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EMPTY SELLA SYNDROME COMPLICATING MENINGITIS: A CASE REPORT

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SUMMARY

Meningitis remains a potentially life threatening emergency and a major public health hazard in sub-Saharan Africa, where it is a significant cause of morbidity and mortality. The prevailing political upheavals in some African countries, with the increasing poverty and poor health facilities (especially lack of immunization), resulting in delayed detection and treatment of meningitis cases, inappropriate usage of antibiotics and development of antibiotic resistance, has caused, unsurprisingly, physicians to encounter different complications of meningitis in affected individuals.

We present the case of a 15-year old female Nigerian who developed communicating hydrocephalus, epilepsy and empty sella syndrome following meningitis, to illustrate the less common sequelae of meningitis.

Attempt is made to review the less common complications of meningitis, and also emphasize the need for increased awareness of these complications and regular detailed neurological evaluation at follow-up of patients treated for meningitis.

INTRODUCTION

Meningitis is inflammation of the meninges, the membranes covering the brain and spinal cord, and it is characterised by pleocytosis. In the past few years, significant advances have been made in understanding the pathophysiology of bacterial meningitis and in development of approaches to management.

Despite these developments, bacterial meningitis remains an important source of morbidity and mortality especially in the developing countries of the world.

In Africa, acute bacterial meningitis has an overall annual endemic rate in the region of 10-50 per 100,000 population, and epidemics of meningococcal meningitis sweep through the sub-Saharan 'meningitis belt' every 8 to 12 years[1].

In the USA and UK, despite adequate treatment, the overall fatality rate from bacterial meningitis is 10% but in Dakar, Senegal during a non-epidemic period from 1970 to 1979 it was 44.2%[2].

Case fatality rates and serious complications from meningitis caused by *Streptococcus pnemoniae* infection have been reputed to be as high as 40%[3]. Mortality rates are usually lower in patients with meningitis caused by *Neisseria meningitidis* (5% to 10%) or *Haemophillus influenza* type b (3% to 6%)[4].

The most common long-term complication is sensory-neural hearing loss, which occurs in about 10% of survivors [3,4]. Other long-term sequelae include seizure disorder, hydrocephalus, learning difficulties and focal neurological problems (e.g. spasticity, paresis, ataxia and cortical blindness)[5].

We present the case of a 15 year-old Nigerian female with long-term sequelae of hydrocephalus, empty sella syndrome and recurrent seizures to illustrate complicated meningitis.

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CASE PRESENTATION

Miss E.C. a 15 year-old Nigerian female student presented to the neurology clinic of the University Teaching Hospital, Benin City on the 14th June, 1999, with history of recurrent headaches and secondarily generalized tonic-clonic seizures of 18 months duration. There were associated urinary and fecal incontinence, salivation and post-ictal headaches and muscular aches. There was no history of tongue biting. Seizures occurred both during the day and night with a seizure frequency of one seizure attack every 4 months i.e. 3 yearly.

In addition, she gave a history suggestive of CSF rhinorrhea three months prior to presentation. She is right handed, with unremarkable birth history but was admitted and treated for meningitis at a mission hospital at the age of 10 years i.e 5 years ago. She gave no history of head injury. Her immunization was not completed. She is the second of four siblings in a monogamous family with no significant family medical history.

Physical examination revealed a young girl with intact sensorium, well developed for her age, with no evidence of pallor, jaundice, peripheral lymph-adenopathy or peripheral fluid retention.

She had a heart rate of 84 beats per minute and blood pressure of 100/70 mmHg. The heart sounds were normal without murmur. Her chest was clinically clear and she had no abnormal findings on abdominal examination.

Neurological evaluation revealed no signs of meningeal irritation or focal lateralizing signs. She however, had blurring of the disc margins bilaterally on fundoscopic examination.

Neuro-psychological evaluation showed significant memory impairment (especially immediate recall). A working diagnoasis

of post-meningtic epilepsy to exclude an intrasellar mass was entertained.

She had laboratory investigations. Results are outlined in Table 1. Lumbar puncture was contraindicated.

Plain skull X-ray revealed features consistent with raised intracranial pressure. She had a CT brain scan done which showed communicating hydrocephalus with empty sella syndrome. (Refer films I and II).

She was treated with carbamazepine 600mg daily in divided doses for seizure control and oral dexamethasone 4mg tid to reduce intracranial pressure. She responded well with cessation of seizures and was referred for ventricular shunting.

She had a successful ventricular shunting and is still being followed up in the clinic.

Table I: **Results of Investigations**

Tests	Dates	Results
1. Full blood count	7/6/99	Normal
2. Urine analysis	7/6/99	Normal
3. Serum Calcium/ Phosphate	7/6/99	Cal. 8.6 mg/dl (8.5-11 mg/dl) Phos. 3.6mg/dl (2-4 mg/dl)
4. Neuro-psychological Assessment	12/6/99 Memory (recognition testing for recall of words and figures)	Recognition memory test – for words 80% correct; for figures 40% correct.
	Continuos performance test for attention	D' 0.29 (poor attention/ concentration B' 0.09.
	Mental speed	Prolonged auditory and visual reaction times AUD.-539+/- 184 millisecs VIS.-414+/-140 millisecs
5. Skull X-ray	8/7/99	Destruction of the sella association with irregularity of the calvarial inner table
6. CT Scan	17/8/99	Communicating hydrocephalus and empty sella syndrome
7. EEG	12/6/99	Primary generalised epileptiform patterns

DISCUSSION

Complications of meningitis, including high mortality, are not unusual in socio-economically deprived countries. The occurrence of complications has been associated with the virulence of the causative organism, the integrity of the host defense mechanism and of the meningeal covering[5].

Complications may be short-term or long-term. Short-term complications include hydrocephalus, sub-dural effusion, cerebral herniation, cranial nerve palsies and seizures[5,6,7]. Long-term sequelae are seen in 20% of survivors of bacterial menigitis[7]. The most common long-term complication is sensori-neural hearing loss.

Klein et al[9] showed that 33% of children with meningitis developed complications at discharge but 11% had residual deficits after 5 years of follow up. Some sequelae can remit over time.

Long-term sequelae that have been described include behavioral problems, language disorders, mental retardation, impaired vision, seizure disorders, focal neurological problems (spasticity, ataxia, paresis, cortical blindness), learning difficulties and hydrocephalus[10].

Empty sella syndrome as a complication of meningitis has been rarely described. Cerebro-vascular complications of meningitis have been most extensively documented in the setting of acute bacterial or chronic tuberculous meningitis. However, vasculitic basilar artery thrombosis, a rare complication in fungal meningitis secondary to Candida infection was recently reported[11].

Syringomyelia has been reported as an unusual acute complication of meningitis, though uncommon[12]. We report two relatively rare complications in a 15 year-old girl, namely communicating hydrocephalus and empty sella syndrome, which were identified five years after the meningitic process.

Hydrocephalus, usually of the communicating type, is commonly seen in newborns. It is a rare complication following meningitis in young adults[9]. The presence of ventricular enlargement alone does not indicate hydrocephalus, as this may be due to loss of parenchymal tissue[10]. But if the enlargement is accompanied by other symptoms and signs of increased intracranial pressure, it is indicative of hydrocephalus. Our patient had evidence of papilloedema on fundoscopic examination, which, in addition to the CT findings of ventricular enlargement, supports hydrocephalus.

This is usually treated by shunting to one of the body cavities[9], which was eventually done for this patient.

Epilepsy is not uncommon as a complication of meningitis, especially in developing countries where CNS infections are rampant. The disparity between percentages of cases of symptomatic epilepsies due to CNS infections reported by Obembe and Ahmed[13] in Kaduna in Northern Nigeria, where there is a higher prevalence of epidemic meningitis, and that reported by Danesi[14] in Lagos, southern Nigeria, proved this point.

Recurrent seizures may occur during the illness and may be a long-term sequela in up to 30% of patients[2]. Our patient had defective immediate recall which is not surprising as impairment of congnitive abilities in epilepsy among Nigerians has been reported, especially memory, attention and psychomotor speed[15].

This, no doubt results in learning difficulties.

Empty or partially empty sella are terms used when the pituitary gland does not fully occupy the sella, and this is found in 6% of routine post-mortems and MRI examinations when a mid-line saggital image is obtained[16]. The prevalence is not known as it is a radiological diagnosis based on CT or MRI performed for headache, CSF leak or late visual deterioration after surgery or radiation therapy for a pituitary lesion[17].

The radiological diagnosis of an empty sella is usually made with care as cystic craniopharnygioma or Rathke's cleft cyst may sometimes mimic the normal anatomical contours both within and above the sella. This distinction is of importance, especially in children with neuroendocrinological abnormalities who could have an empty sella, cyst, or craniopharyngioma. This group usually constitutes about 18% of patients with neuroendocrine symptoms[18].

Empty sella syndrome may be primary or secondary. Primary sella conventionally is not associated with significant endocrine or visual abnormalities. Sensitive dynamic endocrine testing may disclose subtle hormonal changes but hormone replacement is seldom-necessary[19]. It is commonly seen in patients with benign intracranial hypertension and may be sequel to pituitary apoplexy[20].

About 50% of adult patients with a primary empty sella have antipituitary antibodies that indicate previous autoimmune hypophysitis[21].

Secondary empty sella may occur following post-partum necrosis of the pituitary gland. It is more common in multiparous women, presumably due to repeated enlargement and involution of the gland with each pregnancy[16].

Spontaneous CSF rhinorrhoea may be associated with an empty sella with or without raised intracranial pressure[22].

The optic chiasm may herniate down into the empty fossa but this may not be responsible for visual field defects sometimes founds in these patients[23]. Radiation necrosis or arachnoiditis are more likely to be responsible[23].

Our patient had no feature of endocrine dysfunction, the only symptom to suggest empty sella syndrome was the CSF rhinorrhea (CSF leak) in the presence of raised intracranial pressure. It is a condition rarely seen in patients with post-meningitic hydrocephalus[17]. The CSF leak may also occur in the absence of raised intracranial pressure as earlier indicated. Clinicians need a high index of clinical suspicion to identify cases with empty sella syndrome as this symptom is not invariable.

Complication of meningitis, like the ones reported in this patient, may not occur in the immediate post discharge period, hence the need for regular follow-up of all patients treated for meningitis. This enhances early discovery and prompt management. Though prompt initiation of appropriate antibiotic therapy is essential to preventing complications[5], studies, however, have shown the evolution of sequelae following 5 years of follow up after discharge of cases of meningitis who received adequate therapy[6,7,10].

We therefore recommend close follow up of all patients, including those that have been successfully treated for meningitis, especially in the developing countries, as this will help in early and prompt management of complications.

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