Case Report

MultipleBbrain Abscesses in a child with congenital cyanotic heart disease

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Abstract

We report multiple and diffuse supratentorial and infratentorial brain abscesses in a ten months old girl with D- transposition of great arteries. The child was managed medically with intravenous antibiotics for 4 weeks. Her fever settled, however, weakness of right half of the body persisted despite remarkable improvement. Multiple abscesses (about 40 in number), in a child less than 2 years age, associated neutrophilia with toxic granulations and successful therapy with antibiotics alone makes this an unusual case.

Introduction

Brain abscess is a relatively uncommon but potentially life-threatening condition. Predisposing factors for brain abscess include cyanotic congenital heart diseases (CHD); infections of the middle ear, mastoids, paranasal sinuses, and soft tissues of the face, orbit, or scalp; penetrating injury or comminuted fracture of skull; intracranial surgery; congenital lesions of the head and neck and abnormalities of immune system. No underlying illness or source of infection can be found in about 20% cases.[1] CHD is the most common predisposing factor and about 5 to 18.7% patients with CHD develop brain abscess.[2] Cerebral abscesses are evenly distributed between the two hemispheres, and approximately 64 to 76% abscesses are located either in parietal, frontal or temporal lobes. Most brain abscesses are single, but 10-27% are multiple and may involve more than one lobe.[1]

Despite advances in the management of cerebral abscess, associated morbidity and mortality is still appreciable. High index of clinical suspicion and the correct choice of investigations is the key to early diagnosis and successful treatment. Contrast enhanced computed tomography (CT) is the best investigation since non-contrast computed tomography does not identify all cerebral abscesses.[3]

Case Report

A ten months old girl presented with the complaints of fever, diarrhoea and weakness of right half of the body for five days. There was no history of earache or discharge, facial or scalp infection or trauma, seizures, or any other systemic disorder. She had history of bluish discolouration of lips and tongue which was more pronounced during crying; failure to thrive and recurrent episodes of respiratory tract infections since early infancy. Though her birth, feeding and family history were unremarkable yet her development was delayed as compared to her peers. On examination; she weighed 6 Kg and her length was 65 cm (both below the 3rd centile for age). She had some dehydration, marked central cyanosis, grade IV clubbing, tachycardia, tachypnoea, and fever. Examination of cardiovascular system was normal. She had inability to sit, stand and hold any object in right hand. Her deep tendon reflexes were exaggerated. Rest of the systemic examination was normal except few crepitations on chest auscultation.

Investigations showed increased total leukocyte count (TLC-35.7x10^9/L), neutrophilia (neutrophils 80%) with toxic granulations, haemoglobin 11.5g/dl, platelet count of 203x10^9/L and erythrocyte sedimentation rate (ESR) was 45 mm after 1st hour. Her chest radiograph showed mild cardiomegaly and clear lung fields. Her abdominal ultrasonography and renal function tests were normal and blood culture did not grow any organism. Contrast enhanced CT scan of brain revealed multiple (about 40 in number) ring enhancing as well as solid appearing brain abscesses, widespread in frontal, parietal, and temporal lobes and the left lobe of the cerebellum (Figures 1 and 2). A large infarct was also seen in left deep parietal region extending up to sub-ependymal region (Figure 2). No evidence of ventriculitis or cerebritis was seen. No pathology was seen in her mastoid, and

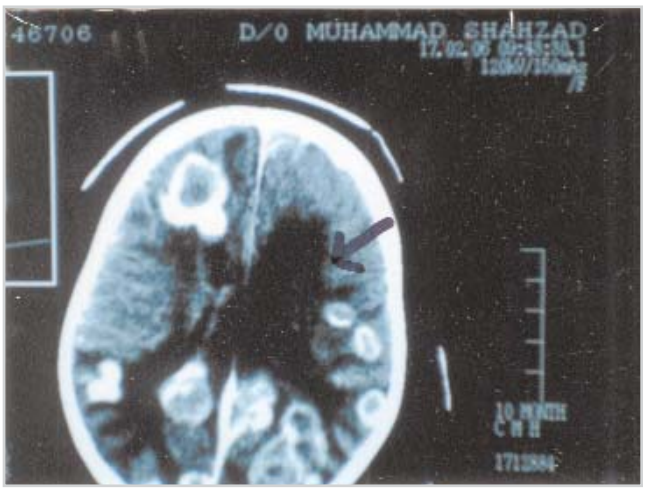

Figure 1. Multiple ring enhancing and solid abscesses in the supratentorial region and a large white matter infarct in the left deep parietal region (arrow).

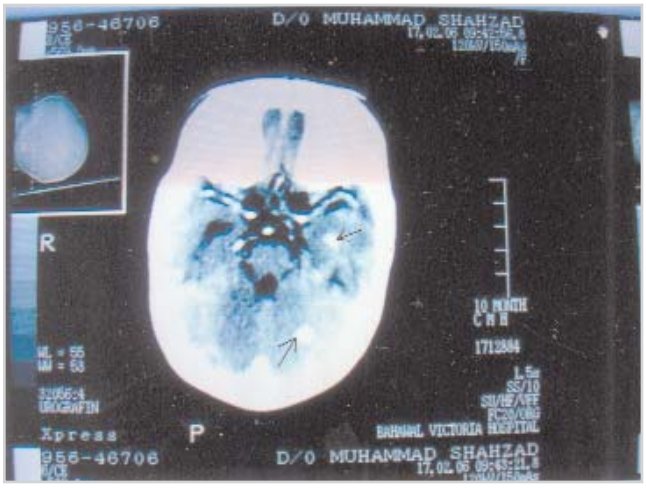

Figure 2. Solid appearing abscesses in the infratentorial compartment (arrows)

nasopharangeal region. Echocardiography revealed transposition of great arteries, pulmonary atresia, persistent foramen ovale and large ventricular septal defect.

She was managed with intravenous (IV) antibiotics; Ceftriaxone and Metronidazole for 4 weeks; IV fluids to correct her dehydration; physiotherapy and antipyretics. After discharge from the hospital she was prescribed oral augmentin and metronidazole for two weeks. Her diarrhoea settled and she became afebrile within one week of treatment. The repeat CT brain after 2 weeks showed decrease in abscess number and size. The weakness of her right half of body improved remarkably but persisted. She was referred to Armed Forces Institute of Cardiology Rawalpindi for management of her cardiac lesions and she is on waiting list for Blalock-Taussig shunt.

Discussion

Transposition of the great arteries, accounts for approximately 5% of all CHD. In this anomaly, the systemic veins return normally to the right atrium and the pulmonary veins return to the left atrium. The connections between the atria and ventricles are also normal. However, the aorta arises from the right ventricle (RV) and the pulmonary artery from the left ventricle. This abnormal connection of aorta with RV bypasses the filtration of the blood through the pulmonary circulation, where bacteria are intercepted by phagocytosis. This may allow direct entry of the organisms to cerebral circulation. Hyperviscosity secondary to compensatory polycythaemia results in minute low-perfusion areas leading to tissue hypoxia and metabolic acidosis. Shunted blood containing microorganisms may seed in such areas, forming a cerebral abscess.[2,4]

Multiple abscesses are particularly associated with cyanotic CHD.[1] The present case had about 40 abscesses widespread in all cerebral lobes and the left cerebellar lobe. Prusty has reported multiple abscesses in 10% of 60 cases of brain abscesses in patients with CHD.[5] Whereas, Shahzad et al[1] reported multiple brain abscesses in 36% cases. This variance may be due to diversity in the types of the predisposing conditions, socioeconomic and regional differences of the study population.

Cerebral thrombi leading to cerebral infarction usually occur in the cerebral veins or dural sinuses and occasionally in the cerebral arteries. These are common in first 2 years of life and are precipitated in the presence of extreme polycythaemia and dehydration.[6] In the present case, gastroenteritis induced dehydration can be the precipitating factor for the large infarct in left parietal lobe, manifesting as right sided haemiplegia.

Brain abscess usually presents within 2 weeks of onset. The presenting features include constant and progressive headache refractory to therapy, vomiting, papilloedema, focal neurological deficits, convulsions, meningism and altered sensorium. However, immunocompromised patients may have an insidious onset.[7]

The microbiology of brain abscess reflects underlying host risk factors. In patients with CHD the most common organisms are Gram-positive cocci, Streptococcus milleri, Streptococcus viridans, microaerophilic, Staphylococcus aureus and anaerobic streptococci.[5,8,9]

Contrast enhanced CT facilitates early detection, precise localization, exact enumeration, accurate characterization, size and staging of the abscess. It also detects hydrocephalus, raised intracranial pressure, oedema and associated infections like subdural empyema, ventriculitis and thus helps in treatment planning. It is also invaluable in assessment of adequacy of treatment and sequential follow up. Radiological differential diagnosis of such small and multiple, ring enhancing and solid lesions involving supratentorial and infratentorial regions include neurocysticercosis, fungal abscesses and metastasis secondary to neuroblastoma in this age group.[7] A detailed history and thorough examination and relevant investigations excluded all these entities in our patient.

Management of brain abscess among cyanotic CHD patients is a little intricate business. These patients possess not only cardiopulmonary risk but a wide variety of coagulation defects which enhance the risk of anaesthesia and surgery. The empirical antibiotic treatment of a lesion resulting from cyanotic CHD is cefotaxime or ceftriaxone and metronidazole which should be changed according the sensitivity pattern of the causative microorganisms.[7,9] The duration of antibiotic therapy depends on the organism and response to treatment, but it is usually 4-6 weeks.[10]

Surgical management of brain abscesses has changed since the advent of CT. If abscess continues to grow despite antibiotic treatment or its size does not decrease within four weeks, a surgical procedure is indicated to confirm the diagnosis, to obtain a sample for culture and sensitivity and to remove as much purulent material as possible. Other indications of surgery are; significant mass effect and neurological deficit, multiple lesions in surgically accessible locations, the multiloculated lesion, the posterior fossa lesions, and the abscess larger than 2.5 cm in diameter.[7,10]

Conclusion

Although rare, the brain abscess has to be remembered in patients who have neurological alterations associated with congenital cyanotic cardiopathy. Computed tomography of the brain is mandatory to confirm the diagnosis.

References

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