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Case Report

CSF Ascites: Review of articles and a case presentation

R. Pourkhalili MD, A. Mirhosseini MD**, HA. Khalili MD****

ABSTRACT

Cerebrospinal fluid (CSF) ascites is a rare complication after ventriculopritoneal (VP) shunts. Most patients have gradual abdominal protrusion without any neurological sign or symptom of shunt malfunction.
We presented a girl with posterior third ventricle glioblastoma and acute hydrocephalus who developed increasingly abdominal protrusion one month after VP shunt operation. Ascites fluid examination showed characteristic findings similar to CSF with no evidence of infection or malignant cells. Ventriculo-atrial shunt revision cured patient's ascites. Review articles of patients with CSF ascites after VP shunt were presented in details.

Key words: Cerebrospinal fluid, Ascites, Ventriculopritoneal Shunt

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A 12 years old girl presented morning headache, vomiting and blurred vision since one month prior to admission. Brain MRI shows a posterior third ventricle mass with active hydrocephalus (Figure1).

At first, a medium pressure VP shunt placed for the patient and 11 days later, definite operation was done by subtentorial supra cerebellar approach and subtotal resection of the tumor. Pathology reported a giant cell type of glioblastoma multiform. Patient developed progressive abdominal distention (Figure 2) which was sever enough for evaluation 23 days after tumor resection.

Ultrasonography showed diffuse ascites with no loculation. There was no complaint of abdominal pain or discomfort and no abnormal neurological sign or symptom developed. There was no evidence of shunt malfunction. Paracenthesis shows transudate (protein=0.3g/dl) with no evidence of infection (WBC=18, 60% lymph, LDH=40, ESR=15, negative cultures and CRP). Cytology was negative for malignant cell. CSF analysis showed mild post operative change; similar to abdominal tap results (WBC=20, 60%Lymph, protein=22 mg/dl, LDH=20).

Based on impression of CSF ascites, VA shunt revised for the patient and ascites resolved steadily thereafter with normal abdominal girth at the end of next month follow-up.

Discussion:

CSF ascites is a rare complication of ventriculoperitoneal (VP) shunts. Several etiologic factors had been discussed in literature, but there is no definite explanation.

Some explain that imbalance between peritoneal absorption 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 [1]. On the other hand, patients with high CSF protein due to chronic infection (tuberculous meningitis) [2] or brain tumors -especially optic glioma [3, 4, 5]- may have difficulties in

*Assistant professor, Department of Neurosurgery, Isfahan University of Medical Sciences, Isfahan, Iran.
**Assistant professor, Department of Neurosurgery, Isfahan University of Medical Sciences, Isfahan, Iran.
***Resident, Department of Neurosurgery, Isfahan University of Medical Sciences, Isfahan, Iran.
Correspondence to: Dr Reza Pourkhalili, Al-zahra hospital, Sofeh Ave, Isfahan, Iran. E-mail: r_pourkhalili@med.mui.ac.ir

CSF absorption through peritoneum. Peritoneal inflammation due to repeated shunt revisions [6], or non-specific inflammatory response to shunt material [7], play role in the other side and decrease absorptive ability of peritoneum.

In recent years vascular permeability factor (VPF) was recognized as a potent mediator of brain tumor angiogenesis, vascular permeability, and glioma growth [8], which can produce hyperpermeability of peritoneal blood vessels, micro vascular extravasations of plasma fluid and proteins through the intra endothelial space, and ascites [9,10,11]. High concentrations of VPF have been found in the serum and brain tumor tissue of patients with both low- and high-grade astrocytoma that may cause ascites in these patients [8, 10].

By all of these, however, most of reported cases have unknown etiology [12-14] and CSF ascites had been resolved in all reported cases by ventriculo-atrial shunt. Different intervals (2 months-3 years) between shunt placement and symptomatic ascites have been reported [1, 3, 6, 7, 13].

In our patient with glioblastoma, the role of VPF may be the most probable concern and further evaluation for endothelial and vasogenic factors in brain tumors, especially glioma grunted.

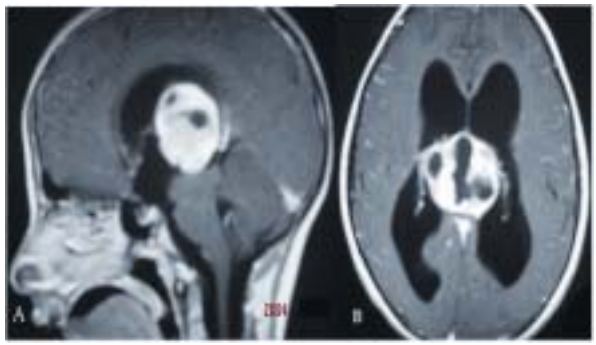

Figure 1. The sagital (A) and axial (B) view of brain MRI T: paramagnet pineal region mass with active hydrocephalus

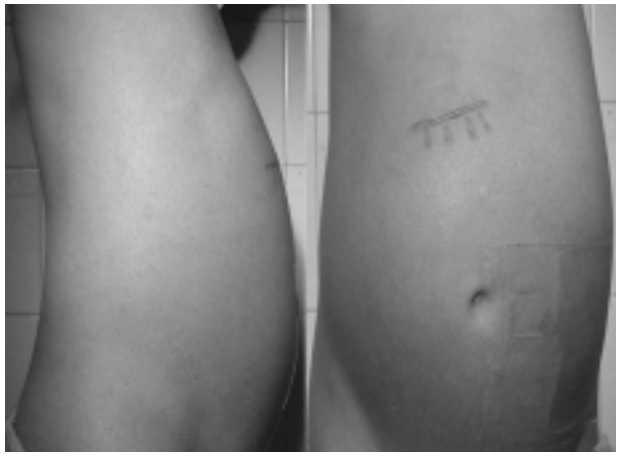

Figure 2. Extensive abdominal protrusion

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