A CASE OF SUBDURAL EMPYEMA SECONDARY TO PYOGENIC MENINGITIS IN AN INFANT

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ABSTRACT

Subdural empyema (SDE) is a life-threatening infection, it accounts for about 15-22% of focal intracranial infections. It might be life threatening if not managed properly in infants and children. We report a case convulsions 14 mo-old infant who was admitted with a 1/52 history of body swelling, 3/7 history of fever, and 7105 with diagnosis of pyogenic meningitis. The cranial ultrasound scan (u/s) revealed bilateral subdural fluid collection, and features of cortical sulci thickening, sequela of meningitis. She was put on IV antibiotics. Then three days later, the brain CT scan showed subdural fluid with right midline shift. But in view of persistent fever and the recurrence of convulsions, a repeat cranial u/s 13 days ago prior to the first revealed a subdural empyema, unfortunately 2 days later, the infant passed on.

INTRODUCTION

Subdural empyema is a collection of pus in the space between the outermost layers of the meninges, the dural and the arachnoid (Gormley et al., 1996). Common causative organisms are anaerobic and aerobic Streptococci, Staphylococci, Haemophilus influenza, and other gram-negative bacilli. Meningitis is the most common cause of SDE in infants (Liu et al., 2010). However, in older children, sinusitis and otitis media are the most common sources (Agrawal et al., 2007). The mortality rate of patients with SDE is around 4%, while the morbidity for survivors is even higher with residual neurologic deficits reaching up to 50%, hemiparesis 15-35% and persistent seizures 12-37.5% (Germiller et al., 2006). Because of its rarity, many medical professionals may not have encountered a case in recent years. This case report aims to increase the awareness of health professionals who treat patients with pyogenic meningitis of the need for early detection of subdural empyema while monitoring these patients and hence address its management; this may improve outcomes, given the associated high morbidity/mortality rate.

Case Report: M.B.: 14 mo old, F, Congolese-refugee, Whose mother address was in Mengo-kisenyi/ Kampala-Uganda. She was a sero-exposed child, who presented with: fever, convulsions x 3/7 and body swelling x 1/52. She was unwell 1/52 prior to admission when she developed body swelling, which started on the lower limbs, then progressed to the upper parts of the body. Three days prior to admission, she developed fever, high grade fever, associated with convulsions, general tonico-clonic (GTC), more marked on the face, right upper and lower limbs, 5 times lasting each around 10 min, associated with vomiting, 2 times, non-projectile. There was no history of loss of consciousness, no history of cough or difficulty in breathing (DIB), no history of diarrhea, or change in micturation habit, no history of flu like illness prior to this episode. This is the index seizure episode, and the third admission, the first one was on September 2013, admitted in 16 C Ward at Mulago Hospital (Kampala) for pneumonia, at this moment she tested HIV negative. The second admission was in a clinic in Kisenyi for a febrile disease, was treated with oral and IV medications ignored by the mother, she improved and she was discharged home where she spent two weeks prior this last admission. Mother attended Antenatal Clinic in DRC 4 times, was tested HIV negative and she delivered a term baby, who cried immediately and was put on breastfeeding. She grew up well, sat at 3-4 months, crawled at 7-8 months, but at 14 mo old she was not able to walk or to talk. She was exclusively breastfed for 6/12, she was weaned to matoke, porridge, green, meat, fish,… Immunization was completed. She was the 5th of her family of 5 children, the other 4 children where apparently well. The father has died, and the mother is a seller of food, she was tested HIV positive when this infant was admitted in 16 C Ward in September 2013, and she was on HAART at Taso Clinic.

Physical examination: She was sick looking, with silky hairs, Temperature 39 Celcius degree, with mild pallor, had no...
jaundice or xerophtalmia, had no oral sore, had no skin lesion, had pitting edema grade II, MUAC was 13 cm. She was conscious, neck was soft with bulging anterior fontanel, had photophobia, pupils were equal size, regular and reactive on light. Had increased tone more marked in the left upper and lower limbs, and increased reflexes and had a bilateral sustained angle clonus. Abdomen was normal fullness with a umbilical hernia and hepatomegaly of 3 cm BCM. Other systems were unremarkable.

Our impression was:
- Pyogenic meningitis
- R/O Space Occupying Lesion syndrome and Malaria

We did a CBC which showed Hb of 6 g/dl, other parameters were in normal range, BS for malaria parasites was negative. DNA-PCR result for HIV was not yet out, CSF analysis revealed Protein: 400 mg/dl; WBC: 1000 cells/mm cube, India ink was negative, gram stain showed gram negative; bacteria the differential showed polymorphonuclear predominance. Culture and sensitivity was to follow up. The cranial u/s scan showed bilateral subdural fluid collection, and features of cortical sulci thickening, sequelae of meningitis. Brain CT scan showed subdural fluid with right midline shift. She was put on IV ceftriaxone, phenytoin, rectal panadol, F75, folic acid and an NGT for feeding. But 10 days later while in the ward, she continued to have fever on and off and recurrent convulsions and she developed low level of consciousness, she was then started on IV vancomycin for possible staphylococcal infection and on Anti TB drugs for possible TBM. Despite these antibiotics, fever persisted. Then on day 15 of admission, a repeat cranial ultrasound showed a subdural empyma and unfortunately the same day, the infant died.

**Discussion of the case:** Stephanov et al. (1979) described Subdural empyma "as the most imperative of neurological emergencies", which, if not treated immediately, is associated with high risk of status epilepticus, spreading cortical venous and cortical venous sinus thrombosis, fulminating cerebritis, brain swelling, cerebral coning and ultimately leads to death. Meningitis is the most common cause of SDE in infants (Liu et al., 2010). In our patient meningitis has been the predisposing factor of subdural empyma in view of the CSF-analysis result. However other authors suggest that Otorhinolaryngeal infection, especially paranasal sinusitis, was the most important predisposing factor in previous studies (Dill et al., 1995). This was not the case for our patient who did not have this kind of infection. Infants and young children with SDE might present with altered mental status, meningeal irritation, and/or signs and symptoms of intracranial pressure. Some studies showed that 40% of patients with SDE present with seizures (De Bonis et al., 2009). Our patient had seizures, reduced level of mental status and vomiting. Patients with SDE due to sinusitis have a wide range of clinical presentation. They can present with the typical symptoms of sinusitis like fever, headache and purulent rhinorrhoea; in addition, tearing of the eyes, photophobia and painful parathesias in the area covered by the trigeminal nerve. Overall, patients with SDE due to frontal sinusitis have more subtle signs and symptoms compared to infection of other sinuses. Among these mentioned symptoms above, our patient had photophobia, and she didn’t present other signs of sinusitis. Imaging of the head is recommended for every patient suspected of having SDE. Cranial ultrasonography is usually the first imaging mode to be ordered in infants because it is safe, cost-effective, and it usually differentiates subdural empyma from subdural effusion (Liu et al., 2010). The u/s scan has been of great help in making diagnosis of SDE in our patient. However MRI is the imaging modality of choice in making diagnosis with sensitivity of 93 % (Bruner et al., 2012), even if Computed Tomography (CT) of the head is considered cost-effective and accessible, however, it can be normal in up to 50% in patients with SDE (Gupta et al., 2011). The CT scan showed subdural effusion in our patient, and this was in the initial clinical presentation, and probably it would have been helpful if it was done later in the clinical presentation when the empyma was forming. Lumbar puncture (LP) is currently contraindicated because of possible cerebral herniation from increased ICP (Haines et al., 1990; Obama et al., 1993). Lumbar puncture was done after excluding the raised ICP by the cranial u/s scan, and it was helpful in making meningitis diagnosis in our patient. Antibiotic therapy as soon as possible with broad coverage for anaerobes, staphylococci, and aerobic streptococci. Antibiotic therapy alone may be adequate for small Subdural empyma (ie, <1.5 cm diameter). Because of the aggressive nature of this disease, however, this option is not widely utilized. The antibiotics should be given for a period of 3-6 week with close monitoring of clinical status (Mauser et al., 1987). Our patient received ceftriaxone on time, and vancomycin came in a little bit later, and therapy was supposed to be given for up to 6 weeks, but our patient died in two weeks’ time, probably she would have benefited from craniotomy (Haines et al., 1990), which allows wide exposure, adequate exploration, and better evacuation of the purulent collection than other procedures. If the organism is unknown, then oxacillin plus ceftriaxone/cefotaxime plus metronidazole is recommended; however, if there is a suspicion for methicillin-resistant S. aureus, then vancomycin instead of oxacillin is warranted (De Bonis et al., 2009). Linezolid is an alternative treatment in case of conventional antibiotic regimen failure (Lefebvre et al., 2009). Nowadays, the survival rate for children with SDE is more than 90% if surgical intervention was done on timely basis (Ramamurthi and Pande, 2005), unfortunately our patient passed on probably because she did not undergo surgery.

**Conclusion and recommendations**

Subdural Empyema is a life threatening entity if not diagnosed early. Brain sonography can be a helpful tool for early diagnosis of SDE in infancy. Subdural empyma should be kept in mind, since early diagnosis and treatment is the most important way for a better outcome. Neurosurgeons should be associated in the management of the subdural empyma. Alternatively, craniotomy is warranted in addition to antibiotics therapy.

**REFERENCES**


