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# Cerebrospinal Fluid Ascites Post Ventriculoperitoneal Shunt after 17 Years


## ABSTRACT

Cerebrospinal fluid (CSF) ascites following ventriculoperitoneal (V-P) shunting for hydrocephalus is an unusual complication. A 29-year-old male with communicating hydrocephalus and V-P shunt *in-situ*, presented with tense ascites. Extensive investigations in the form of complete blood count, renal function test, liver function test, Computerised Tomography (CT) of the brain, Contrast-Enhanced CT of Abdomen, paracentesis was done. Ascitic fluid $\beta_2$-tranferrin test was positive indicating that the ascitic fluid was indeed CSF, therefore a diagnosis of CSF ascites was made. The patient's V-P shunt was converted into a ventriculoatrial shunt. Following the procedure, there has been no refilling of ascites, thus, further confirming the diagnosis of CSF ascites. The etiology of CSF ascites seems to be some unknown pathological process wherein the peritoneum failed to absorb CSF.


**Key words:** Ventriculoperitoneal shunt, Ascites, Ventriculo-atrial shunt

## INTRODUCTION

Ascites is a common symptom encountered by gastroenterologists on a daily basis, however Cerebrospinal fluid (CSF) ascites is an unusual clinical entity. CSF ascites is an infrequent complication of ventriculoperitoneal (VP) shunt. The aetiology of CSF ascites seems to be some unknown pathological process wherein the peritoneum fails to absorb the redirected CSF, from the VP shunt placed, in order to alleviate the complications of hydrocephalus. We are presenting a case of a 29-year-old male with CSF ascites probably due to inefficient peritoneal absorption of CSF.


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## CASE REPORT

A 29-year-old, deaf and mute male, presented with complains of insidious onset, gradually progressing, painless distention of abdomen for the past 4 months. He had no complains of fever, jaundice, abdominal pain, diarrhea, periorbital edema, pedal edema or dyspnea on exertion. He had a past history of VP shunt surgery for communicating hydrocephalus at the age of 2 months in 1991 followed by a shunt revision surgery in 2003.

On examination, patient was stable, with no jaundice, pallor or features of increased intracranial pressure. Umbilicus was transversely stretched and fluid thrill was present. There was no organomegaly. Other systems examination was essentially normal.

The patient had undergone extensive blood investigations and imaging in form of complete blood count (CBC), renal function test (RFT), liver function test (LFT), Computerized Tomography (CT) brain, contrast-enhanced CT scan of the abdomen, whole-body PET-CT. CBC, ESR, LFT, and RFT were within normal limits. CT brain revealed mild to moderate hydrocephalus with shunt *in situ*. CT abdomen (oral and IV contrast) revealed right-sided pleural effusion, massive ascites without loculations and omental nodularity. Liver, spleen, gall bladder, kidneys were normal. CT guided omental nodule biopsy revealed histiocyte rich inflammation of undetermined aetiology. Patient was empirically started on first-line anti-tubercular therapy- rifampicin, isoniazid, pyrazinamide, and ethambutol. However, ascites was persistent. Patient then came to Bombay Hospital.

Therapeutic paracentesis was done and four litres of clear ascitic fluid was removed which revealed a serum-ascites albumin gradient of 0.3 g/dL. Results of all infectious and cytology studies were normal. Ascitic fluid examination, CSF examination and urine routine were not suggestive of any ongoing infectious process. Further evaluation did not indicate the presence of either cirrhotic or noncirrhotic portal hypertension. Ascitic fluid $\beta_2$- transferrin test was positive, indicating that the ascitic fluid was actually, CSF.

Thus, it was concluded that patient had CSF ascites. anti-tubercular therapy was stopped, the VP shunt was surgically

changed to ventriculoatrial shunt. After the surgery there has been no refilling of ascites and patient is completely asymptomatic.

## DISCUSSION

Ascites is defined as the presence of free fluid in the peritoneal cavity. Ascites has varied etiology, most commonly seen as a complication of portal hypertension and nephrotic syndrome. Other causes include-tuberculous peritonitis, peritoneal carcinomatosis, cardiac failure, pancreatic and biliary disorders.[1] In all the above-mentioned conditions, an underlying disease state can be found. In the present case, however, none of the above causes was present. It is, therefore, apparent that the primary problem was an unknown pathology of the peritoneum, which just failed to absorb the CSF.

$\beta_2$-transferrin is naturally present in CSF. Measuring $\beta_2$-transferrin levels is a specific method for identification CSF. β2-transferrin assay is widely used as a screening test for patients with suspected CSF leak.[2] This test is useful in the identification of CSF leaks especially in patients traumatic head injury and episodic otorrhea or rhinorrhea. Here, this test was utilised for identifying the nature of ascitic fluid.

Intra-abdominal complications of VP shunt are often-abdominal abscess, peritonitis and dislodgement of shunt. CSF ascites is an uncommon complication of VP shunt.

CSF ascites is a completely different problem, that can arise due to multiple reasons as elucidated by many case reports in the past. A major cause is an imbalance between peritoneal absorptive capacity and the amount of CSF production. For example, patients with excessive amount of CSF production such as in choroid plexus papilloma are predisposed to developing CSF ascites following VP shunt.[3] Furthermore, patients with high CSF protein content due to chronic infection or brain tumours such as optic gliomas, have difficulties in peritoneal absorption of CSF.[4] Finally, peritoneal inflammation due to repeated shunt revisions or non-specific inflammatory response to shunt material may also play a role in the decreased absorptive capability of the peritoneum. In this case, there was an excessive accumulation of CSF in the peritoneal cavity, probably as a result of an inability of the peritoneum to absorb the CSF due to some unexplained peritoneal inflammatory response.[5]

CSF ascites can develop any time after V-P shunt surgery. In the myriad of cases reported in medical literature, it has been observed as early as 2 weeks after the surgery, to as late as 4 years after the procedure. However, our patient developed CSF ascites 17 years after shunt revision surgery, which is the longest duration reported till date.

Other causes of ascites need to be excluded, by extensively investigating the patient for renal, hepatic and cardiac dysfunction, prior to making the diagnosis of CSF ascites. No renal, hepatic or cardiac dysfunction was found in this patient after thorough investigation.

Treatment of CSF ascites includes diversion of CSF from the peritoneal cavity. The possible solutions include conversion of VP shunts into either Ventriculo-atrial, ventriculo-pleural, or Ventriculo-gallbladder shunts. Ventriculo-atrial shunts are the preferred line of treatment with best short and long-term outcomes.

In this patient, VP shunt was converted into a Ventriculo-atrial shunt with no post-operative complications, good outcome and no recurrence of ascites on follow up in the past 1 year.

## CONCLUSION

Patients of CSF ascites present with insidious onset, progressive and painless abdominal distention. There is no hepatic, renal or cardiac dysfunction. The time period between the placement of shunt and evolution of symptoms can range from weeks to years. Extensive investigations are required in order to exclude other possible aetiologies of ascites. However, in patients with a history of V-P shunt surgery, the possibility of CSF ascites should be kept in mind and can be confirmed by using ascitic fluid $\beta_2$ transferrin assay. After making the diagnosis of CSF ascites, the treatment includes redirection of CSF flow, preferably to the right atrium. The aim of this case report is sensitization of practitioners to this unusual diagnosis and creating new avenues for further discussion.

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