DOI: 10.7860/JCDR/2016/18782.8053

Case Report

Obstetrics and Gynaecology Section

Intracranial Tuberculoma Presenting as Atypical Eclampsia: A Case Report

SENDHIL COUMARY ARUMUGAM[1], SHARMILA MURUGESAN[2], SUNITHA PRADEEP[3], LOPAMUDRA JOHN[4], VASAVI KOLLURU[5]

ABSTRACT

Occurrence of eclampsia before 20 weeks of pregnancy and after 48 hours of delivery in the absence of typical signs of hypertension and or proteinuria is termed as atypical eclampsia. Atypical or non-classic eclampsia will have some symptoms of eclampsia but without the usual proteinuria or hypertension. All patients with atypical onset should undergo neurological evaluation to rule out neurologic causes of seizures. Cerebral tuberculosis is a rare and serious form of disease secondary to haematogenous spread of *Mycobacterium tuberculosis*. Here we present a case of cerebral tuberculoma with seizures in late pregnancy mimicking eclampsia.

Keywords: Cerebral tuberculoma, Hypertension, Proteinuria, Seizures

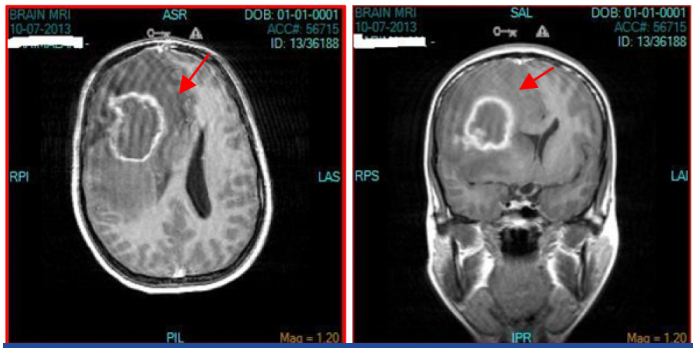
CASE REPORT

A 24-year-old second gravida presented to the antenatal clinic. This was her first visit to the hospital. She was 28 weeks pregnant. She was brought by her mother for abnormal behaviour. The patient was reported to have features of disinhibition (dressing in appropriately in public) and difficulty sustaining attention. She was also becoming extremely forgetful. However, the patient only complained of severe unilateral headache since two months and did not have any insight about these changes in her. There was no aura or vomiting associated with the headache. Her previous pregnancy was five years ago with normal outcome.

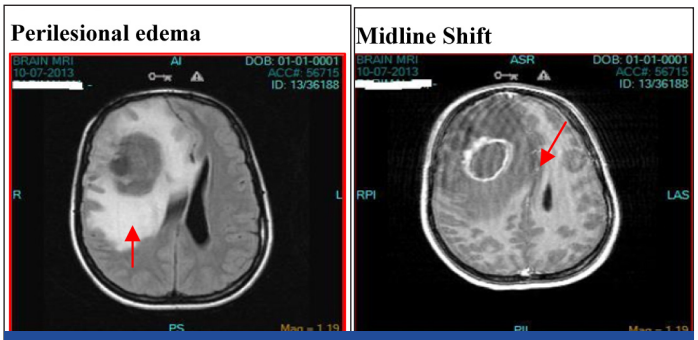
Her general physical examination, obstetric examination and preliminary blood and urine investigations were normal. She was referred to the headache clinic and for psychological assessment to analyse her behavioural change. Ophthalmic examination of the retina showed bilateral papilloedema. Suspecting intracranial mass lesions, she was advised for further imaging investigations. She refused a psychological consultation and was then lost to follow-up.

She then presented to the emergency, at term pregnancy with seizures in early labour. She did not have any hand-held medical records. A diagnosis of antepartum eclampsia was made and she was started on Pritchard regimen. Her blood pressure remained normal and urine albumin was traced and therefore it was considered atypical eclampsia. An urgent ophthalmic opinion revealed bilateral papilloedema. The intrapartum period was short and managed uneventfully. She delivered a healthy neonate vaginally within four hours of admission with an anaesthetist standby.

Postnatally, a contrast enhanced MRI was done. It showed a single, large, round to oval shaped lesion in the right frontal lobe. It appeared hypointense on T1 weighted and FLAIR images and hyperintense on T2 weighted image [Table/Fig-1]. There was



[Table/Fig-1]: A single large, round to oval shaped lesion in the right frontal lobe appearing hypointense on T1 weighted and FLAIR images.



[Table/Fig-2]: A single large, round to oval shaped lesion in the right frontal lobe appearing hyperintense on T2 weighted image with marked perilesional oedema. **[Table/Fig-3]:** Image showing a mass effect by the lesion with compression of the frontal right lateral ventricle associated with midline shift.

marked perilesional oedema [Table/Fig-2]. Post contrast images showed an irregular rim of enhancement. There was mass effect with compression of the frontal right lateral ventricle associated with midline shift [Table/Fig-3]. There was no evidence of meningeal enhancement. The most possible diagnosis of giant tuberculoma was made. The patient was then counselled for a tissue diagnosis which she refused. She was worked up in the tuberculosis clinic for any other foci of tuberculosis which turned out to be negative. She was then started on DOTS antitubercular four drug regimen (IRZE) along with dexamethasone. She responded very well to the therapy. This patient was lost to follow-up.

DISCUSSION

Eclampsia classically presents as hypertension and proteinuria with seizures and or coma after 20 weeks of gestation upto 48 hours of delivery, ccurrence of eclampsia before 20 weeks of pregnancy and after 48 hours of delivery in the absence of typical signs of hypertension and or proteinuria is termed as atypical eclampsia [1]. Atypical or non-classic eclampsia will have some symptoms of eclampsia but without the usual proteinuria or hypertension [1]. All patients with atypical onset are therefore advised to undergo neurological evaluation to rule out neurologic causes of seizures.

Previously undiagnosed space occupying lesions form an important differential diagnosis for atypical eclampsia. Tuberculosis is the second greatest killer after HIV/AIDS due to a single infectious agent and continues to be an important public health problem in developing countries. About a third of the world's population has latent tuberculosis. Although all age groups are at risk it commonly affects young adults. Globally in 2013, 3.3 million women fell ill with tuberculosis, 70% of these women were from African and Southeast Asian regions [2]. Cerebral tuberculosis is a rare and

serious form of disease secondary to haematogenous spread of *Mycobacterium tuberculosis* [3].

The incidence of tuberculosis in pregnancy ranges between 1-2%. Tuberculosis among mothers is associated with a six-fold increase in perinatal mortality and a two-fold rise in prematurity and low birth weight. When associated with HIV, it increases the risk of maternal and infant mortality by 300% [2]. CNS disease caused by tuberculosis is uncommon and comprise only 10-15% of the infections. Tuberculoma is common in endemic areas but its occurrence during pregnancy is rare and of particular interest when its intriguing clinical picture mimics the toxemia of pregnancy.

Dissemination of the disease is common among immunecompromised patients. When dissemination occurs, any organ may be affected. In CNS, the infection is classified as intracranial and spinal [3]. Tuberculosis of the brain may be a part of a systemic disease or occur in isolation usually as a seeding from a silent pulmonary lesion. It may involve the meninges or parenchyma or both. The intracranial tuberculosis usually presents as meningitis and less commonly as tuberculoma [4].

The CNS disease usually begins as a small focus (Rich focus) in the brain, spinal cord and meninges. A tuberculoma forms when a tubercle in the brain parenchyma enlarges without rupturing into the subarachanoid space [5]. Intra cranial tuberculoma may be single or multiple. They may be as large as several centimeters in diameter causing significant mass effect. On microscopic examination, they show centrally caseating chronic granulomatous inflammation surrounded by a wall epitheloid cells, lymphocytes and Langhan's type of giant cells. Usually tuberculomas are surrounded by a thick fibrous wall and calcification can occur in dormant lesions [4].

Intracranial tuberculomas are usually solitary lesions, but 15-34% are multiple. They behave like space occupying lesions and present with signs of raised intracranial pressure, localized neurological deficits, seizures, and even behavioral problems. Seizures are one of the most common symptoms occurring in up to 85% of cases along with symptoms related to elevated intracranial pressure such as headache, papilloedema and lethargy as in the present case [6].

Pandole A et al., reported a similar scenario where the patient presented with tonic clonic seizures near term and had two intracranial tuberculomas which was operated upon soon after delivery [7]. Symptoms of focal mass lesion like weakness, hemiparesis and ataxia occur in 70% of patients. Nabulsi et al., reported a case of an antenatal woman from Somalia, with fulminant disseminated disease who presented with tubercular meningitis and later succumbed to the disease despite initiation of therapy [8].

Emerson et al., reported a case where an immunocompetent pregnant woman presented with features of tubercular meningitis and had 28 intracranial tuberculomas and all of which vanished after a year of anti tubercular treatment [9]. Sayed Ali Ahmadi from Iran reported a case of a pregnant migrant woman from Afghanistan who was having episodes of headache, seizures and weakness in both her pregnancies. Her MRI revealed a single large tuberculoma in the parietal lobe [10]. She was sputum negative for Acid Fast Bacilli (AFB) like the present case.

The diagnosis of tuberculoma during pregnancy is difficult because often the eclampsia becomes the presumptive diagnosis in any convulsive pregnant woman. However, a small percentage of them be could be normotensive and non proteinuric. Such patients should be worked up for neurological causes of seizures.

The standard anti-tubercular regimen has two phases; an intensive phase and a continuation phase. In the intensive phase the patient will receive 4-5 drugs lasting for 2-3 months in order to kill the fast multiplying bacilli. This is then followed by a continuation phase where the patient receives 2-3 drugs lasting for 4-5 months, during which the remaining bacilli are killed so as to prevent a relapse. Some experts advise to prolong this continuation phase to 9-12 months in cerebral tuberculosis because of the associated morbidity. All pregnant and lactating women should be advised to take the full course as antitubercular drugs are safe during pregnancy and lactation. Streptomycin is avoided in pregnancy as it is ototoxic to the fetus [11,12].

A standard course involves 2HRZE and 4HR. (H-isoniazid, R-rifampicin, Z-pyrazinamide and E-ethambutol) INH and pyrazinamide cross the blood brain barrier with ease whereas ethambutol only partially crosses the meninges. Though rifampicin crosses the meninges most of it is pumped out of the CNS. Intracranial tuberculomas grow slowly and become encapsulated. Also the local immunological reaction may induce perilesional secondary granulomatous vasculitis associated with occlusion of the vessel lumen. This further hinders the penetration of the anti-tuberculous drugs into the lesion and thereby delays complete recovery which supports the longer duration of the treatment that is required in these cases [11,12].

CONCLUSION

As atypical pre-eclampsia and eclampsia are very vague entities, it is better to keep other differential diagnoses like a space occupying lesion in mind. In patients coming from areas with a high incidence of tuberculosis a cerebral tuberculoma should be thought of even in the absence of prior history or pulmonary involvement.

REFERENCES

[1] Stella CL, Sibai BM. Preeclampsia: Diagnosis and management of the atypical presentation. *J Matern Fetal Neonatal Med*. 2006;19(7):381-86.
[2] WHO factsheet N*104. Revised March 2015.
[3] Cherian A, Thomas SV. Central nervous system tuberculosis. *African Health Sciences*. 2011;11(1):116-27.
[4] Mathew P Frosch, Douglas C Anthony, Umberto De Girolami. The central nervous system. In: Vinay Kumar, Abul K Abbas editors. Pathologic basis of the disease. 8th edition Elsevier Saunders; 2010. Pp. 1301.
[5] Rock RB, Olin M, Baker CA, Molitor TW, Peterson PK. Central nervous system tuberculosis: pathogenesis and clinical aspects, Clinical Microbiology Reviews. 2008. pp. 243-261.
[6] Muin DA, Wagner K, Burian R, Maghami NG, Lapaire O. Brainstem tuberculoma in pregnancy. case reports in obstetrics and gynecology. 2015;2015:179483. 6 pages http://dx.doi.org/10.1155/2015/179483
[7] Pandole A, Akolekar R, Vaidya N, Kore S, Ambiye VR. Tuberculoma in Pregnancy. *Bombay Hospital Journal*. 2001;43(1):200-01.
[8] Nabulsi BK, Kadi M, AlAbadi H, Alnabulsi RK, Badeghiesh A, et al. Pregnant woman with fulminant disseminated tb to the omentum and placenta. *Gynecol Obstet* (Sunnyvale) 2014:225.
[9] Gasparetto EL, Tazoniero P, de CarvalhoNeto A. disseminated tuberculosis in a pregnant woman presenting with numerous brain tuberculomas – case report. *Arq Neuropsiquiatr*. 2003;61(3-B):855-58.
[10] Ahmadi SA, Roozbeh H, Abbasi A, Bahadori M, Moghaddam KG, Ketabchi SE. Cerebral tuberculoma in pregnancy: overview of the literature and report of a case. *Acta Medica Iranica*. 2011;49(1):64-69.
[11] World health organization. Treatment of tuberculosis: guidelines 4th edition available on WHO/HTM/TB/2009.420.
[12] Tripathi KD. Anti tubercular drugs. In: K D Tripathi editor. Essentials of medical pharmacology.7th edition. Jaypee publishing house; 2013 pp. 765-79.

PARTICULARS OF CONTRIBUTORS:
1. Professor, Department of Obstetrics and Gynaecology, Mahatma Gandhi Medical College and RI, Pondicherry, India.
2. Assistant Professor, Department of Obstetrics and Gynaecology, Mahatma Gandhi Medical College and RI, Pondicherry, India.
3. Assistant Professor, Department of Radiology, JIPMER, Pondicherry, India.
4. Associate Professor, Department of Obstetrics and Gynaecology, Mahatma Gandhi Medical College and RI, Pondicherry, India.
5. Assistant Professor, Department of Obstetrics and Gynaecology, Mahatma Gandhi Medical College and RI, Pondicherry, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dr. Sendhil Coumary Arumugam,
9-Thillai Mestri Street, Pondichery-605001, India.
E-mail: sendhilcoumary1975@yahoo.in

FINANCIAL OR OTHER COMPETING INTERESTS: None.

Date of Submission: **Jan 25, 2015**
Date of Peer Review: **Feb 06, 2016**
Date of Acceptance: **Apr 26, 2016**
Date of Publishing: **Jun 01, 2016**