic episode must have an abnormally and persistently elevated or expansive mood for 1 week, or for any duration if the patient is hospitalized[3]. In addition, the patient must have symptoms such as racing thoughts, distractibility, and inflated self-esteem or grandiosity. In severe cases, patients may have psychotic symptoms (eg, auditory hallucinations, delusions). Patients with BD often have other co-morbidities, including anxiety and alcohol use as well as other substance abuse disorders, which makes treatment challenging for health care providers[4,5]. Previous evidence has shown that clinical factors such as the

Case Report

Choledochal Cyst as a Diagnostic Pitfall: A Case Report

Rashid MM[1], Ahsan SMM[2], Nur MA[3], Hassan MZ[4], Zafr AHA[5], Ahmed AHMT[6]

ABSTRACT

Introduction: *Choledochal cyst is an aneurysmal dilatation of the bile duct; it is a rare condition with an incidence between 1:100,000 and 1:150,000 live births in the developed countries, although it is probably more common in the developing countries. Most of the patients are diagnosed in childhood with classical symptoms. Adult case diagnosis is very difficult as classical symptoms are usually absent in adult and many other gastrointestinal diseases mimic this. Surgery is the treatment of choice for choledochal cyst.*

Case presentation: *We report a case of choledochal cyst in a young 22 year old female patient, presented to the out-patient clinic with a history of right upper quadrant abdominal pain. The preoperative ultrasound diagnosis was missed. For this, patient suffered a lot.*

Conclusion: *So, early suspicion of this rare disease is important, because early surgical intervention is the only option to avoid the complications of this disease.*

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Introduction:

Choledochal cyst is a rare congenital but not familial anomaly of the intrahepatic or extra-hepatic biliary tract. Cystic dilatation may affect any part of the biliary tree and may occur singly or in multiple numbers. The estimated incidence of choledochal :cysts in western countries varies between 1 in 100,000 to 1 in 150,000 individuals. The rate of incidence is higher in Asia and occurs more frequently in women and male & female ratio is 1:4[1].

The clinical classification describing five different types and subtypes, was revised in 1977s by Todani and colleagues. The most common cystic dilatation is type-1 with diffuse or segmental fusiform dilation of the common hepatic or common bile duct. This type accounts for 50-85% of cases[2].

The etiology of choledochal cysts still remains unclear. One of the hypothesis is the presence of an anomalous pancreatico-biliary ductal confluence proximal to the regulatory control of the sphincter mechanism within the duodenal wall[3]. This predisposes reflux of pancreatic fluid into the bile duct and activation of pancreatic enzymes with deconjugation of bile acids. The combination of activated pancreatic enzymes and deconjugated bile acids induces chronic inflammation causing dilatation of the biliary tree. Oligo-ganglionosis at the terminal portion of the bile duct causing functional obstruction and proximal dilatation has been implicated recently in the etiology[4].

The leading symptoms of choledochal cyst include cholestatic jaundice and abdominal pain. A palpable mass occurs in less than 20% of the cases. In adults, chronic and intermittent abdominal pain is the most common symptom. Recurrent cholangitis and jaundice may also occur. A choledochal cyst is rarely symptomatic, but should be considered if dilatation of bile duct or ampulla is demonstrated. The main diagnostic tool for detection of choledochal cyst, specially in children, is ultrasonography. In adults, Computed Tomography can confirm the diagnosis. However, ERCP or MRCP are the most valuable diagnostic methods and can accurately show dilated segments of the biliary tree[5].

Surgery is the treatment of choice for choledochal cyst. Complete excision of all cystic lesion is recommended because of the potential risk of recurrent cholangitis and the high risk for malignant degeneration[6]. Excision of the cyst and reconstruction of the biliary tree by choledocho/hepatico-jejunostomy with Roux-en-Y loop is the standard procedure[7].

Case Report:

The case is a 22 years old lady presented in a private facility with right upper abdominal colicky pain along with nausea and vomiting. She had similar attack of recurrent abdominal pain. Clinical examinations revealed normal findings. Laboratory findings (Blood Count- Total WBC- 12500/cumm, Serum Creatinine- 0.8 mg/dl, Serum Bilirubin- 1.2 mg/dl) were found normal except USG of whole abdomen which revealed multiple bright echogenic structures of various sizes casting posterior acoustic shadow within the gall bladder. The CBD and pancreatic duct were normal. With this USG findings, the case was diagnosed as chronic cholecystitis with cholelithiasis.

Then Open Cholecystectomy was done on 19th February, 2015. Post-operative period was uneventful initially. But on 15-17th post-operative day, the patient developed abdominal pain, fever, nausea and vomiting. On 22nd post-operative day, the

Table-1: Characteristics of patients with or without headache at Stroke onsets (n=100)

Risk Factors	Headache (n=29)	No Headache (n= 71)
Age	61.9±10	71.4±10
Sex		
Male 57 (57%)	14(48%)	43(60.5%)
Female 43(43%)	15(51%)	28(39.4%)
Cigarette smoking	12(41.3%)	42(59.1%)
Hypertension	19(65.5%)	42(59%)
Diabetes	8(27.5%)	32(45%)

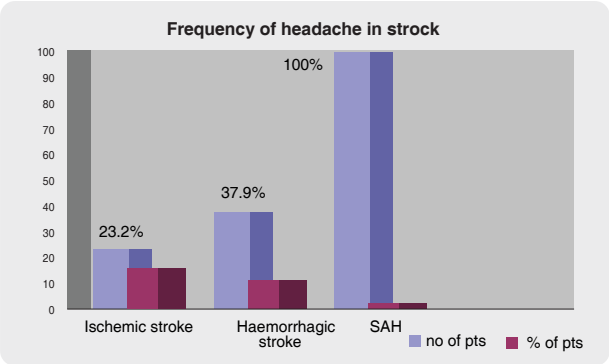

Figure-1 Frequency of Headache in Stroke

Table-2: Distribution area of Ischemic Stroke

	Patient with headache (n=16)	Patient without headache (n=53)
Carotid artery distribution area		
ACA	4	9
MCA	3	15
Posterior circulation area	7	21
Multiple lesions	1	5
No lesion	1	9

Table 2 showing the relation between the headache and distribution area of ischemic stroke. In ischemic stroke patients, headache were more common in posterior circulation stroke (25%) than in anterior circulation stroke (17%) (Table 2)

Table-3: Characteristics of the pain and accompanying symptoms of headache (n=29)

Side of headache	
Unilateral	10 (34%)
Bilateral	19 (66%)
Severity	
Mild	8 (27.59%)
Moderate	12 (41.38%)
Severe	9 (31%)
Quality of Headache	
Dull	9 (31%)
Throbbing	8 (28%)
Pressing	12 (41%)

Characteristics of the pain and accompanying symptoms of headache were presented in Table 3. Among 29 stroke patients with headache 10 (34%) of them had unilateral headache, 19 (66%) of them had bilateral headache. This study also described the severity and quality of headache. It was seen that among 29 stroke patients with headache 9 (31%) of them had severe headache, 12 (41.38%) of them had morderate headache, 8 (27.59%) of them had mild headache. 12 (41%) of them had pressing headache, 9 (31%) of them had dull headache and 8 (28%) of them had throbbing headache.

Table 4: Severity of Headache Among Different Stroke Paitents

Types of Stroke	Mild	Moderate	Severe
Ischaemic stroke	5	9	2
Haemorrhagic stroke	3	3	5
SAH			2

Table 4 showing the severity of headache among different stroke. It was seen that headache was more severe in sub arachnoid haemorrhage (100%) followed by intra cerebral haemorrhage (45%)

Discussion

This study was performed in Department of Neuromedicine and three adult Medicine units of Chittagong Medical College Hospital (CMCH), Chattagram, Bangladesh from March 2010 to August 2010. The hospital is a 1000 bed tertiary referral hospital for Chattagram City and the surrounding districts covering around 10 million population. In this study, peak age of incidence was between 50-59 years (30%), followed by 60-69 years (25%), 70-79 years was (22%). Bashar et al., 1995; Manadir et al 2001; and Chowdhury et al., 1991; found the similar age incidence between 5th to 7th decade in our country.

Men suffer more than women from stroke and it affects male 1.5 times more than female (Thompson and Morgan, 1990). In this study 57% male and 43% were female i.e. male incidence 14% higher than female and the ratio was 1:3, which is closer to the Thompson (1990) study but a similar study was conducted by Mannan (2001) and Alamgir (1975) previously shows gross difference (M:F=4:1) with higher male predominance. The present male to female ratio (1.3:1) might reflect the positive attitude and awareness of the society toward female and the availability of the hospital management to the female patients.

In the study, headache was found to be relatively common phenomenon in stroke. It shows headache was present in nearly one third(29%) of acute stroke patient, similar to Vestergaard K et al., 1993; and Verdelho et al., 2008; where they found headache in 27% and 28% respectively.

This study also shows (Table 2) that the frequency of headache varies with the type of headache. Study shows headache is more common in haemorrhegic stroke. It shows ferequency of headache in haemorrhegic stroke is 38%. Similar frequency also observed by Verdelho et al., 2007 and Koudstaal et al.,1991 which are 39% and 36% respectively.

The frequency of headache in infarction observed in this study is 23% which is similar to that reported in other prospective studies. Portenoy et al., 1985; found the frequency of headache in infarction to be 27%, Vestergaard et al., 1993; found to 26%.

This study also shows in ischemic stroke, headache is more common in stroke in the posterior cerebral circulation (30%), than stroke in the carotid distribution area (20%) which is similar to other studies like Tentschert et al., 2005; and Verdelho et al.,

spasm, disseminated intravascular coagulation, hemorrhagic shock) furnished by AP, while PE-E itself prepares the ground for AP. The fact that PE-E is difficult to diagnose when AP is the onset of symptom may be responsible for the underestimation of its contribution towards the induction of severe renal damage mentioned by[5].

The risk factors for ARF included Organ System Failure (OSF), bilirubin > 12 µmol/L, uric acid > 5.9 g/dl, abruptio placenta and oliguria were significantly associated with eclampsia. Acute renal failure with eclampsia is a frequent situation that required intensive management when risk factors were present. The need for dialysis was a rare condition. In our setting many patients come late to hospital with complications at time including renal complications. Therefore, this study designed to find out the renal failure in patients with eclampsia.

Materials and Methods:

This descriptive cross sectional study was carried out on one hundred eclampsia patients with renal impairment and normal renal function with singleton pregnancies admitted the Department of Gynecology & Obstetrics in Dhaka Medical College and Hospital, during the period from October 2012 to March 2013. Patient refused to sign consent paper, known cases of essential hypertention, diabetes mellitus, chronic renal disease, liver disease, epilepsy, psychiatric problems and incompletely investigated patients were excluded from the study. Out of 100 cases, 31 patients had renal failure and rest 69 had normal renal function. The objectives of the study along with its procedure, alternative diagnostic methods, risks and benefits of this study were explained to all patients in easily understandable local language and then informed consent was taken from each patient. All the patients were evaluated by detail history and clinical examination and all information were collected in a pre-designed structured data collection sheets. Eclampsia was defined as the occurrence of seizures in the presence of pre-eclampsia (shown by hypertension- diastolic blood pressure of at least 90 mmHg, proteinuria one “plus” or at least 0.3 g/24 hour occurring after 20 weeks gestation). Acute renal failure was defined by the following criteria: a serum creatinine concentration > 140 mmol/L at the time of admission in intensive care unit without preexisting renal disease. Physiologic variables was measured at the time of ICU admission to calculate severity scoring indexes: Acute Physiology and Chronic Health Evaluation System II (APACHE II) score, the Simplified Acute Physiology Score (SAPS), the number of Organ System Failure (OSF), and Simplified Acute Physiologic Score applied to Obstetric (SAPSO)6. The variables were recorded at admission in hospital: demographic data: age, weight, mean gestational age, systolic and diastolic blood pressure before any therapeutic, gravidity, clinical findings; abdominal pain, nausea and vomiting, jaundice, edema, proteinuria by dipstick, laboratory findings (platelet count, hemoglobin, bilirubin uric acid concentration, proteinuria, aspartate aminotransferase and lactate dehydrogenase values, prothrombin time, fibrinogen, blood urea nitrogen and creatinine serum concentration. Urine was also analyzed for proteinuria on admission and a 24-hour urine collection was performed to determine total protein clearance. Diagnosis of HELLP syndrome required the following laboratory findings: hemolysis defined by abnormal peripheral smear, increased bilirubin (> 12 mmol/L) and increased lactic dehydrogenase (LDH > 600 U/L), elevated liver enzymes defined as increased serum aspartate aminotransferase (ASAT) level > 70 IU/L, alanine aminotransferase (ALAT) level > 60 IU/L and low platelets defined as platelet less than 100,000mm[3,7]

Criteria For Impairment Of Renal Function:

Impairment of renal function was indicated by the elevation in serum creatinine (upper limit of normal in pregnancy is 0.9 mg/dl), blood urea (upper limit of normal in pregnancy is 15 mg/dl or 2.5 mmol/L) and uric acid level (upper limit of normal in pregnancy is 4.5 mg/dl). Two random clean-catch or catheter urine specimen with 2+ (1g albumin/L) or more on reagent strip or 1+ (0.3g albumin/L) if specific gravity less than 1030.

Statistical Analysis:

Statistical analyses of the results were obtained by using window based computer software devised with Statistical Packages for Social Service (SPSS-20). The mean values were calculated for continuous variables. The quantitative observations were indicated by frequencies and percentages. Student t-test was used for continuous variables. P values <0.05 was considered as statistically significant. .

Results:

Most of the patients age belonged to ≤20 years in both groups, which was 14 (45.2%) in renal failure group and 44 (63.8%) in normal renal function group. Most of the patients were housewives in both groups (96.8% vs 95.7%). More than a half (54.8%) of the renal failure group had primary level of education and 37 (53.8%) in normal renal function group. Majority of the patients came from lower-income socioeconomic status that was 26 (83.9%) and 45 (65.2%) in impaired and normal renal function respectively, which were not statistically significant between the renal failure and normal renal function group of the eclamptic patients. Most of the patients had no ante natal visits in both groups, which was 17 (54.8%) in renal failure group and 46 (66.7%) in normal renal function group. The mean gestational age was found 34.2±3.7 weeks in renal failure group and 33.6±2.8 weeks in normal renal function group.

Table I: Type of eclampsia between the impaired and normal renal function group (n=100)

Type of eclampsia	Renal failure (n=31)		Normal renal function (n=69)	
Antepartam eclampsia	n	%	n	%
	19	61.3	30	43.3
Intrapartum eclampsia	9	29	23	33.3
Postpartum eclampsia	3	9.7	16	23.2

Table II: Comparison of renal function between the impaired and normal renal function group of the eclampsia patient (n=100)

Particulars	Renal failure (n=31)	Normal renal function (n=69)	P value
	Mean±SD	Mean±SD	
Serum creatinine (mg/dl)	1.21±0.18	0.94±0.27	0.001[S]
Blood urea (mmol/L)	11.18±3.25	8.26±3.12	0.001[S]
Serum uric acid (mg/dl)	9.93±3.77	7.94±3.27	0.008[S]
Total urinary protein (g/24 hour)	5.93±3.51	3.88±3.68	0.01[S]
Urinary volume (ml/24 hour)	1738±1037	20945±897	0.027[S]

s=significant, P value reached from unpaired t test

:presence of anxiety and alcohol use are associated with non adherence to bipolar medications, and that these factors may also contribute to hypomanic or manic episodes.6 Current consensus guidelines recommend pharmacotherapy as first-line treatment for patients. Lithium, valproate, or second generation antipsychotics (SGAs) represent first-line options, with valproate being preferred over lithium for mixed episodes[7,8]. Combination therapy, consisting of different pharmacologic classes, can be implemented in severe cases. Overall, SGAs are equally, if not more, effective than lithium and valproate for the acute treatment of mania. This was recently confirmed in a meta-analysis that systematically reviewed 68 randomized controlled trials of mood stabilizers and antipsychotics in the treatment of mania[10]. The main outcome of the study was to assess the mean change of the Young Mania Rating Scale (YMRS) over 3 weeks as well as treatment discontinuation rates. Overall, antipsychotics tended to be more efficacious and had fewer discontinuations compared with mood stabilizers. Although there are several treatment options targeting manic symptoms, reported relapse and recurrence rates range from 39% to 52% per year with varying medication regimens[11]. Non adherence to medication also has a substantial impact on patient outcome and recovery. A study reported that 10%–60% of patients with mood disorders are non adherent to treatment, which is possibly related to medication side effects[12]. Conversely, those who are adherent with medications may develop untoward side effects associated with chronic use, including weight gain, metabolic disturbances, and movement disorders[13]. As a result, it is necessary to seek new agents that are well tolerated to improve patient adherence and overall outcomes. The aim of this review is to evaluate the pharmacology, safety, and efficacy of paliperidone extended release (ER) for the treatment of BD. Paliperidone (9-OH-risperidone) is an SGA currently FDA approved for the treatment of schizophrenia and schizoaffective disorder. Paliperidone has the potential to treat the manic symptoms of BD. It is the major active metabolite of risperidone and it has many similarities with its parent compound that may contribute to potential efficacy in the treatment of manic episodes in BD. Additionally, paliperidone appears to have fewer side effects than risperidone, which may improve patient adherence.

Methods:

Ten hospitalized patients (6men and 4 women) with average age 35.6 years and average duration of illness 2.2 years, with acute bipolar I mania (2 of them with a mixed episode) were put on paliperidone (4 patients on 6 mg and 6 patients on 9 mg). They were all receiving a mood stabilizer (3 were on valproate, 2 on lithium, and one on topiramate) although compliance was partial in at least 4 of the cases. The primary outcome measure was the mean change in the Young Mania Rating Scale (at baseline average: 40,3) and secondary measures included the 21-item Hamilton Rating Scale for Depression (HAM-D-21)

Result:

Paliperidone ER provided improvement of acute mania within 4 days, continuing over 4 weeks and sustained over 16 weeks in 9 of our 10 patients. It was discontinued in one at day 8 due to worsening of her symptoms. Paliperidone was generally well tolerated and helped patients achieve and maintain remission without occurrence of depressive symptoms. In 6 of the patients on 9 mg, paliperidone was lowered to 6 mg after 8 weeks without recurrence of symptoms. No patient developed major depression.

Discussion:

Berwaerts et al conducted a 3-week, double-blind, Placebo controlled, dose–response study in adult patients [n = 443; mean {standard deviation (SD)} age: 39 (10.9); 53% males] with a DSM-IV-TR diagnosis of BD I, most recent episode of mania or mixed mania. The study included a 7-day washout period, followed by 3 weeks of double-blind treatment and a 1-week follow-up for safety assessments. Participants were randomized to receive either paliperidone ER 3, 6, or 12 mg/day or placebo. Patients receiving paliperidone ER 12 mg had significant improvements in mean (SD) YMRS total score at the endpoint compared with placebo (−13.8 [9.67] vs −9.8 [10.60]; P , 0.025) based on last observation carried forward analysis. Another study evaluated the efficacy and safety of Paliperidone ER in a 12-week randomized, placebo and active control study with quetiapine in adult patients (n = 486; mean [SD] age 39 years [10.9]; 58% males) with a DSMIV- TR diagnosis of acute manic or mixed episodes of BD.33 There was a 1-week washout period, followed by a 3-week double-blind phase in which patients received flexible dosing of paliperidone ER, at 3–12 mg/day. A 9-week double-blind maintenance phase was continued, where patients either continued flexible dosing with their active treatment or switched from the placebo to paliperidone ER 6 mg. Paliperidone ER was initiated at 6 mg/day and titrated or tapered in 3 mg increments with a maximum dose of 12 mg/day as clinically necessary. Quetiapine was initiated at 100 mg/day on day 1 and forced titration to 400 mg/day at day 4, with adjustments of 200 mg/day to a maximum of 800 mg/day. Median doses were 9 mg for the paliperidone ER group and 600 mg for the quetiapine group during the 3-week phase. Based on the last-observation-carried-forward analysis, at the end of 3 weeks, paliperidone ER-treated patients had a significant mean reduction in least square means (LSM) YMRS total score compared with the placebo group (LSM difference from placebo was −5.5; 95% CI −7.57, −3.35; P , 0.001). At the end of 12 weeks, LSM for changes in YMRS total score between quetiapine and paliperidone ER was −1.7 (95% CI −0.47, 3.96). The lower limit of 95% CI (−0.47) was greater than the noninferiority margin of −4, so paliperidone ER was considered noninferior to quetiapine in this study. Additionally, mean (SD) change in Global Assessment of Functioning scores was significantly improved in the paliperidone ER group (12.2 [11.1] compared with the placebo (6.7 [13.5]) at 3 weeks (P , 0.001). There was a higher percentage of remitters in the paliperidone ER-treated group compared with the placebo at 3 weeks (52% [99/190] vs 28% [30/104], P , 0.001). As a result, it appears that paliperidone ER 3–12 mg is as effective as quetiapine for the treatment of acute mania; however, the higher doses may be more effective, as the median dose was 9 mg.

Conclusion:

Well tolerated and effective therapies for bipolar mania are required. It is well known that patients with bipolar disorder appear more sensitive to antipsychotics. Paliperidone provided in our small sample significant improvement of acute mania and maintained its effect for 4 months. Paliperidone ER may be a safe and effective treatment option for acute mania and provide additional benefit over monotherapy for the management of the manic phase but also for control of mood symptoms in the long run, particularly in preventing manic relapses.

patient's condition deteriorated and developed features of peritonitis. New set of investigations had been done and the test results showed Hb%- 10.2 gm/dl, Total WBC count - 12000/cumm, Serum Bilirubin- 1.0 mg/dl, Serum Creatinine- 0.6 mg/dl, Chest X-ray showed sub-pulmonic pleural effusion in right lung with pneumonitis.

Then emergency laparotomy followed by peritoneal toileting and insertion of a sub-hepatic drain tube was done on 24th day after cholecystectomy (on 15/03/2015). Patient's general condition improved after this operation. Patient was discharged along with drain tube with an advice to follow up. During follow up the investigation reports revealed: S.Bilirubin- 0.7 mg/dl, S. ALP- 100 U/L, Prothrombin Time: 16 sec (control 13 sec), INR- 1.16. MRCP,which was not done previously, showed multiple dilated intrahepatic biliary duct with dilated CHD & CBD, with abrupt cut off of biliary tree in the lower part CBD and non visualization of lower 6 mm of CBD just proximal to terminal end and prominent pancreatic duct was apparent upto its terminal opening. The findings were suggestive of distal CBD transaction and/or stricture with proximal dilatation and mild fluid collection in peri-hepatic and sub-hepatic spaces with a drain tube in situ. With these reports, the patient was referred to a tertiary level private facility specialized for gastro-intestinal & hepatobiliary intervention for further management.

Upon consultation with a hepatologist, the patient underwent ERCP, 17 days after the emergency laparotomy. ERCP showed narrowing of distal end of CBD with gross saccular dilatation of rest of the CBD and CHD. Multiple negative shadows were present within CBD. There was evidence of dye leakage at the level of cystic duct stump. The ERCP diagnosis was Choledochal cyst with choledocholithiasis with biliary leakage from cystic duct stump. Papillotomy and Plastic Stenting (7fr 10 cm) was done temporarily and she was advised for biliary reconstructive surgery.

Patient's symptoms improved after therapeutic stenting but not relieved permanently. Then, patient-party went to a neighboring country for taking better treatment. At the overseas hospital, after initial investigation, stent was removed and brush cytology was done. ERCP revealed huge dilatation of CBD in upper part with narrowing in lower part. Again a (10fr 10 cm) plastic stent was placed in biliary tree and she was advised for surgery. The cytology report, however, suggested adenocarcinoma(?).

Then the patient returned to home country and consulted a hepatobiliary surgeon, who reviewed her clinical history and found that she had repeated attack of abdominal pain, fever with chills and rigor. After further evaluation & assessment, she was planned for biliary reconstruction with excision of the Choledochal cyst. Pre-operative investigations showed Hb%-12 gm/dl, ESR- 25mm in 1st hour, Total WBC- 11500/cumm, S. Creatinine- 0.8 mg/dl, S.Bilirubin- 0.8 mg/dl, SGPT- 110U/L, SGOT- 90 U/L, S. ALP- 540 U/L, Prothrombin-Time-15 sec (control 13 sec), INR- 1.17. She was operated on 16/10/2015, 8 months after the emergency laparotomy. Right subcostal incision with midline extension up to Xiphi-sternum was made. There was no ascites, peritoneal seedlings or liver metastases, but severe adhesion around GB fossa with liver bed and duodenum was present. After adhesiolysis and kocherization of duodenum, a huge Choledochal cyst was found. Total excision of cyst and Roux-en-Y hepatico-jejunostomy was done. A drain tube was placed in right sub-hepatic region.

The histopathology report of excised cyst revealed ulcerated mucosa with infiltration of acute or chronic inflammatory cells. No malignancy or granuloma could be found.

The post-operative period was uneventful. Patient started oral diet on 4th post-operative day (POD). Drain tube was removed on 7thPOD and then discharged. Stitch was removed on 14thPOD. The wound was healthy. She came back after one month for follow up and reported no complain with overall improved general condition.

Discussion:

This case report highlights the difficulties involved in making a correct diagnosis and the proper operative treatment of Choledochal cyst, as the pathological and clinical characteristics of chronic calculus cholecystitis often overlap with that of choledochal cyst.

Choledochal cyst is a rare abnormality (1 in 100,000 to 1 in 150,000 individuals) of the biliary tree of unknown etiology and has a strong female predominance (male & female ratio 1:4)[1]. Most of the cases are diagnosed in childhood where only 25% patients are initially seen as adults[8]. The clinicaltriad of jaundice, a palpable mass and abdominal pain occurs only in one-third of all patients, mostly in child cases. Although abdominal pain is the most common presenting symptom in adult cases, most have non-specific clinical symptoms. The incidence of biliary tract cancer in patients with choledochal cysts increases with age[9]. The non-specific symptoms of choledochal cysts, including abdominal pain with or without jaundice, are common in many other illnesses of the upper gastrointestinal tract. So it may be frequently overlooked in the differential diagnosis specially in adults. In our case, only abdominal pain was the complaint, which led her to seek medical attention. So, in absence of jaundice and palpable mass, choledochal cyst was easily mistaken as a differential diagnosis. The first imaging modality generally used for the biliary tree is ultrasonography which, with the exception of type-3 and type-4 cysts, will show a cystic mass usually at the porta hepatis separated from gall bladder. Sensitivity of ultrasonography in making diagnosis is 71-97%[10].

As all ultrasonography is limited by body habitus, bowel gas and operator dependent, unfortunately the diagnosis of the choledochal cyst in our case report had been missed. More-over, modern techniques like CT scan, MRCP, ERCP etc were not applied for confirmation of diagnosis due to unavailability of that facility. Ultimately the patient underwent open cholecystectomy as a case of cholelithiasis. The operative field was not explored properly and per-operative cholangiography was not done as no abnormality could be identified during operation. Although open cholecystectomy is a safe procedure, the complication rate has been reported to range from 0.1-0.2%, though this has decreased in the current era. The development of post-operative biliary peritonitis may be due to injury to biliary tree, or ligatures slipping off from cystic duct stump, or most commonly, transection of Luschka ducts; due to which this patient presented with persistent abdominal pain, nausea, vomiting and/or abnormal liver function tests.

MRCP is considered to be the gold standard for the diagnosis of any biliary pathology[5]. The sensitivity of MRCP for diagnosis has been reported as high as 90-100%[11]. So, when ever any complication develops following cholecystectomy, MRCP is the gold standard for diagnosis as well as exclusion of biliary

2007; The reason for this difference is not known. One possibility may be that the cerebral vasculature of meninges in the posterior circulation is more heavily innervated by nociceptive afferents than the carotid circulation area. (Tentschert et al., 2005;).

Headache is more often bilateral 51% than unilateral 37% which is similar to Gorelick et al., 1986; Koudstaal et al., 1991; and Verdelho et al., 2007;.

This study also shows the severity of headache. Despite the general conviction that haemorrhagic stroke is associated with more severe headache than ischaemic stroke, few studies Gorelic et al.,1986; and Arboix et al., 1994; have explored severity of headache according to stroke type. This study shows about 45% patients of haemorrhagic stroke described headache as severe, where as only 13% patients of ischemic stroke had severe headache. It is probably due to the fact that in haemorrhagic stroke, headache is caused by mechanical strech and blood product that diffuse and irritates trigemino-vascular system at the basis of skull.

This study also describes the quality of headache the patients experienced. It shows majority (41%) of the patients describes headache as pressing headache which is similar to what Portenoy et al.,1985; found in there patient group (45%).

This study is a very small scale study in respect to number of samples, time and place. Only the hospitalized patients were included in the study.

So, firm conclusion cannot be made from the study. However an attempt has been made to describe headache in acute stroke. To describe headache in acute stroke more precisely, study can be carried out for a prolong period of time taking large number of samples, as this hospital based small scale study may not reflect the actual scenario.

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Table III: Comparison of Haemoglobin, Platelet count, Blood pressure and Electrolytes between the impaired and normal renal function group of eclampsia patient (n=100)

Particulars	Renal failure (n=31)	Normal renal function (n=69)	P value
	Mean±SD	Mean±SD	
Haemoglobin (g/dl)	10.48±2.86	9.67±2.55	0.16^{ns}
Electrolytes (mmol/L) Sodium	2.57±0.75	2.1±0.94	0.015^{s}
PotassiumChloride	3.8±0.63	4.12±0.66	0.025^{s}
HCO3	103.96±5.33	104.96±5.85	0.016^{s}
	19.74±4.9	22.78±4.47	0.001^{s}

s=significant, ns= not significant, P value reached from unpaired t test

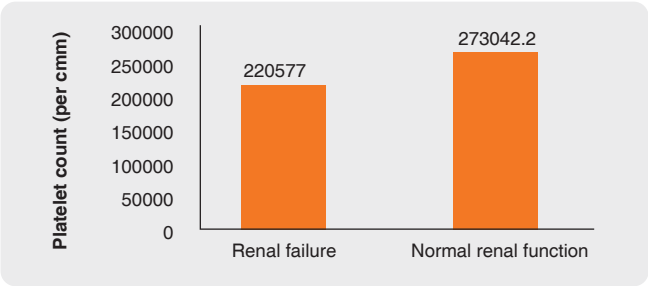

Figure 1: Bar diagram shows mean Platelet count (per cmm) of the study subjects

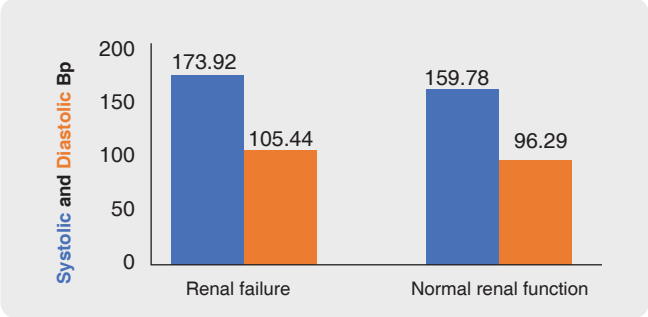

Figure 2: Bar diagram shows Mean Systolic BP and Diastolic BP of the study subjects

Discussion:

Preeclampsia-eclampsia, pregnancy related disorders are major problems and can cause morbidity and mortality. In pregnancy, there are many causes and promoter factors for development of acute renal failure. The most common cause of ARF is acute tubular necrosis[9].

In this current study, out of 100 eclampsia patients, out of which 31% had renal failure. In our country[8] study showed 30% of the eclamptic patients were found to have impairment of renal function. The incidence of renal failure in[10] study was 25.8% and 19.3% observed by[11] in Turkey. The above findings are still high with compared to that found in developed countries (4%) reported by[11,12]. The difference in the incidence is explained by the definition of acute renal failure (ARF). Some authors included only the patients requiring hemodialysis, others, patients with oliguria, others, patients with creatinine level >105 mmol/L. Others remain evasive on the definition of ARF. The threshold of 140 mmol/L was selected in an arbitrary way and corresponds to what is usually retained in the literature[13,14]. The second reason is that the majority of the studies focused on the ARF in the preeclamptic patient, thus, excluding the severe forms like eclamptic patients with numerous lesions. Moreover, the place of study (intensive care or gynecoobstetric service) as well as the country plays a considerable role in the incidence of this complication. In developing countries, the incidence of eclampsia is 10 times higher than that observed in the developed countries and therefore, complications are higher. Furthermore, as it is the case of our hospital, it constitutes a center of reference for the assumption of responsibility of these patients with intensive care and the possibility of performing hemodialysis. Many patients are transferred secondarily in our hospital and their transportation is not medically equipped. Many authors insist on the fact that the term and the time of the occurrence of convulsions lead to complications[12]. A term lower than 32 SA is one of the factors that cause ARF.

Incidence of eclampsia has been found to be higher in the young women who are less than 25 years of age in studies carried out in Bangladesh and other countries[8,13]. 55.6% of the patients in the renal failure group were above 20 years of age whereas only 25% of the patients in the normal renal function group were above 20 years of age.

In[8] study, 72% of the eclamptic patients had ante partum eclampsia which is the commonest type of eclampsia as shown in different studies[15,16] studied 53 pregnancies complicated by eclampsia in two tertiary centers in North Carolina, USA and found that 53% of them had antepartum seizures, 36% had intrapartum and 11% had postpartum seizures[15]. Like other studies most of the eclamptic patients (66%) in this study were primigravida.

Khan[4], study showed significant difference in blood urea and serum creatinine level between case and control groups. Mean serum uric acid level was significantly higher in the study group (6.88±1.89 mg/dl) compared to the control group (3.97±0.8 mg/dl, p<0.01). Higher serum uric acid level in the eclamptic patients has also been observed by other investigators[4,8].

Results from the present study corroborate the findings from earlier reports[4,8] that nulliparous women are at increased risk of preeclampsia and eclampsia. It is believed that this high risk is related to the maternal first exposure to chorionic villi, specifically the trophoblast, which is of foetal origin. Eclampsia was also observed in a number of multigravidas in this study. Similar observation was also made by other investigators[4,13]. Out of 31 eclamptic patients with renal failure 61.3% were primigravida whereas 82.6% of the patients with normal renal function were primigravida. 38.7% of patients in the renal failure group were multigravida compared to 17.4% of patients in the normal renal function group. So it appears from these results that primi eclamptic patients are at risk of developing impairment of renal function. Similar observation was made by[13] in Dhaka Medical College Hospital.

Generally it is seen that patients from the higher income group go to the private health centers and lower income group come to the government hospitals, a definite conclusion can not be drawn regarding the economic status as this study was hospital based. However it is seen that the socioeconomic status has inverse relationship with the incidence of eclampsia. Lower socioeconomic status appears to be a risk factor for renal

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pathology. After MRCP, patient was diagnosed as a case of distal CBD transection and/or stricture with proximal dilatation.Then, ERCP was done with the intention of confirmation of the diagnosis as well as therapeutic intervention for management of bile duct injury. Ultimately the patient was diagnosed as a case of Choledochal cyst after ERCP, and therapeutic biliary stenting was done. She was advised for biliary reconstruction surgery as a definitive treatment of Choledochal cyst. In fact, there was no justification for repeat ERCP and re-stenting. However, the brush cytology (at repeat ERCP) report came as 'adenocarcinoma', which was false positive and was not supported after excised specimen biopsy. As choledochal cyst may go into malignant transformation due to repeated cholangitis[6], so surgeon faced difficulty in taking decision about operative procedure and outcome. Fortunately, the biopsy report (from excised cyst) was chronic inflammatory change without having any malignant cell or granuloma.

So, this case report demonstrates the difficulties of pre-operative diagnosis, diagnostic dilemma, leading to inappropriate decision, all of which are responsible for the sufferings of the patient as well as huge financial burden.

Conclusion:

Choledochal cyst is a rare congenital anomaly of the intrahepatic or extra-hepatic biliary tract. Most of the patients are diagnosed in childhood with classical symptoms. Adult case diagnosis is very difficult as classical symptoms are usually absent. So choledochal cyst should be considered in the differential diagnosis in all patients with a history of biliary colic, intrabiliary calculus, mechanical jaundice and dilatation of bile duct, specially in younger patients. Delay in the diagnosis increases the frequency of associated biliary pathology, malignant alteration and suboptimal surgical therapy.

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failure in the eclamptic patients[3,8]. It may be due to pre-existing subclinical renal disease in this class of patients which has not been detected before.

As like the present study[17] found that the eclamptic patients with the renal impairment had significantly more proteinuria than the eclamptic patients with normal renal function. So degree of proteinuria may act as a predictor for impaired renal function in the patients with eclampsia. The investigators also found systolic and diastolic blood pressure in the impaired renal function group were significantly higher than the systolic and diastolic blood pressure of normal renal function group ($p<0.05$) and platelet count was significantly lower in impaired renal function group compared to the normal renal function group ($p<0.01$). Activation of haemostatic system has been documented in eclampsia and its most common manifestation is thrombocytopenia. Toxaemia has been thought of a disease in which there is abnormal platelet vessel wall interaction. There was also significant difference in the serum uric acid level between the impaired renal function group and normal renal function group, which are closely resemble with the present study.

Activation of haemostatic system has been documented in eclampsia and its most common manifestation is thrombocytopenia. Toxaemia has been thought of a disease in which there is abnormal platelet vessel wall interaction, investigators had documented impaired production of PGI_2 by the vessel wall in women with preeclampsia and eclampsia[18,19]. An imbalance between PGI_2 and thromboxane A_2 would favour platelet aggregation.

Limitation of The Study:

Due to moribund state of eclampsia patients and due to lack of facilities renal ultrasonography and renal biopsy could not be done. As the study was confined to a very limited number of hospitalized patients the conclusions may not reflect the actual situations in the community. Sometimes of the attendants of some patients were unable to respond properly to the questionnaire, as they were not relatives of the patients.

Conclusion:

The first priority in the management of eclampsia is to prevent maternal injury and to support respiratory and cardiovascular functions. During or immediately after the acute convulsive episode, supportive care should be given to prevent serious maternal injury and aspiration assess and establish airway potency and ensure maternal oxygenation. During this time, the bed side rails should be elevated and padded, a padded tongue blade is inserted between the teeth (to avoid inducing gag reflex) and physical restraints may be needed. Further large study can be undertaken by including large number of patients in multiple tertiary level hospitals.

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Review Article

Urticaria : Evaluation and Newer management options

Akter A[1], Khanam SM[2], Rahman MM[3], Alam MS[4], Mannan MA[5]

ABSTRACT

Introduction: *Urticaria(also known as hives, nettle rash) is a vascular reaction of skin(upper dermis and mucous membrane) marked by intensely pruritic, raised wheals; evanescent in character with or without edema of the deeper cutis. It is usually a self-limited, benign reaction, but can be chronic. Rarely, it may represent serious systemic disease or a life-threatening allergic reaction. Urticaria has a lifetime prevalence of approximately 20 percent in the general population. It is classified into spontaneous versus inducible types.*

Discussion: *Urticaria is mediated by mast cell degranulation. Mast cells can be activated by immunologic and non-immunologic mechanisms, which lead to degranulation of inflammatory mediators including histamine, leukotrienes, and prostaglandins. Release of these mediators causes the characteristic pruritus, vascular permeability and edema. Remission is common in majority of patients with acute spontaneous urticaria (ASU); however, in chronic cases, less than 50% had remission.*

Conclusion: *Diagnosis is made clinically. Treatment includes avoidance of triggers, although these can be identified in only 10 to 20 percent of patients with chronic urticaria. First-line pharmacotherapy for acute and chronic urticaria is nonsedating second-generation antihistamines (histamine H_1 blockers), which can be titrated to larger than standard doses. First generation antihistamines, histamine H_1 blockers, leukotriene receptor antagonists, and brief corticosteroid bursts may be used as adjunctive treatment. More than one-half of patients with chronic urticaria will have resolution or improvement of symptoms within one year.[1]*

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Introduction

Urticaria is a heterogeneous group of diseases with many subtypes. It is characterized by well-circumscribed, intensely pruritic, raised wheals (edema of the superficial skin) typically 1 to 2 cm in diameter, although they can vary in size and may coalesce; they also can appear pale to brightly erythematous (Figures 1 through 4). Urticaria can occur with or without angioedema, which is a localized, nonpitting edema of the subcutaneous or interstitial tissue that may be painful and warm. Although typically benign and self-limited, urticaria and angioedema can be symptoms of anaphylaxis, or may indicate a medical emergency. Urticaria can occur on any part of the skin. Angioedema primarily affects the face, lips, mouth, upper airway and extremities, but can occur in other locations. In both conditions, the onset of symptoms is rapid, usually occurring within minutes. Individual urticarial lesions typically resolve within 24 hours without treatment, although angioedema may take up to 72 hours.[2] Usually there are no residual lesions remaining after symptom resolution, except for possible excoriations from itching. Urticaria, with or without angioedema, can be classified as acute or chronic. In acute urticaria, although individual wheals resolve within hours, they can recur for up to six weeks, depending on the etiology. In chronic urticaria, flare-ups recur for more days but not for more than six weeks. Urticaria occurs across all age ranges and has a lifetime prevalence of approximately 20 percent in the general population, with the chronic form affecting 1 percent of the population.[3]

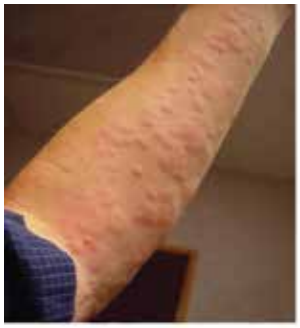

Fig. 1

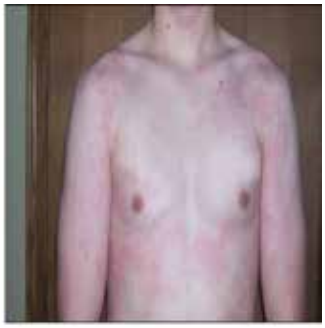

Fig. 2

Fig. 3

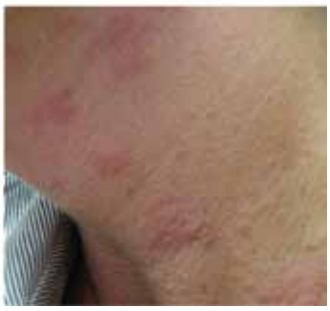

Fig. 4

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Dosage and Administration

For Adults:

Treatment Regimen		Titration Phase			Maintenance Dose
Monotherapy (Newly diagnosed patients)	Once Daily	Week 1 + 2	Week 3 + 4	Week 5 + 6	300 mg/day. maximum dose: 500 mg
		100 mg/day	200 mg/day	300 mg/day	
Adjunctive therapy	Twice daily	Week 1	Week 2	Week 3 to 5	300 to 500 mg /day (Once daily/Twice daily)
		50 mg/day	100 mg/day	Increase 100 mg/week	

For Children (6 years and above)

Titration Phase			Maintenance Dose	
(Once Daily)	Week 1	Week 2 to 8	Patients of weight 20 to 55 kg	Patients of weight >55 kg
	1 mg/kg/day	Increase 1 mg/kg/week	6 to 8 mg/kg/day (once a day)	300-500 mg/day (once a day)

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Original Article

Mortality and Morbidity Benefit of STEMI Patients after Complete ST Segments Resolution after Different Reperfusion Strategy.

Rahman MH[1], Basunia AZ[2], Afrin SF[3], Gomas HI[4] , Rahman A[5]

ABSTRACT

Background: *: Reperfusion therapy is the cornerstone for treating acute ST segment elevation myocardial infarction (STEMI). Effective reperfusion in STEMI can be achieved by either fibrinolysis or primary percutaneous coronary intervention (PPCI). PPCI generally produces better outcomes than fibrinolysis but is not widely available. ST-segment abnormalities play a fundamental role in assessment and decision making for patients with STEMI. The current theory holds that ST-segment resolution or recovery after reperfusion therapy signifies effective myocardial tissue perfusion and myocardial salvage.*

Objectives: *To compare the mortality and morbidity benefit after complete ST-segment resolution after primary PCI and fibrinolysis therapy in patients with acute ST-segment elevation myocardial infarction.*

Method: *This observational study was conducted in National Heart Foundation Hospital and Research Institute. A total 66 patients were studied and they were grouped on the basis of their treatment modality. Group I underwent primary PCI and group II received fibrinolytic therapy for acute STEMI. Mortality and morbidity benefit after complete ST-segment resolution after primary PCI and fibrinolysis (with Streptokinase) was observed.*

Result: *Regarding the morbidity, significantly higher number of patients of group II developed acute LVF (33.3% vs 6.1%, p=0.005) and cardiogenic shock (18.2% vs 3.0%, p=0.046) than group I and rescue PCI needed more in group II than group I (15.2% vs 0%, P = .020). Tamponade and stroke was not developed in any patients in both the groups after complete ST segment resolution and there was no significant difference observed between two groups in the development of bleeding from any site (15.2% vs 6.1%, p=0.230), renal failure (15.2%) vs 9.1%, p=0.451) and death (3.0% vs 6.1%, p=0.555) after complete ST segment resolution.*

Conclusion: *There was no mortality benefit but there was morbidity benefit after complete ST segment resolution after Primary PCI than fibrinolysis therapy (with Streptokinase) in patients with acute STEMI.*

Key words: *ST-Segment Elevation Myocardial Infarction; Primary PCI; Fibrinolysis; ST-resolution; Outcome.*

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Introduction:

Acute Coronary Syndrome includes ST segment elevation myocardial infarction, Non-ST segment elevation myocardial infarction and unstable angina[1]. Reperfusion therapy is the cornerstone for treating acute STEMI[2]. Effective reperfusion in STEMI can be achieved by either fibrinolytic therapy or primary percutaneous coronary intervention (PCI) without antecedent fibrinolysis (also generally known as primary angioplasty)[3].

Precordial electrocardiographic mapping, including analysis of both the ST segment and the QRS complex, which has been developed over the past several decades, is being used in studies on patients with acute myocardial infarction[4]. The standard 12 lead surface ECG is of utmost importance for the diagnosis and localization of acute myocardial infarction (AMI)[5]. It can be used to segregates the patients of AMI presenting with ST-Segment elevation from those without ST-Segment elevation[6].

Simple and rapid measures are needed for timely assessment of the quality of reperfusion therapy in acute STEMI. Although successful recanalization of the epicardial vessel is a necessary condition, it is the microvascular flow that most strongly correlates with outcome. ST-segment changes reflect myocardial rather than epicardial flow and hence yield prognostic information beyond that provided by coronary angiogram alone[7].

ST-segment abnormalities play a fundamental role in assessment and decision making for patients with AMI. Beyond determining infarct location and candidacy for acute reperfusion therapy, the extent of ST-segment elevation or deviation (sum of elevation and depression) provides important prognostic information. Early recovery of ST-segment deviation, in particular, has been shown to lower short- and long-term risk of death, recurrent ischemia, reinfarction, and congestive heart failure (CHF). This relationship is known to be robust and constitutes the basis for guideline recommendations promoting reassessment of ST segments 90 minutes after initiation of therapy and

Classification

Table-1: Classification of urticaria[4,5]

Type	Subtype	Definition
Spontaneous urticaria	Acute spontaneous urticaria	Spontaneously occurring wheals and/or angioedema for <6 Weeks.
	Chronic spontaneous urticaria	Spontaneously occurring wheals and/or angioedema for 6 weeks or longer.
Type	**Subtype**	**Precipitating factors**
Inducible urticaria or physical urticaria	Cold urticaria	Cold objects, air, fluids, wind
	Delayed pressure urticaria	Vertical pressure (stimulates wheal reaction within 3-12 hours)
	Heat urticaria	Localized heat
	Solar urticaria	Radiation from ultraviolet and/or visible light
	Symptomatic dermographism	Mechanical shearing forces (stimulates wheal reaction within 1-5 minutes)
	Vibratory angioedema	Vibratory forces such as pneumatic hammer (stimulates wheal reaction within 1-2 hours)
	Aquagenic urticaria Cholinergic urticaria	Water Increasing core body temperature
	Contact urticaria	Contact with substance that predisposes patient to wheal reaction

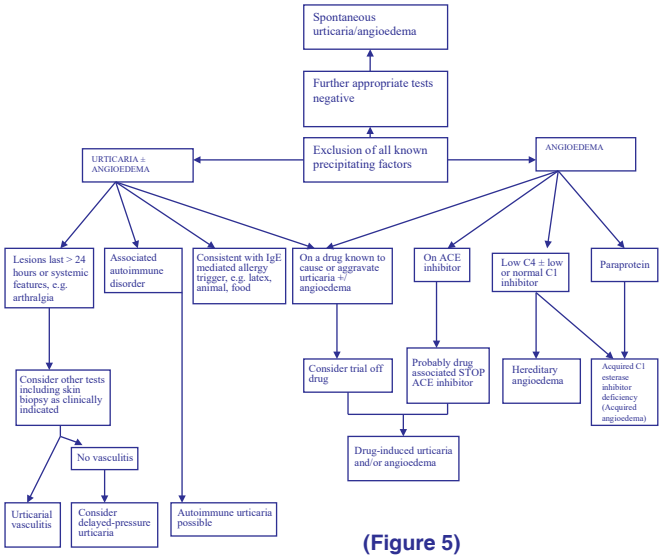
Table-2: Triggers of Acute and Chronic urticaria [6,7,8]

Classification	Acute urticaria (<6 weeks)	Chronic Inducible urticaria (>6 weeks)
Triggers	Infection	Dermographism
	Food	Cholinergic
	Medication	Cold-induced
	Venom	Solar
	Latex	Aquagenic
	Contact	Vibration
	Idiopathic	Delayed-pressure

Mechanisms:

Urticaria is mediated by mast cell degranulation. Mast cells can be activated by immunologic and non-immunologic mechanisms, which lead to degranulation of inflammatory mediators including histamine, leukotrienes, prostaglandins and cytokines, chemokines. Release of these mediators causes the characteristic pruritus, vascular permeability and edema.

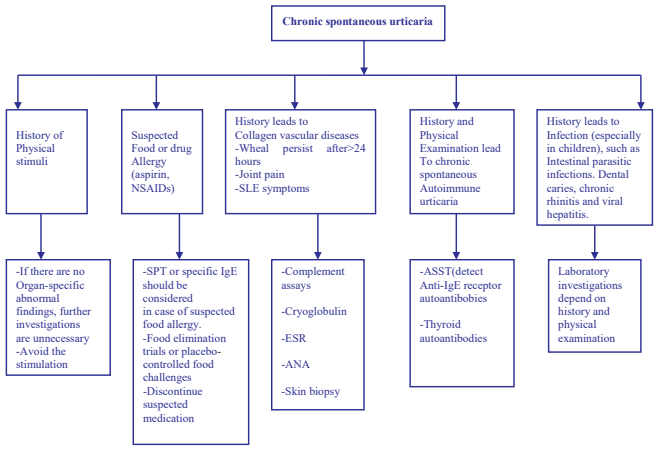
Diagnostic approach for urticaria[9]:



(Figure 5)

Investigations:

The diagnosis is usually made clinically and on history, particularly in acute urticaria and no investigations are needed. In chronic or recurring cases tests may include:[10,11]



(Figure 6)

Guidelines for management of patients with urticaria (Figure 7,8)[12-18]

1. Treating the underlying causes: If the cause can be identified, eliminate the cause. For example- drug, food, infection.[19]

2. Non-pharmacotherapy to minimize skin hyper-responsiveness:

2.1 Prevention of and care for dry skin

It is recommended to regularly apply cream or lotion without perfume to keep the skin moist and reduce skin sensitivity.

2.2 Avoidance of skin stimulation

Precipitating factors, such as scratching, wearing tight clothes, carrying heavy objects, friction massage, steam and hot vapor, body scrub, using perfume, heavy sun exposure, and exposure to too hot or too cold temperatures should be avoided.

3. Medical treatment

3.1 Antihistamines

H_1 -antihistamines are commonly used to control the symptoms of urticaria. There are 2 generations of antihistamines, including:

A. Second-generation (non-sedating) antihistamines:

Cetirizin, Levocetirizin, Fexofenadin, Loratadine, Desloratadine, Rupatadine[4,5,20] are long acting drugs. These are considered first line therapy. For better symptom control, the medication should be dosed daily. Treatment guidelines suggest that if normal doses are not successful, titration up to two to four times the usual dose in the next step.

B. First-generation (sedating) antihistamines:

Chlorpheniramine, Diphenhydamine, Cyproheptadine, Hydroxyzine[4,5,20]. The common side effects of this generation of antihistamines are drowsiness, sedation and dry mouth. EAACI/GA2LEN/DEF/WAO Guideline 2013 recommends the use of first-generation (sedating) -antihistamines only when second-generation non-sedating antihistamines are not available.

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