Gynecology & Obstetrics
ISSN: 2161-0932

Nabulsi et al., Gynecol Obstet (Sunnyvale) 2014, 4:5
DOI; 10.4172/2161-0932.1000225

Case Report Open Access

Pregnant Woman with Fulminant Disseminated TB to the Omentum and Placenta

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Abstract

This is a case report of a young 24 year old Somali woman in her 27th week of gestation who was given Rifampicin, Ethambutol, INH, Pyridoxine and Pyrazinamide as a treatment for systemic TB. She did not respond to the treatment. She died because of brainstem infarction (brain death). According to MRI results, multiple brain tuberculomas were seen suggesting brain TB. Brain biopsy was not done and the treatment was initiated at her 27th week of gestation. Patient arrested and was transferred to ICU with GCS of 3-4/15. Cesarean section was done at the 29th week of gestation and the infant was not infected. There were query tuberculosis seeding scattered all over the patient's omentum and placenta.

A specimen was taken for histopathology, which demonstrated that the placenta and omentum contained focal areas of microinfarctions and necrotizing granulomas consistent with tuberculosis. We emphasize that screening should be done during pregnancy to discover dormant infection, asymptomatic disease and to lower the incidence of congenital TB. The aggressive early treatment for dissemination of the disease, especially when associated with pregnancy, and the importance of early diagnosis and therapy will result in regression of the lesions.

Keywords: Multiple brain tuberculoma; Pregnancy; Tuberculomas; Miliary TB

Introduction

Tuberculosis is an infectious disease caused by *Mycobacterium tuberculosis,* which has high morbidity and mortality and represents a public health problem, especially in developing countries [1,2]. The greatest disease burden is during the childbearing years of 15 to 49, with 80% of all deaths from TB occurring in this group[3]. Worldwide, TB is the number one infectious cause of death among women, killing more than 1 million women each year. TB currently is responsible for more deaths annually than all other causes of maternal morbidity combined [3]. In communities in which TB is endemic, pregnant women are at high risk for getting the infection especially with resistant organisms. The most common presentation is the pulmonary form, and less commonly the extrapulmonary form which can disseminate from lungs to other organs through blood. Tuberculosis is common in immunocompromised patients, and immunodeficiency related to pregnancy severe enough to cause dissemination of the *Mycobacteria* to any organ including central nervous system, omentum and placenta which is exceedingly rare. When dissemination occurs, any organ may be affected. The central nervous system infection may manifest as meningitis, abscess or tuberculoma formation [4,5]. The most common presentation of intracranial tuberculoma is as meningitis and when it affects the brain parenchyma, it presents in the form of single lesions [5,6]. Aggressive dissemination of tuberculosis related to pregnancy is rarely reported in the literature. We present a young Somali patient which presented with wide spread disseminated tuberculosis of the lungs, omentum, placenta and fulminant neurological disseminated intracranial tuberculoma at the 27th week of gestation.

Case Report

A 24 year old Somali woman in her 27th week of gestation (G5 P3+1) Presented to KAUH complaining of: Pervaginal (PV) bleeding for one day and fever for 2 months prior to admission. The patient was in her usual state of health until 2 months back when she started to notice on/off attacks of fever associated with night sweats but she had no chills. She had loss of appetite and loss of weight, about 10 kg through the last 2 months. One day prior to her admission she had PV bleeding. The bleeding started suddenly and continued for several hours. She had no history of travel or contact with TB patients. She lives with her family and had a low income. She worked as a housewife, with no history of smoking or alcohol intake. No family history of similar illness or other illnesses. She gave a history of fainting in the bathroom for a few minutes. Systemic Review was negative except for dry cough for one month and dyspepsia. On physical examination, she was febrile 38.5°C and generally looked ill. She had slight pallor, no palpable lymph nodes. Chest, abdomen and central nervous system examination were unremarkable. She was stable along the first week after admission. However, at the end of the first week, she started to look very ill and developed a severe headache, throbbing in nature mainly in the temporal area, and she was sweating profusely. She had neck stiffness, but Kernig's and Brudzinski's sign's were negative. Non contrast CT of the brain showed multiple isodense grey matter lesions seen involving the right cerebellar hemisphere, right frontoparietal, right frontal and left frontal with surrounding low attenuated areas representing vasogenic edema with some mass effect on the fourth ventricle and overlying sulci, and no haemorrhage. The midline was central with no midline shift. The supra tentorial ventricular system is within normal. Radiologist's impression: findings are highly suggestive of an infection process in particular TB. Clinical correlation and follow up with MRI was recommended. Anti TB was started empirically as

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Received March 17, 2014; **Accepted** May 22, 2014; **Published** May 28, 2014

Citation: Nabulsi BK, Kadi M, AlAbadi H, Alnabulsi RK, Badeghiesh A, et al. (2014) Pregnant Woman with Fulminant Disseminated TB to the Omentum and Placenta. Gynecol Obstet (Sunnyvale) 4: 225. doi:10.4172/2161-0932.1000225

follow: Rifampicin 600 mg/NGT/OD, Ethambutol 1 gm/NGT/OD, INH 300 mg/NGT/OD, Pyridoxine 40 mg/NGT/OD, Pyrazinamide 2 gm/NGT/OD, Dexamethazone 8 mg/IV.

The level of consciousness started to deteriorate gradually. MRI was done Axial T1 with contrast (Figure 1), showed ring enhancing mass lesions and Axial T2 (Figure 2), showing dark mass centrally surrounded by edema. However, CSF was clear and no organisms were isolated. It was repeated on three different days. Polymerase Chain Reaction (PCR) was negative.

EEG showed generalized slowing more marked over the anterior area. Chest x-ray showed multiple rounded nodule 1-2 mm suggesting miliary TB. But sputum for Acid Fast Bacilli (AFB) was negative on three different occasions and Bronchoscopy was done and it was negative for pulmonary miliary TB. Transbronchial lung biopsy showed lung tissue with mild interstitial fibrosis. Z-N stain was negative. Bronchial wash cytology revealed suppurative exudates. HIV, HCV and Toxoplasma all were negative. Cytomegalovirus IgG was positive 6.7 IU/m1 CMV Ab-IgM was negative.

At the 3rd week the patient deteriorated and the level of consciousness was GCS 6-9. Suddenly thereafter she had cardiac arrest. She was intubated and recovered within 25 minutes and transferred to ICU [GCS 3/15], pupils non-reactive. Fetal assessment post code was satisfactory (only tachycardia). On 29th week of gestation, an emergency classical cesarean section was done due to: fetal distress and near maternal brain death. In the operating room there were query tuberculosis seeding scattered all over on the patients omentum and placenta. A specimen was taken for histopathology which demonstrated that the placenta and omentum were containing focal areas of microinfarctions and necrotizing granulomas consistent with tuberculosis. Status of the infant was normal, except for lung immaturity and the infant was intubated in the neonatal ICU. Sputum for acid fast bacilli was done 3 times and was negative. TB culture was negative. CSF was clear and no organisms were isolated. Serology: HBsAG, HCV and HIV were negative. Blood culture: Staphylococcus species (coagulase negative) was isolated. In the end of the 3rd week, she had bradycardia and she was not for code.

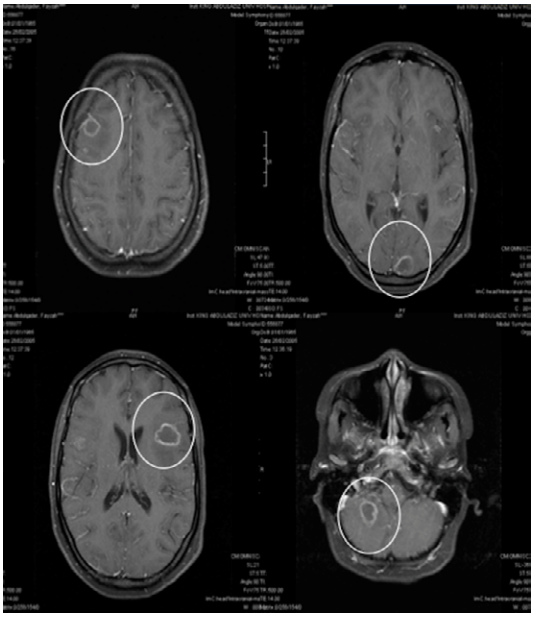

Figure 1: MRI Axial T1, Showing ring enhancing mass lesions.

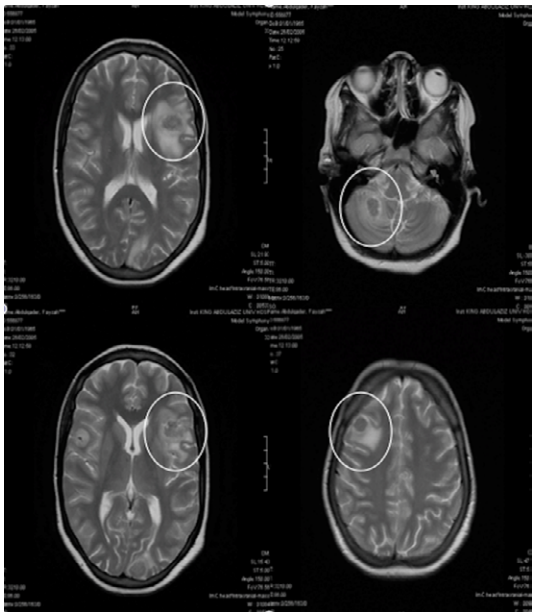

Figure 2: MRI Axial T2, Showing dark mass centrally surrounded with edema.

Discussion

The involvement of central nervous system, caused by tuberculosis, often occurs in the form of tuberculosis meningitis and tuberculoma [6]. Signs and symptoms in the clinical process of brain tuberculomas are generally silent, and the complaints gradually increase [7,8]. In our patient she presented with Pervaginal (PV) bleeding and fever which was not related to the central nervous system. In the hospital she started to deteriorate and the level of consciousness started to decline with severe headache. The disease progressed until the patient had brain death in less than 2 weeks from the date of presentation.

On T1-weighted images, granulomas typically appear as isointense–hyperintense masses with single or multiple ring enhancement, accompanied by a slightly hyperintense rim that is surrounded by a rim of slight hypointensity. On T2-weighted images, granulomas appear as isointense–hypointense masses surrounded by a hypointense rim [9]. In our case, brain MRI revealed multiple rounded lesions which appeared hyperintense centrally on T1 accompanied by more hyperintense rim surrounded by hypointensity at the periphery. While on T2, it appeared as central hypointensity with hypointense periphery (edema surrounding the lesions). MRI axial T1 suggested bacterial abscess, TB, metastasis or fungi. While MRI axial T2 suggested TB or fungi.

In one third of the cases, cerebrospinal fluid findings and clinical findings may not support the diagnosis and diagnosis needs to be confirmed by biopsy [10]. In our patient, we could not perform the biopsy because of the patient's status which was unsuitable for brain biopsy. Also, nothing was there to bear out that this is a TB case until the patient went for emergency classical caesarean section. Histopathological examination of the placenta and omentum revealed microinfarctions and necrotizing granulomas consistent with tuberculosis.

The most important factor affecting the prognosis in CNS tuberculosis and brain tuberculomas is early beginning of anti-TB therapy [11]. The probability of irreversible brain destruction, and

formation of sequel lesion increases with late initiation of the therapy [12,13]. Similarly we started anti-TB therapy in the first week of presentation but the patient presented in a very advanced stage and the TB disseminated to the omentum, placenta and the brain forming multiple lesions. Due to this, the most important thing in brain tuberculosis diagnosis is the suspicion of the disease and to hasten the diagnostic procedure. Tuberculosis shows very different clinical patterns depending on the organs it involves. CNS tuberculosis, and symptoms and signs depending on this, may or may not be seen with pulmonary involvement. And our patient chest X-ray was suggestive of military TB with no signs or symptoms for pulmonary involvement but the entire test including bronchoscope was negative for TB.

In the literature there were similar cases, except in the aggressiveness of the disease course. A pregnant Somali woman, suffered from a progressive hemiparesis, epilepsia, behavioral problems and low fever for the last five years. MRI of the brain showed multiple lesions with contrast enhancement. Extensive tests were done on serum and cerebrospinal fluid, which did not reveal any cause. A brain biopsy revealed only necrosis, but bacterial culture and polymerase chain reaction (PCR) supplied the diagnosis of 'tuberculosis'. PCR of the cerebrospinal fluid remained negative. In the meantime, the chest X-ray showed miliary tuberculosis. Subsequently the patient was treated successfully with tuberculostatic agents. Her healthy infant who was born via caesarean section was treated with the tuberculostatic agents as well [14].

Conclusion

Physicians should be aware that brain tuberculoma could occur as a relatively silent clinical event. Detection of brain involvement by contrast CT, MRI and taking a biopsy from the lesion for histopathologist confirmation is important as early as possible, since early diagnosis prevents further deterioration after prompt administration of therapy.

Acknowledgment

Special thanks goes to Abdulrahman Altaher, MD and Haleh Hashemi medical student for helping in the literature review.

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