Challenges in the management of cerebrospinal fluid ascites: a case report

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Abstract

Cerebrospinal fluid (CSF) ascites is an uncommon sequela of ventriculo-peritoneal (V-P) shunt. We report a case of CSF ascites in a 7-year-old girl with craniopharyngioma and the challenges and limitations we faced in her management. Ascites completely resolved after a ventriculo-atrial (V-A) shunt surgery. Abdominal paracentesis, diuretics, and ventriculo-pleural shunt were not effective in the management of the CSF ascites.

Keywords Challenges · Management · Cerebrospinal fluid · Ascites

Introduction

A V-P shunt is a viable option for the diversion of CSF in neurosurgical practice. Though common, it could be associated with uncommon complications, one of which is CSF ascites. It is a diagnosis of exclusion, and when confirmed, an alternative site of CSF absorption may need to be explored. The pleura space and the right atrium could provide such an alternative. Though several complications have been reported after V-P shunt, CSF ascites is not commonly reported. As of 1984, there were only 18 publications on the subject, this increased to 42 by 2016 [1, 2]. This case report intends to highlight the challenges in the management of a case of CSF shunt ascites in our practice.

Case description

The index patient was a 7-year-old right-handed female who was diagnosed with obstructive hydrocephalus from a sellar/suprasellar tumour. She was worked up and had an

emergency right Kocher's V-P shunt with findings of clear CSF under intense pressure. CSF biochemistry was normal, and CSF culture yielded no growth. She had a remarkable improvement and was discharged on the 5th-day post-op to allow for adequate planning for definitive tumour resection.

Three months later, she re-presented with progressive abdominal distension, which was first noticed 4 weeks post-op. There were no features suggestive of bowel obstruction. She had no limbs or facial oedema. Physical examination revealed features of chronic malnutrition with significant weight loss. Her GCS on admission was 15. Shunt hardware was clinically assessed, and together with brain MRI, revealed the shunt to be functional. She had a grossly distended abdomen which was non-tender with an abdominal girth of 78 cm. There was respiratory distress at rest.

An abdominal ultrasound scan revealed echo-free peritoneal fluid. She had aseptic bedside peritoneal paracentesis, which yielded free-flowing straw-coloured fluid. Culture yielded no growth, and AAFB was negative. Peritoneal fluid biochemistry was essentially normal, and cytology was negative for tumour cells. Serum protein (total and albumin), urinalysis, liver function tests were all within normal limits. Repeat cranial MRI showed good ventricular decompression. A diagnosis of CSF ascites secondary to V-P shunt in a patient with sellar/suprasellar tumour with background malnutrition was made.

Ascitic fluid was tapped serially over 3 days (3.6 L in total) but recurred within 1 week. Reviews by paediatric teams ruled out cardiac, renal, and liver pathologies. Following a multidisciplinary meeting with paediatricians, cardiothoracic surgeons, anaesthetists, and nutritionists,

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the decision to divert CSF to an alternate site was made. She had the V-P shunt converted to a ventriculo-pleural shunt after tapping the abdomen dry. CSF protein was 0.24 g/dl. However, 4 days after the conversion, she developed progressive respiratory distress and was found to have clinical features of right pleural effusion, which was confirmed with a chest radiograph (Fig. 1). A total of 600 ml of fluid was drained from the right pleural space via a chest closed tube thoracostomy drain. She then had a conversion of the ventriculo-pleural shunt to a V-A shunt 7 days after the ventriculo-pleural shunt. Post-op radiograph after 48 h revealed resolution of the pleural effusion and respiratory distress (Fig. 2). No further recurrence of ascites was reported. The patient was then nutritionally rehabilitated, optimized, and had craniotomy and tumour excision done. Histology turned out to be adamantinomatous craniopharyngioma. She later died from surgery-related complications.

Discussion

To date, there are few published cases of CSF ascites in the literature worldwide and from Nigeria in particular [1, 3]. Due to the reliability of the peritoneal cavity as a site for CSF diversion, its unavailability could pose a great challenge in the management of hydrocephalus. The peritoneum can absorb up to 500 ml/24 h of normal saline in dehydrated children, and after the phase of osmotic absorption, the average fluid absorption rate is 33 ml/h [4].

Fig. 1 Pleural effusion on the 4th day after ventriculo-pleural shunt

Fig. 2 X-ray showing resolution of effusion after V-A shunt and CTTD

In CSF ascites, there is an excessive accumulation of CSF in the peritoneal cavity resulting from an inability of the peritoneum to absorb the CSF [5]. It has been suggested that this state of disequilibrium could result from primary peritoneal failure, increased CSF volume, increased CSF protein, infections (peritonitis), eosinophilic rejection of the catheter, immunological reaction to vaccination or in some cases, no definitive aetiology [1, 5, 6]. Tumours such as optic gliomas and craniopharyngiomas have been associated with CSF ascites attributable to possible increased CSF proteins [2, 5, 7]. In addition to possible specific proteins produced by optic gliomas, there could be an escape of tumour proteins into the subarachnoid CSF via defective blood–brain barrier resulting in increased protein levels which in turn impairs CSF absorption through the arachnoid villi [8]. Adegbite & Kahn reported considerably elevated CSF protein prior to extirpation of recurrent craniopharyngioma with subsequent reduction in protein to normal levels and non-recurrence of ascites on subsequent V-P shunt [9].

The interval between V-P shunt and the onset of the ascites could vary from 1 day to 12 years post-surgery [10]. In our patient, it was noticed at 4 weeks post-V-P shunt.

At the time of re-presentation (3 months post-V-P shunt), the patient was also malnourished with significant weight loss. She was unfit for tumour resection, needed an alternate site for CSF diversion, and was psychologically disturbed. The “lock-down” and scaling down of clinical

services from the COVID-19 pandemic delayed tumour resection. Interestingly, her neurology remained improved over the period, and shunt function was optimal despite the abdominal distention. It is probable that the intra-abdominal pressure has not exceeded the intracranial pressure. At her age, her normal ICP is expected to be < 10–15 mmHg. The normal intra-abdominal pressure is defined as 5–7 mmHg, and intra-abdominal hypertension occurs only when this pressure rises and is sustained above 12 mmHg [11]. The cranial-abdominal pressure gradient and the distensibility of the anterior abdominal wall could have contributed to the continued CSF flow despite the ascites. Since CSF ascites is a diagnosis of exclusion, detailed clinical, radiological, haematological, and biochemical investigations are necessary to rule out common causes of ascites such as protein-energy malnutrition, liver, renal, and cardiac diseases.

Aseptic abdominal paracentesis relieved the abdominal distension but was limited by recurrence. However, it also allowed us the time for multi-disciplinary consultation and treatment planning. Though no tumour cells were detected on cytology of the peritoneal aspirate, there were concerns that tumour cells could be seeded into the right atrium directly, hence we opted for a conversion of the V-P shunt to a ventriculo-pleural shunt in the first instance. This was the first ventriculo-pleural shunt in our centre. The pleural space absorptive capacity increases several-fold, from the baseline rate of 0.01 to 0.02 to 0.22 to 0.28 ml/kg per hour upon introduction of excess fluid [12]. This absorptive capacity should be sufficient to handle CSF presented to the pleura at an expected normal production rate of 0.3–0.5 ml/h and even more. Unfortunately, pleural effusion occurred, necessitating revision within a week of the procedure. Pleural effusion is a recognized complication of the ventriculo-pleural shunt in the literature [1, 5]. Though we had no peritoneal biopsy to confirm peritoneal pathology, it is probable that the same reason for the failure of peritoneal absorption could also affect the pleura since both are mesothelial structures [5]. A V-A shunt was eventually done after a chest tube thoracostomy to drain the effusion. Due to the unavailability of the appropriate V-A shunt hardware, we used the distal catheter of Chhabra shunt hardware for the V-A shunt. The patient remained stable and was successfully worked up for the craniotomy for tumour excision after 4 weeks of no recurrence of ascites.

Conclusion

Though uncommon, CSF ascites is a possible complication of V-P shunt and can occur without obvious CSF abnormalities. The initial tasks of the clinician are to rule out

differential diagnoses, relieve the patient of distress where present, and optimize patients for definitive treatment if applicable. Under such circumstances, it is possible for the shunt function to be preserved despite marked ascites. A V-A shunt is a viable alternative and should be considered ahead of ventriculo-pleural shunt in the absence of any contra-indication.

Declarations

Conflict of interest The authors declare no competing interests.

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