# A Clinical Study of Acute Meningitis in Adults in Rangpur Medical College Hospital

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#### Abstract

**Background:** Meningitis is an inflammatory condition involving the meninges of the brain and spinal cord. Acute meningitis is global burden of disease, has a high case-fatality rate and survivors can have severe lifelong disability. It can have infectious causes, such as bacteria, mycobacteria, viruses, fungi, or parasites, or be associated with autoimmunity, cancer, or reactions to medication. Risk factors that predispose individuals to meningitis and epidemics include malnutrition, household overcrowd-ing, HIV infection, absence of immunisation, indoor air pollution, and sickle cell disease. Bacterial meningitis can rapidly become fatal and lead to severe disability in those who survive. Purpose of this study was clinical study of acute meningitis in adults.

**Materials and methods:** This cross sectional observational study was conducted in Department of medicine, Rangpur Medical College Hospital, among the 100 patients who presented with sign symptoms of meningitis. Detailed history, clinical examination and thorough investigations were done and the findings were recorded on a proforma. Data was processed and analysed with the help of computer program SPSS and Microsoft excel.

**Results:** The mostly affected groups were 16-20 and 31-40 years. Mean age was 33.04 (±18.08 SD) years. There was an overall male preponderance with a male to female ratio 2.12:1. Fever headache, and vomiting was most common symptoms. Altered consciousness was present in 80% of cases, focal neurological deficit and rash were present in 10% cases. CSF findings are the hallmark of diagnosing various types of meningitis. Colour of CSF, glucose and protein concentration of CSF found in this study correlates with maximum authority. On evaluation of aetiology, 66% cases were due to bacteria and 24% and 10% cases were tuberculous and viral aetiology respectively. Outcome of pyogenic meningitis was good and mortality was 6%. Inspite of treatment 8 out of 24 (33%) tuberculous meningitis patient's were died. One case of bacterial and one case of viral meningitis were missed during follow up.

**Conclusion:** Meningitis is foremost causes of morbidity and mortality. It is recommended that provision of proper health care support, rapid detection and other investigation facilities reduce the disability.

Key words: Clinicopathological evaluation; CSF; Meningitis.

## INTRODUCTION

Meningitis remains a serious global health problem. The term meningitis describes inflammation of membranes (Meninges) and/or CSF that surrounds brain and spinal cord. It is a global burden of disease, has a high casefatality rate and survivors can have severe lifelong disability. Incident cases globally increased from 2.50 million (95% UI 2.19-2.91) in 1990 to 2.82 million (2.46-3.31) in 2016.<sup>1</sup> Bacterial

meningitis is the most common and notable infection of the central nervous system, can progress rapidly, and can result in death or permanent debilitation. Not surprisingly, this infection justifiably elicits strong emotional responses and, hopefully, immediate medical intervention. The advent and widespread use of antibacterial agents in the treatment of meningitis have drastically reduced the mortality caused by this disease.<sup>2</sup> Bacterial meningitis is a neurologic emergency, progression to more severe disease reduces the patient's likelihood of a full recovery. A blood culture and lumbar puncture should be performed immediately to confirm the diagnosis.<sup>3</sup>

Streptococcus pneumoniae and Neisseria meningitidis are the most common and most aggressive pathogens of meningitis. Clinical and experimental studies have established a more detailed understanding of the mechanisms resulting in brain damage, sequelae and neuropsychological deficits.<sup>4</sup> During the last several years, much has been learned about the pathogenesis of meningitis. At any given time, the following is a brief presentation of current knowledge of the subject. Some bacteria that cause meningitis have pili that allow the bacteria to attach to specific mucosal cells and, subsequently, to colonize mucosal surfaces of the nasopharynx. The distribution of specific mucosal and epithelial cell receptors probably determines the sites of colonization. This concept has been proposed most convincingly for Haemophilus influenzae and Neisseria meningitidis.<sup>5,6-9</sup> Viral meningitis and / or meningo encephalitis are predisposed to seasonal as well as environmental factor. But such co-relation is present neither in cases of Herpes simplex encephalitis nor in bacterial meningitis.

The portals of entry for bacteria capable of causing meningitis and the mechanisms by which entry is gained are not well understood. The portals of entry probably are sites at which the bacteria actively (By direct invasion with or without damage to the host cells) or passively (By phagocytosis) enter subepithelial tissues and, subsequently, enter the blood circulation. N. meningitidis is known to be phagocytized by nasopharyngeal epithelial cells.8 While in the blood circulation, the bacteria that cause meningitis must avoid being phagocytized by polymorphonuclear leukocytes and reticuloendothelial cells and must avoid being lysed by complement and specific antibody. Eventually, the bacteria enter the subarachnoid space and, thus, the CSF. The most likely portals of entry into the subarachnoid space are areas of minimal resistance such as choroid plexuses; dural venous sinuses; the cribriform plate; cerebral capillaries; sites of surgical, traumatic, or congenital central nervous system defects; or sites of parameningeal infection (e.g., epidural abscess)<sup>6</sup>.

The most common etiologic agents of acute meningitis are enteroviruses (Primarily echoviruses and coxsackieviruses) and bacteria (Streptococcus pneumoniae and Neisseria meningitides. Patient age and other factors (ie, immune status, post neurosurgery, trauma) are associated with specific bacterial pathogens.<sup>10</sup> Almost all bacteria that are pathogenic to man have the potential to produce meningitis, but it is unclear why a relatively small number of pathogens account for most cases of meningitis. For example,group B Streptococcus, the E. coli K1 strain and Listeria monocytogenes are the main causes of neonatal meningitis, and S. pneumoniae, Neisseria meningitidis and Haemophilus influenza type b cause most cases of meningitis after the neonatal period.<sup>11</sup>

In tuberculous meningitis, infection seeds through the blood stream from the primary lesion or site of chronic infection. Infection of meninges results from rupture of a microtubercle with discharge of tuberculoprotein and micobacteria into the subarachnoid space. These events will be marked by an episode of fever and meningeal irritation caused by the intrathecal tuberculin reaction. Subacute inflammation, specially of the basal meninges produce cranial nerve lesions, cerebral arteritis causing infarction, impairment of CSF absorptions or obstruction of the CSF circulation causing hydrocephalus, in the spinal cord, spinal arachnoiditis, producing multiple radiculopathy or myelopathy.

#### MATERIALS AND METHODS

This descriptive, cross sectional study was conducted in Department of Medicine, Rangpur Medical College Hospital, Rangpur, from 1st January, 2010 to 31 December,2010. Total 100 meningitis patients were selected. Detailed history, physical examination, neurological examination with others systemic examination and essential investigations like CBC, RBS, Serum creatinine, Chest X ray, CSF study were done in every patient. Patients with features of fever, headache, vomiting, meningeal irritation, e.g. neck rigidity, positive Kerning's sign and/or positive Brudzinskis sign considered meningitis.

Lumber puncture was done as early as possible. CSF and blood were sent for laboratory study. Other supportive investigations were done accordingly. Data of the cases were collected through regular follow up. Diagnosis was based on correlating clinical

finding with results of investigations and therapeutic response. All the information were recorded in a structured questionnaire. Data was checked and rechecked for omissions, inconsistencies and improbabilities. Data analysis was performed by Statistical Package for Social Science (SPSS), version-22. Data was edited, coded and entered in to the computer. Statistical analyses were done and level of significance was measured by using appropriate hypothetical testing. Level of significance (p value) is set at 0.05 and confidence interval at 95%. Results were presented as text, tables and diagram.

#### RESULTS

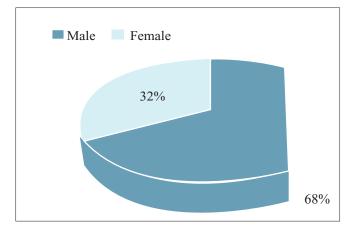
Total of 100 patients fulfilling inclusion/exclusion criteria were studied. Results and observations are given below:

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#### Table I Age distribution of patients (n=100)

Age (years)	Number of patients	Percentage (%)
16-20	32	32
21-30	18	18
31-40	32	32
41-50	12	12
51-60	2	2
>60	4	4
$Mean \pm SD$	28.1±5.8	

Table I shows demographic profile of patients. In this series, the maximum number of patients (34%) was 16-20 and 31-40 years age group. Mean age of study population was  $28.1\pm5.8$  year (Table I). Most of the patients were male in sex (68/100, 68%) with male to female ratio of 2.12:1 (Figure 1).



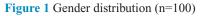


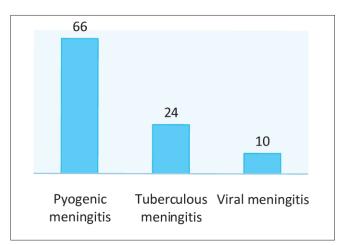
Table II shows the clinical presentation. All patients (100%) faced their crisis with acute onset fever, and then 98% of cases presented with headache, then altered mental status (80%) of cases. Present study also gives impression that vomiting, focal neurological deficit & new onset seizure also the commonest presentation. Clinical sign (Table III) revealed that, nuchal rigidity in 90.0%, positive kernig's sign in 86.0%, Brudziski's sign in 30.0% of patients.

## Table II Clinical symptoms of patients (n=100)

Presentation	Number	Percentage (%)
Fever	100	100
Headache	98	98
Vomiting	94	94
Altered consciousness	80	80
Focal neurological deficit	10	10
Seizure	10	10
Skin rash	8	8

Table III Clinical sign of patients (n=100)

Presentation	Number	Percentage (%)
Fever	90	90
Nuchal rigidity	90	90
Positive kernig's sign	86	86
Altered consciousness	74	74
Brudziski's sign	30	30
Cranial nerve lesion	14	14
Extensor planter	10	10



**Figure 2** Distribution of the patients according to type of meningitis (n=100)

Among the 100 cases of meningitis, 66.0% cases were diagnosed as pyogenic meningitis and 24.0% tuberculous meningitis.

## Table IV CSF study findings (n=100)

CSF study	Pyogenic	Tuberculous	Viral
Physical findings Clear	6	20	10
Turbid	60	4	0
Average glucose mg/dl	34.0	39.6	80.0
Average protein mg/dl	197.6	164.5	89.9
Average cell count/ mm	<sup>3</sup> 4142	236	62
	(Mostly	(Mostly	(Lymph
neu	trophilic)	lymphocytes)	ocytes)

Table IV shows the CSF study findings. Outcome of meningitis (Table V) revealed that high mortality in tuberculous meningitis (about 33%).

## Table V Outcome of meningitis (n=100)

Outcome	Pyogenic	Tuberculous	Viral
Cured	60	16	8
Expired	4	8	0
Unknown	2	0	2

## DISCUSSION

Infection of CNS is a medical emergency. The incidence of bacterial meningitis in the USA is >2.5 per 100000 people per year. The exact incidence of viral meningitis is impossible to determine since most cases go unreported to public health authorities. There is no exact report yet in our country. So it is reasonable to study on this health problem in our set up.

In this study hundred consecutive cases were recruited. The mostly affected groups were below 40 years of age comprising 82%. The maximum age incidence in meningitis of this study was between 16-20 and 31-40 (n=32 in each, 32%). There is an overall male preponderance with a male to female ratio

2.12:1 (n=68 vs n=32). The male preponderance in this hospital based study is most probably due to wide exposure to the various of infective agents and avoidance of hospital admission by female in this series maximum patient's were presented with fever, (100%), headache/nuchal rigidity, (98%) and vomiting (94%). Altered consciousness was present in about 80% of patient's. 10% patient had with focal neurological deficit and seizure, 8% patient presented with skin rash which were meningococcal meningitis. Maximum patient's had components of classical triads of meningitis (i.e. fever, headache and nuchal rigidity). These findings correlate with the study done by Marlene L. Durand, Stephen B.and et al.<sup>12</sup>

In this series examination of patients reveal maximum patient's had fever, (90%), neck rigidity (90%) and positive Kernig's sign. Brudzinski's sign was present in only 30 (30%), patient. Cranial nerve palsy was present in 14 (14%) patient of which most frequent were  $6^{th}$  nerve and common etiology was tuberculous meningitis which correlates with the study done by G.E. Thwaites and et al.<sup>14</sup>

Incidence of pyogenic meningitis was 66% (n=66), Tuberculous meningitis was 24% (n=24) and viral meningitis was10% (n=10). In cases of pyogenic meningitis maximum prevalent organism was meningococcus 66.6% (n=44), second organism was pneumococcus

27.27% which correlates with various study.<sup>13,14</sup> About the etiological agents of adult bacterial meningitis, I failed to identify organism in 4 cases of bacterial meningitis.

Colour of CSF was turbid in maximum cases of bacterial meningitis and clear in maximum cases of tuberculous meningitis and all case of viral meningitis. In this series CSF glucose concentration was very low (34mg/dl) and protein concentration was high (197.6 mg/dl) in case of pyogenic meningitis. In tuberculous meningitis the finding was more or less same. In viral meningitis CSF glucose and protein was more or less normal. These findings correlates with result of other finding.<sup>15-17</sup>

CSF cell count was very high (Average >4000/mm<sup>3</sup>) in case of pyogenic meningitis of which most were neutrophils. Cell count in case of tuberculous meningitis modest (average 236/mm3) of which most were lymphocytes. These findings correlate with the findings of maximum authority.<sup>14,15</sup>

In maximum cases of pyogenic meningitis there was neutrophilic leukocytosis and average total leukocyte count was about 15000/mm3 which slightly differ from study done by G.E Thwaites (>15000/mm3)<sup>14</sup>. Hospital outcome of treated case of pyogenic meningitis was good and mortality was 6.67%. Inspite of treatment 8 out of 24 (33.3%) of tuberculous meningitis patient's was died. Outcome of viral meningitis was satisfactory. These findings correlate with findings of various researchers.

#### CONCLUSIONS

The diagnosis of meningitis can be very challenging for emergency physicians, and it requires combining historical and physical examination findings to form an overall clinical impression. Many patients with disease will not present with classic signs and symptoms, and physical examination alone has been shown to be insufficient to diagnose meningitis. Lumbar puncture should therefore be utilized when sufficient diagnostic uncertainty remains, and caution should be exercised when interpreting results to differentiate likely viral versus bacterial disease. Systemic steroids are an important adjunctive treatment for bacterial meningitis and should be administered along with the first doses of broad spectrum empiric antibiotics in the emergency department.

## DISCLOSURE

All the authors declared no competing interest.

## **REFERENCES**

- GBD 2016 Meningitis Collaborators. Global, regional, and national burden of meningitis, 1990–2016: a systematic analysis for the Global Burden of Disease Study 2016. Lancet. 2018; 17(12): 1061-1082.
- 2. Gray LD and Fedorko DP. Laboratory diagnosis of bacterial meningitis. Clinical Microbiology Reviews. 1992; 130-145.
- 3. Smith L. Management of Bacterial Meningitis: New Guidelines from the IDSA. Am Fam Physician. 2005;71(10):2003-2008.
- 4. Hoffman O and Weber J. Pathophysiology and treatment of bacterial meningitis. Ther Adv Neurol Disord. 2009; 2(6): 401-412.
- Guerina, N. G., S. Langermann, and H. W. Clegg. Adherence of piliated Haemophilus influenzae type b to human oropharyngeal cells. J. Infect. Dis. 1982;146:564.
- McGee, Z. A., and J. R. Baringer. Acute meningitis, In G. L. Mandell, R. G. Douglas, and J. E. Bennett (ed.), Principles and practice of infectious diseases, 3rd ed. Churchill Livingstone, New York. 1990;741-755.
- McGee, Z. A., D. S. Stephens, and L. H. Hoffman. Mechanisms of mucosal invasion by pathogenic Neisseria. Rev. Infect. Dis. 1983;5:708S714S.
- 8. Stephens, D. S., L. H. Hoffman, and Z. A. McGee. Interaction of Neisseria meningitidis with human nasopharyngeal mucosa: attachment and entry into columnar epithelial cells. J. Infect. Dis. 1983;148:369-376.
- Stephens, D. S., and Z. A. McGee. Attachment of Neisseria meningitidis to human mucosal surfaces: influence of pili and type of receptor cell. J. Infect. Dis. 1981;143:525532.
- Baron E, Miller M, Weinstein M, Richter S, Gilligan P, Thomson Jr T et al. A Guide to Utilization of the Microbiology Laboratory for Diagnosis of Infectious Diseases: 2013 Recommendations by the Infectious Diseases Society of America (IDSA) and the American Society for Microbiology (ASM). Clinical Infectious Diseases. 2013;1-93.
- 11. Kim K. Pathogenesis of Bacterial Meningitis: From Bacteremia to Neuronal Injury. Nature Reviews. 2003; 4: 376-385.
- 12. Marlene L. Durand, Stephen B. Calderwood, David J. Weber, et al. Acute Bacterial Meningitis in Adults -- A Review of 493 Episodes. N Engl J Med. 1993; 328:21-28.
- Thwaites GE, Chau T, Stepniewska K, Phu NH, Chuong LV, Sinh D et al. Diagnosis of adult tuberculous meningitis by use of clinical and laboratory feature. The Lancet. 2002; 360(3): 1287-1292.
- Kaen L.Roos, Kenneth L. Tyler. Meningitis, Encephalitis, Brain abscess, Empyema. In Fauci AS, Braunwald E, Kasper DL, et al. 17th edition. Harrison's Principles of Internal Medicine. New York: The McGraw- Hill Companies. 2008;2621-2641.
- C.M.C. ALLEN, C.J. LUECK, M. DENNIS. Neurological disease. In: Nicholas A. Boon, Nicki R. Colledge Brian R. Walker, 21th edition. Davidson's Principles & Practice of Medicine, Churchill Livingstone Elsevier. 2006;1224-1228.
- 16. Logan SA, MacMahon E. Viral meningitis. BMJ. 2008; 336: 36-40.
- 17. Spanos A, Harrell FE, Durack DT. Differential diagnosis of acute meningitis. An analysis of the predictive value of initial observations. JAMA. 1989; 262: 2700-2707.