Case Report

Cerebrospinal Fluid Ascites: A Patient Case Report and Literature Review

G. Musa*, A. Gots, M. C. Lungu, M. Mutumwa

Department of Surgery, Livingstone Central Hospital, Akapelwa Street, P. O. Box 60091, Zambia

ABSTRACT

Cerebrospinal fluid ascites following ventriculo-peritoneal shunting for hydrocephalus is a very rare complication. We present the first case at our hospital. A 3year old female with communicating hydrocephalus presented with massive ascites. It recurred with 2-3weeks of ascitic tap to dryness. Other possible causes of ascites were excluded by normal renal and liver function tests and clinically normal cardiac function. Tests done on the ascitic fluid showed that it was consistent with cerebrospinal fluid with no signs of infection. CT abdomen excluded abdominal masses and CSF pseudo-cyst. A ventriculo-Atrial shunt was done and the ascites slowly regressed over the next 2-3 weeks with no recurrence. The reason why the peritoneum failed to absorb the CSF was not established.

INTRODUCTION

Ventriculo-peritoneal shunts are the most common procedure done for hydrocephalus. They have commonly been associated with many complications including infection, obstruction, dislodging etc. Rarely the patient may present with progressive abdominal distention due to accumulation of CSF. Commonly this presentation is a pseudocyst. Due to inflammation, the omentum forms a cyst around the tip of the shunt forming a fluid filled sac. In very rare incidences ascites occurs due to either production-absorption mismatch or a non-absorbing peritoneum.

Corresponding author:
Musa Gerald
Department of Surgery,
Livingstone Central Hospital,
1 Akapelwa Street, P. O. Box 60091, Zambia

LITERATURE REVIEW

CSF ascites has been reported in a wide array of cases. Up until 2016, 42 cases of CSF ascites had been reported [1]. CSF ascites is an uncommon complication, and in a retrospective study of 300 children by Rush DS et al, it was associated with 1.3% of shunt complications [2]. In most cases no underlying disease state can be found. This makes it apparent that the primary problem is inability of the peritoneum to absorb CSF. No definite explanation has been offered for the inability of the peritoneum to absorb the CSF.

Some proposed factors that may contribute to the development of CSF ascites have been postulated. Excess production of CSF as in a choroid plexus papilloma or villous hypertrophy of the choroid plexus. Patients with high CSF protein secondary to chronic infection (e.g. tuberculosis) or brain tumors, especially optic glioma. Peritoneal inflammation due to repeated shunt revisions or non-specific inflammatory response to shunt material has also been considered.

A publication by the neurology society of India reports 2 cases of CSF ascites. The first case of a 3-month old female baby having a VPS inserted for congenital hydrocephalus and only developing CSF ascites 3 ½ years later, following several revisions and a suspected infection (not proved by microbiology). The second case of a 7year old boy who had astrocytoma and hydrocephalus, had a VPS and developed CSF ascites 4 months later. Both patients were found to have high CSF protein. However, no evidence of infection was found in

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either of the patients. The two children had peritoneal biopsy done to exclude tuberculosis. Histologic examination revealed eosinophilic infiltration and mild chronic inflammation respectively both of which could not explain the massive ascites [3]. A case report of a 2year old boy, who developed CSF ascites after having a VPS since he was 5 days old for gross congenital hydrocephalus. This patient only had one shunt revision during this period. No evidence of infection or malignancy was found [4].

A case report of a 9month old Caucasian male, with multiple congenital abdominal abnormalities repaired at birth. Following an E. coli wound infection, he developed meningitis and ventriculitis which complicated intoaqueductal stenosis and hydrocephalus. At 55days old a VPS was performed. The child required shunt revision at 6months for blocked shunt and was well until 9months when he developed ascites following routine Diphtheria-pertussis-tetanus immunization [5]. Another case of a 37year old woman who required a VPS for idiopathic intracranial hypertension, developed CSF ascites about 5 years later. In this case Corynebacterium non-Jeikeium(JK) was grown from CSF from the shunt. The shunt was removed, infection was treated and a ventriculo-atrial shunt was placed [1].

The treatment of choice for CSF ascites has been to remove the VPS and replace it with a ventriculoatrial shunt and has shown good results. The case reports above were all treated in this manner and the ascites resolved thereafter. One case of a 27year old woman who developed CSF ascites 14years after VPS placement. When she developed CSF ascites a ventriculo-pleural shunt was placed instead, the patient developed pleural effusion and eventually had to have a ventriculo-atrial shunt placed [6].

DESCRIPTION OF CASE REPORT

A 3year old female presented with communicating hydrocephalus of undetermined cause. A ventriculo-peritoneal shunt was inserted and she was discharged. Two weeks later, she presented with abdominal distension with no associated pedal or periorbital edema and no cardiovascular symptoms. She had no features of liver failure. The distension had been slowly progressive with no associated vomiting, diarrhea, pain or fever. She had no urinary or cardiovascular symptoms.

Examination

She was stable, no jaundice nor pallor, mildly dyspneic

No features of increased intracranial pressure. Shunt was functional

Abdomen was grossly distended, non-tender and no organomegally.

Other systems where essentially normal.

Investigations

Pre-shunt CT Brain showed bilateral lateral, third and fourth ventricular dilatation. Lateral ventricles dilated more than the others. No other abnormalities noted.

Abdominal ultrasound and CT abdomen showed huge ascites with no localised pouch.

FBC/DC-normal,

Creatinine- 48.8umol/l, Urea-2.94mmol/l,

ALT- 11IU/l, AST- 45IU/l, ALP- 245IU/l

Total protein 70g/l, Albumin- 43.5g/l

Hepatitis B- Negative

<u>Tests done on ascitic fluid</u>

Clear fluid with supernatant

Fluid glucose: 5.36mmol/l (serum RBS- 6.8mmol/l)

Microscopy: White blood cells: 4/ml, No organisms

Treatment

Ascitic fluid was tapped slowly to dryness but itaccumulated slowly over 2weeks.The shunt was repositioned from the epigastric region to the right hyponchodrium but after 1week of observation, the fluid had accumulated.The patient was prepared for ventriculo-atrial shunt insertion.

The VAS was inserted by making a purse string suture on the right internal jugular vein. The preferred facial veins where too small to allow the diameter of the shunt tubing. During insertion, the shunt was flushed with heparin.X-rays done intra-operatively confirmed the tip of the shunt at the 6th thoracic vertebra.

The post-operative period was unremarkable. The wound healed well and the ascites did not accumulate. She was on high dose intravenous antibiotics for 12 days prior to discharge on oral antibiotics. On day 12 post VAS, she was discharged. The ascites had not accumulated. At one-month review, she had no complaints and had no ascites. At 3months review, she was developing well, had attained appropriate milestones with no complaints.

DISCUSSION

Ascites is the accumulation of freely circulating fluid in the peritoneal cavity. It has been documented to frequently occur as a complication of portal hypertension and nephrotic syndrome in childhood and occurred rarely as a result of conditions such as Tuberculous peritonitis, peritoneal carcinomatosis, cardiac, pancreatic and biliary disorders. In all these, however an underlying disease state can be found. In our patient none of the above causes present. It is apparent that the primary problem was the peritoneum, which just failed to absorb the CSF.

Intra-abdominal complications of Ventriculoperitoneal (VP) shunt are often abdominal abscess, peritonitis and dislodgement of shunt. CSF ascites is a rare complication of Ventriculoperitoneal shunt. Different intervals between shunt placement and symptomatic ascites have been reported and several etiologic factors discussed, but there was no definite explanation. Our patient developed ascites just 2 weeks from Ventriculoperitoneal shunt insertion, the shortest reported interval.

Some explained that imbalance between peritoneal absorption capacity and amount of CSF production was the major cause. By this definition, patients with excessive amount of CSF production like choroid plexus papilloma were at risk of developing CSF ascites following VP shunt. On the other hand, patients with high CSF protein due to chronic infection or brain tumors especially optic gliomas may have difficulties in CSF absorption through the peritoneum. Peritoneal inflammation due to repeated shunt revisions or non- specific inflammatory response to shunt material may also play a role in the decreased absorptive ability of peritoneum. In our literature review, some patients had peritoneal biopsy done but showed no significant inflammation to explain the ascites. However, peritoneal biopsy was not done in our patient.

To make a diagnosis of CSF ascites, other causes of ascites needed to be excluded by extensively investigating the patient for renal and hepatic dysfunction. Our patient had normal renal, hepatic and cardiac function

The treatment for CSF ascites required the CSF drainage to be diverted from the peritoneal cavity. VP shunts can be converted to Ventriculo-atrial, ventriculo-pleural, or Ventriculo-gallbladder shunts. Ventriculo-atrial shunts were preferred in literature with good outcomes. In one of the cases reviewed, VP shunt was converted to ventriculo-pleural shunt and the patient developed pleural effusion. In our patient, VP shunt was converted to Ventriculo-atrial shunt with good outcome. The patient recovered well and ascites did not reoccur.

CONCLUSION

The patient who presents with CSF ascites will have abdominal distention with no tenderness and not attributable to hepatic, renal or cardiac dysfunction. The patient could present weeks to years between time of shunt placement and evolution of symptoms. It will require multiple diagnostic tests to exclude other possibleetiology and should be managed by redirecting flow of CSF preferably to the right atrial.Our case aims at sensitizing practitioners on this problem and opening up for further discussions.

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