



CODEN [USA]: IAJPBB

ISSN: 2349-7750

**INDO AMERICAN JOURNAL OF
PHARMACEUTICAL SCIENCES**<http://doi.org/10.5281/zenodo.1341239>Available online at: <http://www.iajps.com>

Research Article

**ANALYSIS OF NEUROLOGICAL SYMPTOMS AND SIGNS OF
CNS TUBERCULOSIS IN PAKISTAN**M.Husnain Nabi¹, Tooba Iqbal², Iqra Khan³¹Medical Officer at BHU mochi wali, Muzaffargarh.²Woman Medical Officer at DHQ hospital, Muzaffargarh.³Woman Medical Officer at Jinnah burn and reconstructive surgery centre Lahore

Source(s) of support in the form of grants, equipment, drugs, or all of the above: None.

Conflict of interest: None

Abstract:

Introduction: Tuberculosis can affect any tissue or system of body. Common presentations of tuberculosis are primary complex, tuberculous lymph adenitis and progressive primary disease. Due to introduction of BCG vaccination just after birth the clinical pattern of tuberculosis has changed. **Aims and objectives:** The basic aim of the study is to analyze the neurological symptoms and signs at the presentation with CNS tuberculosis. **Methodology of the study:** This study was conducted in the Paediatric Department of DHQ hospital Muzaffargarh. In this study 50 children aged from 6 month to 13 years suspected of having tuberculous meningitis were included in the study. **Results of the study:** A total 50 patients were included in the study. Out of these 50 patients 32 patients were in highly probable TBM group and 18 cases in group II (Probable TBM) and 3 patients belonged to group III (Possible TBM). Out of these 50 patients 27(50.94%) patients were male and 23 (49.05%) were females with male to female ratio of 1.03:1. **Conclusion:** It is concluded that early recognition and timely treatment of CNS TB is critical if the considerable mortality and morbidity associated with the condition is to be prevented.

Key words: TB, Diseases, BCG**Corresponding author:****Dr. M.Husnain Nabi,**

Medical Officer at BHU mochi wali,

Muzaffargarh, Pakistan

E-mail: husnainnabi09@gmail.com

QR code



Please cite this article in press M.Husnain Nabi et al., *Analysis of Neurological Symptoms and Signs of CNS Tuberculosis in Pakistan.*, Indo Am. J. P. Sci, 2018; 05(08).

INTRODUCTION:

Tuberculosis can affect any tissue or system of body. Common presentations of tuberculosis are primary complex, tuberculous lymph adenitis and progressive primary disease. Due to introduction of BCG vaccination just after birth the clinical pattern of tuberculosis has changed. No age is immune to tuberculosis. It may affect any age ranging from intrauterine to upper limit of paediatric age. Incidence of infection increases as the age advances [1]. Tuberculosis is an ancient disease that is known to have existed in prehistoric times. Tuberculosis is one of the commonest communicable diseases in a majority of the developing countries [2]. It is caused by the *Mycobacterium tuberculosis*, which usually affects the lungs but may cause lesion in any organ or tissue of human body. Among infectious diseases tuberculosis is at present the leading cause of death. In 1990, 1.7 billion persons, (1/3 of the world population) were infected with *Mycobacterium tuberculosis*. Eight million new cases of tuberculosis (pulmonary and extrapulmonary) occur yearly with 2.9 million deaths [3].

In Pakistan, tuberculosis is generalized and wide spread. There have been two prevalence surveys conducted in 1960-62 and 1974-78 with similar results. According to these surveys 54% of the entire population is infected and this infection rate goes as high as 80% in age groups of 20-29 years and above. According to these surveys infection rate in children from 0-14 years of age was 25% in 1960-62 and 22% in 1974-78, 1.6% of the population above 10 years of age had chest radiograph suggestive of active

cavitary or non cavitary pulmonary tuberculosis and 0.3% were sputum positive on microscopy and/or culture [4].

Neurotuberculosis is one of the serious complications of primary tuberculous infection. Tuberculous meningitis is its most dreaded form and is the main cause of death and disability in children [5]. Tuberculous meningitis (TBM) the most dangerous form of extra pulmonary tuberculosis occurs in 7-12% of tuberculosis patients in developing countries. In 1985, 5% of 4000 extra pulmonary cases of tuberculosis in the USA were due to tuberculous meningitis [6]. Tuberculous meningitis remain a common treat to health. Outcome in tuberculous meningitis is strongly associated with the stage of disease at presentation [7]. The incidence of residual neurological handicap or death rises steeply where appropriate treatment is not initiated until after the emergence of reduced conscious level and focal neurological signs. Delay in diagnosis is directly related to poor outcome [8].

Aims and objectives

The basic aim of the study is to analyze the neurological symptoms and signs at the presentation with CNS tuberculosis.

METHODOLOGY OF THE STUDY

This study was conducted in the Paediatric Department of DHQ hospital Muzaffargarh. In this study 53 children aged from 6 month to 13 years suspected of having tuberculous meningitis were included in the study.

Table 01: Tuberculosis Prevalence Survey in Pakistan Table giving Age specific percentages of Infection in two surveys by Tuberculin Reaction (with no BCG Lesion)

Age	Survey (1961-62)	Survey (1974-78)
0-4	4.8	3.4
5-9	23.1	13
9-14	47.7	45.5
15-19	70.5	67.9
20-24	78.3	82.2
25-29	78.3	82.1
30-34	80.6	87
35-39	80.6	86.7
40-44	82.36	90.5
45-49	82.6	90.8
50+	82.2	88.3
Total	54.7	5

RESULTS OF THE STUDY:

A total 53 patients were included in the study. Out of these 53 patients 32 patients were in highly probable TBM group and 18 cases in group II (Probable TBM) and 3 patients belonged to group III (Possible TBM). Out of these 53 patients 27(50.94%) patients were male and 26 (49.05%) were females with male to

female ratio of 1.03:1. The variety of neurological signs and symptoms in these patients at the time of initial diagnosis is summarized in table 02. The reported length of any symptoms before admission ranged from 5 days to 4 months with the mean of 18.2 days and median of 20 days.

Table 02: Neurological symptoms or signs at the time of admission in 53 children with CNS tuberculosis

Symptoms/sign	No. of patients	Percentage (%)		
1 Fever		53		100%
2 Irritability		10		18.86%
3 Lethargy		35		66.03%
4 Unconsciousness	35	66.03%		
5 Seizures		50		94.33%
6 Weakness		10		18.86%
7 Hemiparesis		10		18.86%
8 Nuchal rigidity		30		56.60%
9 Brudzinski sign		25		47.16%
10 Kernig sign		25		47.16%
11 Deep tendon reflex abnormality	35	66.03%		
12 Hypertonia		38		71.69%
13 Hypotonia		10		18.86%
14 Babinski sign		20		37.73%
15 Full anterior fontanelle	5	9.43%		
16 Cranial nerve paresis	35	66.03%		
17 Irregular respiration	20	37.73%		
18 Posturing		20		37.73%

DISCUSSION:

Tuberculosis is still a major health hazard in children in India. Annual rate of infection (ARI) is 3%. Prevalence of active disease in the population is 15-25/1000 population, one fourth of them being bacillary or open cases of tuberculosis⁹⁻¹⁰. Thus out of total estimated 813 million population almost 15 million are infectious [11]. Incidence of tuberculosis in children depends upon the magnitude of infectious adults who form the reservoir of tuberculous disease. Children having primary tuberculosis rarely if ever infect other children. Tuberculosis is more common among the socioeconomically deprived people. It is more common in non-white population of Western countries [12]. Children under the age of 5 years have an overall case rate five times higher than rate of children between 5-14 years. The gender ratio for paediatric tuberculosis is 1:1.

Tuberculosis is more prevalent in winter and spring season in the Northern hemisphere [13]. Close contact among family members during winter and more frequent coughing produced by winter and

spring respiratory infections is a determining factor. Changes in mycobacterial disease morbidity and mortality are occurring due to HIV which predisposes to active Mycobacterial disease [14]. Incidence of tuberculosis in Pakistan is not different from other developing countries. In Pakistan tuberculous infection is generalized and wide spread. We had two prevalence surveys in 1960-62 and 1974-78 with almost similar results (Table I). According to these surveys, 54% of entire population was infected and this infection rate goes as high as 80% in age group of 20-29 years and above. Infection rate in children from 0-14 years of age was 25% in 1960-62 and 22% in 1974-78 surveys. There is average risk of 5% for evolution from infection towards disease during the years following infection [15].

Certainly the true incidence as well as trends of morbidity and mortality will continue to remain unknown unless a more useful definitive diagnostic tool and/or certain criteria are formed. It is generally assumed that the activated macrophages can kill tubercle bacilli but this has been difficult to prove

experimentally [17].

CONCLUSION:

It is concluded that early recognition and timely treatment of CNS TB is critical if the considerable mortality and morbidity associated with the condition is to be prevented.

REFERENCES:

1. Lurie MB, Dannenberg Jr AM. Macrophage function in infectious disease with inbred rabbits. *Bacteriol Rev* 1965; 29: 266-276.
2. Dannenbrg Jr AM. Immune mechanism in the pathogenesis of pulmonary tuberculosis. *Rev Infect Dis* 1989; (Supp 12) S369-S378.
3. Dannenberg Jr AM, Tomashefski Jr. JF. Pathogenesis of pulmonary tuberculosis In: *Pulmonary diseases and disorders*, 2nd edn. ed Fishman AP. New York, McGraw Hill 1988; 1821-1842.
4. Grange JM, Noble WC, Yates MD, Collins CH. Inoculation mycobacteriosis. *Exp Dermatol* 1988; 13: 211-220.
5. Dannenberg Jr. AM. Pathogenesis of tuberculosis native and acquired resistance in animals and humans In: *Microbiology* Washington DC. American Society for Microbiology 1984; 344-354.
6. Inove T, Yoshikai Y, Matsuzuki G, Nomoto K. Early appearing g/d bearing T cell during infection with Calmette Guerin Bacillus J. *Immunol* 1991; 146: 2754-2762.
7. Youmans GP. Relation between delayed hypersensitivity and immunity in tuberculosis. *Am Rev Resp Dise* 1975; 111: 109-21.
8. Bothamley G, Hand Grange JM. The koch phenomenon and delayed hypersensitivity tubercle 1991; 72: 7-11.
9. Unanue ER. The regulatory role of macrophage in antigenic stimulation. Part II: Symbiotic relationship between lymphocytes and macrophages. *Adv Immunolo* 1983; 13: 1-136.
10. Orme IM. The kinetics of emergence and loss of mediator Tlymphocytes acquired in response to infection with mycobacterium tuberculosis. *J Immunol* 1987; 138: 293-298.
11. John M, Grange. Tuberculosis In: Topley and Wilson's. *Principles of bacteriology virology and immunity* eds. Edward Arnold London 1990; 3: 94-121.
12. Rook GAW. The role of vit D in tuberculosis. *Am Rev Resp Dis* 1988; 138: 768-770.
13. Rook GAW, Al-Attiyah R. Cytokines and kock phenomenon. *Tubercle* 1991; 72: 13-20.
14. Khomenko AG, Litvinov VI, Chukanova V PandPospelov LE. Tuberculosis in patients with various HLA phenotype. *Tubercle* 1990; 71:187-92.
15. Moreno C, Taverne J, Mehlert A. Lipoarabinomannan from mycobacterium tuberculosis induces the production of tumour necrosis factor from human and anurine macrophages. *Cli Exp Immunol* 1989; 76: 240-245.
16. Ridley DS, Ridley MJ. Rationale for the histological spectrum of tuberculosis. A basis for classification. *Pathology* 1992; 19: 198-192.
17. Festenstein F, Grange JM. Tuberculosis and the acquired immune deficiency syndrome. *J App bacteriol* 1991; 71:19-30.