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SHORT REPORT

***Brucella melitensis* infection of ventriculo-peritoneal shunt: A form of neurobrucellosis manifested as gastrointestinal symptoms**

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Summary A report of a 9 year-old child with Myelomeningocele who has a ventriculo-peritoneal (VP) shunt presented with gastrointestinal symptoms and peritonitis. The patient had no CNS symptoms but the cerebrospinal fluid was positive for *Brucella melitensis*. We discuss neurobrucellosis in children, its various presentations, complications and challenges in treatment, choice of antibiotics and duration.

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Introduction

Brucellosis is an endemic zoonotic disease in Saudi Arabia. It is a disease with protean manifestation that involves almost any body system. Musculoskeletal manifestations in the form of arthralgia and/or arthritis are the most common. However,

in endemic areas rare presentations like neurobrucellosis should be diagnosed in any patient with focal or nonspecific neurological manifestations. Rare presentations of neurobrucellosis like brain abscess, cranial nerve neuropathy, Guillian Barre syndrome, and ventriculo-peritoneal shunt infection do occur. Some patients with neurobrucellosis may go unnoticed as they present only non-specific symptoms like headache and depression. This report is about a 9 year-old child with myelomeningocele and ventriculo-peritoneal shunt presented with gastrointestinal but no neurological symptoms, found to have a positive cerebrospinal

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fluid for *Brucella Mellitensis*. Discussions on neurobrucellosis in children, its various presentations, complications, and challenges in treatment, including antibiotic choice and therapy duration are presented herein.

Case report

A 9 year-old child with Myelomeningocele who has a Ventriculo-Peritoneal (VP) shunt inserted at 6 months of age for hydrocephalus was presented with progressive abdominal distension, vomiting, and fever. He had no acute neurological manifestations. Computed tomography (CT) of the abdomen revealed distended bowel loops with no obstruction, multiple mesenteric lymph nodes associated with free fluid and peritoneal enhancement. The findings were suggestive of peritonitis with ileus. There were not enough peritoneal fluid to aspirate but aspiration of the VP shunt reservoir yielded yellowish cerebrospinal fluid (CSF) with white blood cell count $18/\mu\text{L}$ (88% lymphocytes and 9% polymorphs), protein 780mg/dL, and glucose 45mg/dL. The Gram stain was negative but CSF culture yielded growth of *B. melitensis* from two separate taps, 3 days apart. The *B. melitensis* titer in the CSF was only 1:20. Peripheral blood culture was negative in multiple occasions, and the peripheral *B. melitensis* titer was very high at 1:20,480. The patient did not have any neurological symptoms, the VP shunt was functioning, and CT of the brain was read as unchanged from previous one. The patient received intravenous doxycycline, rifampin, ciprofloxacin, and gentamicin for 2 weeks. He was discharged on oral doxycycline, Bactrim, and rifampin following CSF sterility and clinical improvement. Two weeks later he returned with altered mental status (GCS 13/15) and worsening of gastrointestinal symptoms with intestinal obstruction. Abdominal ultrasound revealed a large fluid collection. The VP shunt reservoir tap revealed increased WBC to 126 (84% lymphocyte and 8% polymorphs), sugar of 90 and protein 360mg/dL with a CSF *B. melitensis* titer of 1:640. The shunt was externalized and external ventricular drainage established. Exploratory laparotomy revealed a peritoneal pseudocyst adherent to the small bowel and multiple small bowel adhesions. Peritoneal fluids culture was negative for *B. melitensis*. Adhesiolysis was done and a longer course of IV antibiotics given (4 weeks). Ventriculopleural shunt was inserted before discharge and the patient received oral doxycycline and rifampin for 10 months in addition

to co-trimoxazole for the first 4 months. His condition had improved and *B. melitensis* titre normalized (CSF titer decreased to 1:40 and peripheral titer to 1:5120) after completion of therapy.

Discussion

Brucellosis is an endemic disease in Saudi Arabia. The Ministry of Health reported an annual incidence rate among Saudis at 18.42/100,000 population. However, studies from different regions showed an incidence approaching 138/100,000 population. Sekait reported brucella seroprevalence of 15% in Saudi Arabia [1]. This high incidence is attributed to rearing animals, especially camels, and consumption of their raw milk. It is believed that milk pasteurization remove a lot of its valuable constituents. Brucellosis has varied clinical presentations. Almost any organ system can be involved. The most common form of clinical manifestation is musculoskeletal, i.e., arthritis. Neurobrucellosis is not uncommon in adults but rare in children; 7% and <2% of cases respectively [2].

The clinical presentation of neurobrucellosis is variable with meningitis and meningoencephalitis being the most common. Other presentations of neurobrucellosis include polyradiculopathy, myelitis, hearing loss, visual disturbances, chronic vomiting, coma, and psychiatric disorders mainly depression. Rare CNS manifestations include: brain abscess, hydrocephalus, pseudotumor cerebri, Guillian Barre syndrome, cranial nerve palsy, cerebral venous sinus thrombosis, subdural and epidural collection, and stroke have been reported [3,4]. Therefore, clinical manifestations of neurobrucellosis can range from subtle manifestations like fever and malaise to forthright meningitis. CNS involvement is usually through hematogenous route. However, few case reports showed ascending infection through ventriculo-peritoneal shunt and vice versa where neurobrucellosis could give rise to peritonitis. Locutura et al., reported a male adult who presented signs of peritonitis and was found to have positive ascetic fluid for brucella. He was treated and 4 months later was presented with neurobrucellosis [5].

Anderson and Mortensen reported a 27-year old lady with signs of increased intracranial pressure and found to have hydrocephalus that required VP shunt insertion. Three months later she was admitted with abdominal distension and ascites. *B. melitensis* was isolated from CSF, ascetic fluid, and shunt system [6]. Alexiou et al., reported a 2-year old child who has VP shunt who showed signs of

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