Scholars Journal of Applied Medical Sciences (SJAMS) **ISSN 2320-6691 (Online)**
Sch. J. App. Med. Sci., 2014; 2(6H):3383-3385 **ISSN 2347-954X (Print)**

(An International Publisher for Academic and Scientific Resources)
www.saspublishers.com **DOI:** 10.36347/sjams.2014.v02i06.106

Case Report

Hypogonadotropic Hypogonadism in as a Sequel of Tubercular Meningitis

Surekha Tayade[1*], Amardeep Tembhare[2]
[1]Professor, Dept of Obstetrics and Gynecology, Mahatma Gandhi Institute of Medical Sciences, Sewagram, Wardha, Maharashtra, India
[2]Assistant Professor, Dept of Obstetrics and Gynecology, Mahatma Gandhi Institute of Medical Sciences, Sewagram, Wardha, Maharashtra, India

***Corresponding author**
Dr. Surekha Tayade
Email: surekhatayademgims@yahoo.co.in

Abstract: The most common cause of meningitis is Tubercular meningitis (TBM) and it leads to irreparable neurological consequences, morbidity and mortality if not treated. The usual complication of TBM in survivors are neurologic sequelae including mental retardation, sensorineural hearing loss, hydrocephalus, endocrinological disorders, cranial nerve palsies, stroke-associated lateralizing neurological deficits, seizures and coma. We are presenting here a case report of a 20 year old female who presented to us with primary amenorrhoea as a result of hypogonadotrophic hypogonadism due to pituitary stalk injury caused by childhood tubercular meningitis.
Keywords: Hypogonadotrophic hypogonadism, Primary amenorrhoea, Tubercular meningitis.

INTRODUCTION

The most common cause of meningitis is Tubercular meningitis (TBM) and if not treated in time it leads to irreparable neurological consequences, morbidity and mortality [1-5]. In India tuberculosis is a very common cause of severe morbidity and mortality. The individuals who are having the highest risk of developing TBM include young children with primary tuberculosis and patients with immunodeficiency caused by aging, malnutrition, or disorders such as HIV and cancer [2, 3].

Out of all space occupying lesions (SOL) in the brain 20% of the lesions occur only because of tuberculosis. In tuberculosis the involvement of Central nervous system (CNS) includes meninges, cerebrum, and cerebellum, but rarely, it can also involve brainstem, basal ganglia and thalamus [3].

The usual complication of TBM in survivors are neurologic sequelae including mental retardation, sensorineural hearing loss, hydrocephalus, endocrinological disorders, cranial nerve palsies, stroke-associated lateralizing neurological deficits, seizures and coma [3].

We are presenting here a case report of a 20 year old female who presented to us with primary amenorrhoea as a result of hypogonadotrophic hypogonadism due to pituitary stalk injury caused by childhood tubercular meningitis.

CASE REPORT

A 21 year old woman reported to the gynecological outpatient unit of a rural tertiary care unit with presenting complaints of not having attained menarche till date. She also had increased frequency of a micturition, 4-5 times in the night since 10 yrs. When we elicited the past history t it was found that she had suffered from tubercular meningitis with hydrocephalus with optic atrophy at the age of 5 years and had taken antitubercular therapy for the prescribed period. She also had dimness of vision in the childhood and so had taken a writer for the 10^{th} and 12^{th} standard board examinations. She had been investigated for primary amenorrhea in the past and was diagnosed as hypogonadotrophic hypogonadism. On clinical examination, the general condition of the woman was fair. She was of average height for her age. Her vital parameters were stable. She was obese with a body mass index of 23 kg/m^2. Her secondary sexual characters were not developed. She had a flat chest with small under developed breasts (Tanner Stage 1), with no axillary and pubic hairs. There was no thyroid swelling. There was no abnormal finding on per abdomen examination with no organomegaly. On local examination, the external genitalia was infantile and labia minora was not developed. Labia majora was only covering the introitus. Hymen was intact. On per rectal examination, uterus was felt as a small pea sized, knob like structure.

On biochemical investigations, her dihydro epiandrosterone sulphate (DHEA-S) level was reduced (21.88µg/dl), 17 OH progesterone was reduced (<0.10ng/ml), sex hormone binding globulin (SHBG) was normal (29.84 nmol/L), serum cortisol was normal (5.61ug/dl) androstenedione was reduced (0.39ng/ml), testosterone was reduced (2.94ng/dl), luteinizing hormone was reduced (<0.07mIU/ml), follicle stimulating hormone was reduced (1.29mIU/ml) and prolactin was normal (8.25ng/ml). Her thyroid profile (T3 = 120.9ng/dl, T4= 4.7ug/dl and TSH= 3.18mg/ml) and blood sugar levels were normal (Fasting=84 mg/dl, Post meal=112mg/dl). Her karyotype was 46 XX. The biochemical investigations confirmed hypogonadotrophic status.

On ultrasonography the uterus was grossly hypoplastic, anteverted measuring 2.42x1.26x1.56 cms and myometrial echo was under developed. The endometrial cavity was also not developed. Both ovaries were grossly hypoplastic and showed no well developed follicular structures. Her electroencephalographic study showed poorly developed P100 waveform in bilateral recordings. MRI brain study suggested hypoplasia of pituitary stalk, hypothalamus, mamillary body, optic chiasma and tuber cinerum suggesting towards sequel of tubercular meningitis.

Counselling of the woman and her relatives was done and she was started on estrogen therapy for 6 months for breast development. She was referred to tertiary eye care center for management of eye problems.

DISCUSSION

Though the prevalence of childhood tuberculosis is not rare in India, involvement of the hypothalamo-pituitary axis as a sequel of TBM is extremely rare [3]. The mycobacterium bacilli mainly affects lungs, bones and other structures but it can also reach to the brain and its appendages, hypothalamo-pituitary axis and optic chiasma via the hematogenous route or from the local spread from the skull base to cause various central nervous system manifestations [3, 4, 9].

Though the dysfunction of hypothalamus and pituitary because of tuberculosis is very rare nowadays, out of all the hypothalamo-pituitary axis involvement by tuberculosis almost 60-70% had endocrinological manifestations [3, 8]. The commonest neurological manifestations of TBM are headache, visual symptoms, anterior pituitary hypo function, hyperprolactinemia and diabetes insipidus [8, 13, 14].

Our patient had hypogonadotropic hypogonadism suggesting possibility of hypopitutarism and optic atrophy, which suggests optic nerve involvement due to tuberculoma or exudation around optic chiasma. As found in our patient optic atrophy is a common feature in suprasellar extension of sellar tuberculomas where visual field defects are seen.

Histo-pathological confirmation of pituitary tuberculoma in our patient was not necessary as the diagnosis was clearly made on the basis of clinical history, clinical presentation, biochemical and radiological findings. In spite of completing treatment for TBM in childhood, she developed neurological sequel. This is possible, as intracranial tuberculoma can develop or enlarge during antitubercular therapy due increased host reaction to the tuberculous protein [9].

The treatment for pituitary sequelae of tuberculosis is not well defined till date but various regimens have been suggested in the form of steroids but the drug, dosage and duration of use are still not standardized as there is paucity of clinical knowledge of pituitary tuberculoma [14-16].

The present case was managed with antikochs treatment for a period of one year in childhood, estrogen therapy and ophthalmic consultation for optic atrophy. She received symptomatic treatment from tertiary eye care center in the form of medications and spectacles. Counselling of the woman and relatives was done.

This case study does suggest a need to assess all individuals with primary amenorrhea for a workup for pulmonary or extra pulmonary tuberculosis. After diagnosis, the serum concentrations of LH, FSH and estradiol should be assessed periodically along with bone mineral density as a part of surveillance.

REFERENCES

1. Girgis NI, Sultan Y, Farid Z, Mansour MM, Erian MW, Hanna LS *et al*.; Tuberculous meningitis, Abbassia Fever Hospital—U.S. Naval Medical Research Unit No. 3—Cairo, Egypt, from 1976 to 1996. Am J Trop Med Hyg., 1998; 58(1): 28-34.
2. Rock RB, Hu S, Gekker G, Sheng WS, May B, Kapur V *et al*.; Mycobacterium tuberculosis-induced cytokine and chemokine expression by human microglia and astrocytes: effects of dexamethasone. J Inf Dis., 2005; 192(12): 2054–2058
3. Verdon R, Chevret S, Laissy JP, Wolff M; Tuberculous meningitis in adults: review of 48 cases. Clin Inf Dis., 1996; 22(6): 982–988.
4. Kent SJ, Crowe SM, Yung A, Lucas CR, Mijch AM; Tuberculous meningitis: a 30-year review. Clin Inf Dis., 1993; 17(6): 987–994
5. Bidstrup C, Andersen PH, Skinhøj P, Andersen AB; Tuberculous meningitis in a country with a low incidence of tuberculosis: still a serious disease and a diagnostic challenge. Scand J Inf Dis., 2002; 34(11): 811–814

6. Berenguer J, Moreno S, Laguna F; Tuberculous meningitis in patients infected with the human immunodeficiency virus. New Eng J Med., 1992; 326(10): 668–672.
7. Farer LS, Lowell AM, Meador MP; Extrapulmonary tuberculosis in the United States. Am J Epid., 1979; 109(2): 205–217
8. Sunil K, Menon R, Goel N, Sanghvi D, Bandgar T, Joshi SR *et al*.; Pituitary Tuberculosis. J Assoc Physicians Ind., 2007; 55: 453-456.
9. Jain R, Kumar R; Suprasellar tuberculoma presenting with diabetes insipidus and hypothyroidism-a case report. Neurol India, 2001; 49(3): 314-316.
10. Henry M, Hlzman RS; Tuberculosis of the brain, meninges, and spinal cord. In Rom WN, Garay SM et al. editors; Tuberculosis. 2nd edition, Lippincott Williams & Wilkins, Philadelphia, Pa, USA, 2004: 445–464.
11. Garg SK, Bandyopadhyay PK, Dash RJ; Hypogonadotropic hypogonadism. An unusual complication of tuberculous meningitis. Trop Geogr Med., 1987; 39(3): 296-298.
12. Singh S; Pituitary tuberculoma: Magnetic resonance imaging. Neurol India, 2003; 51(4): 548-550.
13. Ahmad FU, Sarat Chandra P, Sanyal S, Garg A, Mehta VS; Sellar tuberculoma: An unusual infection. Indian J Tuberc., 2005; 52: 215-217.
14. Sundar US, Ramteke VV, Vaidya MS, Asole DC, Moulick ND; Suprasellar tuberculoma presenting as panhypopituitarism. J Assoc Physicians India, 2010; 58: 706-709.
15. Ashkan K, Papadopoulos MC, Casey AT, Thompson DN, Jarvis S, Powell M *et al*.; Sellar tuberculoma: Report of two cases. Acta Neurochir (Wien), 1997; 139(6): 523-525.
16. Mageshkumar S, Patil DV, Philo Aarthy JA, Madhavan K; Hypopituitarism as unusual sequelae to central nervous system tuberculosis. Case Report. Ind J Endocrinol Metabol., 2011; 15(7): 259-262.