Introduction

Tuberculous lesions of the central nervous system are endemic in the Indo-Pakistan subcontinent. Other parts of the world are also showing an increased incidence, concomitant with the spread of AIDS and related immunosuppression.

The diagnosis and treatment of intracranial TB remains challenging. This is due to its multiple patterns of presentation, mimicking brain tumors such as gliomas and pyogenic abscesses. The symptoms and signs are those of any intracranial mass lesion such as seizures, hemiparesis and cognitive disturbances. The ring enhancing pattern of tuberculomas on contrast scans is non-specific and so is the uniform enhancement sometimes seen with these lesions. Multiple lesions may be present. A history of pulmonary involvement is found in about half the cases.

The usual approach to management of intracranial tuberculomas is excision of the accessible lesions followed by chemotherapy. However in some cases the lesion is located at an inaccessible site. In such cases medical therapy is the only available option. The problem of compliance with long term medical treatment is well recognized. In addition the emergence of drug resistant strains of M. Tuberculosis is an increasing concern. This report describes a case where medical treatment alone was used to treat the difficult problem of inaccessible intracranial tuberculoma. This resulted in complete resolution of the intracranial disease.

Case Report

Clinical Presentation and Imaging

This 38 year old female presented with a one month history of generalized tonic clonic seizures and left hemiparesis. There was a family history of pulmonary tuberculosis. Examination showed grade 3/5 power in the left upper and lower limbs. There were no other neurological or systemic abnormalities. Chest X-Ray showed clear lung fields. A contrast CT scan of the brain was performed which showed a uniformly enhancing lesion above the right thalamus indenting the right lateral ventricle (Figure 1). There was significant surrounding edema.

Management and Clinical Course

The patient was started empirically on four drug anti-tuberculous regimen. This included isoniazid, rifampicin, pyrazinamide and ethambutol. After a period of four weeks the patient noted gradual improvement in her power on the left side. During this time, she remained on Carbamazapine and there were no further seizures. No further neurologic deficits were noted during the next few months. The patient returned to her normal daily activities during this time.

A CT scan was repeated six months after the start of therapy. This showed complete resolution of the lesion. No
enhancing areas were seen (Figure 2).

Discussion

The conventional treatment of intracranial tuberculoma is biopsy or excision of accessible lesions followed by chemotherapy. However, in some parts of the world such as the Indo-Pakistan subcontinent, tuberculosis is very much an endemic disease and the management is somewhat different. This is due to the recognition that a high percentage of deep seated enhancing lesions represent tuberculomas.4 Therefore biopsy of deep seated lesions is not always necessary, especially in the setting of concomitant extracranial tuberculosis or a family history of this disease. In such cases, empiric therapy with anti-tuberculous chemotherapy appears justified.5

We described the case of a middle aged lady who presented with mild hemiparesis and a history of seizures for one month. There was a family history of pulmonary TB. Keeping in view these features, it was decided to empirically treat with antituberculous therapy. Within a period of three months the clinical symptoms resolved and CT scan showed no evidence of residual disease.

This case is unusual because medical therapy usually results in gradual resolution of intracranial TB. Radiologic response is usually not complete with some scarring or encephalomalacia visible many years later.

Empiric medical therapy is a good alternative to biopsy or excision of deep seated intracerebral lesions. The response is usually good and may result in rapid and complete resolution as demonstrated by this case.

References


Blue Rubber Bleb Nevus Syndrome: associated with Severe GI Bleeding requiring one hundred Blood Transfusions

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Introduction

BRBNS is a rare entity characterized by distinctive venous malformation of skin and gastrointestinal tract, and less frequently of other sites.1 The vascular lesions in the GIT bleed frequently leading to chronic iron deficiency anaemia. The lesions in other sites bleed rarely while skin lesions do not bleed at all.2-4

Gascoyen was the first person who, in 1860 noticed the association of cutaneous and GIT vascular lesions with severe GI blood loss.5 A century passed by and, in 1958, William Bennet Bean coined the term Blue Rubber Bleb Nevus Syndrome, a term quite nicely chosen to describe the typical blue, rubbery, bleb like vascular lesions.6 Medicine regards traditions, so it is also named as Bean Syndrome.

The syndrome affects both sexes and all races, mainly in White and Japanese races.7,8 Typically the venous malformations are present at birth or appear in early childhood but adult onset cases have been reported in the literature.1,9-11 The exact pathogenesis of the disorder is yet to be evaluated. Early diagnosis, detailed evaluation and a good follow up of these cases is recommended for effective management. We report here a case of BRBNS in an eight years old girl who presented with typical cutaneous and vascular lesions with severe blood loss requiring about a hundred blood transfusions over a period of three years. To our knowledge this is the first case report of BRBNS from this country and also the first one of the reported cases associated with such severe bleeding and massive transfusion requirement and its associated complications.

Case Report

The patient is an eight years old girl, the product of a full term, uncomplicated pregnancy. At birth parents noticed two bluish macules over the anterior chest. With increasing age, similar new lesions appeared all over the body. These, lesions were soft, non-tender and of variable sizes. At three years of age, parents noticed an obvious change in the colour of her stools which turned tarry black.

The patient gradually became pale and developed symptoms of iron deficiency anaemia. She was put on iron supplements and she showed clinical improvement. At the age of five years, iron supplements were discontinued without medical advice and within two months she became so pale that she needed a blood transfusion. Since then she is on regular blood transfusions with decreasing interval