Case Report: Cerebrospinal fluid (CSF) Ascites

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Abstract:

Cerebrospinal Fluid (CSF) ascites is a rare complication after ventriculoperitoneal (VP) shunts. Most patients have gradual abdominal protrusion without any neurological sign or symptoms of shunt malfunction. We present a boy with congenital hydrocephalus who de-veloped increasingly abdominal protrusion twenty two months after VP shunt operation. Ascites fluid examination showed characteristic findings similar to CSF with no evidence of infection or malignant ascites. Patients reported with CSF ascites after VP shunt were re-viewed and presented in detail.

Keywords: Cerebrospinal fluid, Ascites, ventriculoperitoneal shunt
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Introduction

CSF ascites is a rare complication of ventriculoperitoneal (VP) shunt. The peritoneum is unable to absorb the CSF, due to the sheer volume of CSFproduced or due to, as of now, an unexplained pathology in the peritoneum. One child who suffered from ascites secondary to VP shunt are presented here.

Case report

A 2 year old boy with gross congenital hydrocephalus was treated with VP shunt on the 5th day of life. Then he underwent VP shunt revision and CSFleakage repair at age of three months due to subgaleal CSF collection.

This child gradually developed positional Plagiocephaly with peculiar upward migration of both parietal bones with an element of twisting and towering, and inward movement of frontal bones. At age of eighteen months cranial reduction and remodeling surgery was done for him due to this progressive skull deformity. There was no episode of any postoperative surgical site or CSF infec-tion. This child did well for 12 months, He had settled into his new and more rounded skull shape. Clinically and radiologically his Ventriculoperitoneal shunt was working adequately. Neurologically child was progressing, though much behind his expected milestones. His general health was good. He had seizure disorder which was well con-trolled on Phenobarbiton. After almost 12 months of skull corrective surgery patient presented to hospital with abdominal distension and dis-comfort, but no neurological signs or symptoms devel-oped. There was no evidence of shunt malfunction.

Paracenthesis showed transudate (protein=13 g/L) with no evidence of infection (WBC= 35 cells/cm: 11% Neutro-phil, 57% Lymphocyte, 20% monocyte, LDH= 63 U/L) and peritoneal fluid and tissues were negative for TB. In addition, cytology was negative for malignant cell. CSF analysis showed no WBC, Protein= 0.16 g/L, Glucose= 3.1 moL/L. Abdominal Ultrasound and CT scan showed no abnormality relating to Abdominal viscera or perito-neal. This ascites recurred after percutaneous tapping twice.

Based on impression of CSF ascites, right sided Ventricu-loatrial shunt was inserted for the patient and ascites re-solved steadily thereafter with normal abdominal girth at the end of 2 weeks post surgery with no complication at the end of six weeks follow up.

Discussion

Ascites has been defined as 'accumulation of excess fluid within the peritoneal cavity [1]. The commonest cause of ascites is cirrhosis of the liver, closely followed by other serious hepatic diseases [2]. In all of these, however, an underlying disease state can be found. In our patient none of the above two causes existed. It is apparent that the primary problem was in the peritoneum which just failed to absorb the CSF.

CSF ascites is a rare complication of ventriculoperitoneal (VP) shunts. Different intervals (2 months-3 years) between shunt placement and symptomatic ascites have been reported [3-6]. And several etiologic factors had been discussed in literature, but there is no definite expla-nation.

Some explain that imbalance between peritoneal absorp-tion capacity and amount of CSF Production is the major cause. By this definition, patients with excessive amount of CSF production like choroid plexus papilloma are at risk to developing CSF ascites following VP shunt [3,7, 8]. On the other hand, patients with high CSF protein due to chronic infection (tuberculous meningitis) [9] or brain tumors –especially optic glioma [4,8,11] may have diffi-culties in CSF absorption through peritoneum. Peritoneal inflammation due to repeated shunt revisions [5] or non-specific inflammatory response to shunt material [9], play role in the other side and decrease absorptive ability of peritoneum. Also in brain tumors, especially in astrocy-toma and glioblastoma, increased vascular permeability can cause microvascular extravasation of plasma into the peritoneal cavity and cause ascites [12-15]. By all of these, however, most of reported cases have unknown etiology [16-19] and CSF ascites had been resolved in all reported cases by ventriculo-atrial shunt and occasionally by ventriculoperitoneal shunt with upgrading the pressure valve [20].

However, in our patient we did not find out a clear under-lying etiology for CSF ascites at peritoneal level. There-fore, we should take into consideration that CSF ascites can develop as V-P shunt complication even if there is no clear explanation for the underlying etiology.

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