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A case report with literature review

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Delayed cerebrospinal fluid ascites following ventriculoperitoneal shunt. A case report with literature review

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ABSTRACT

Background: Cerebrospinal fluid (CSF) ascites is an abnormal accumulation of CSF within the peritoneal cavity caused by the peritoneum's inability to absorb the CSF, following a ventriculoperitoneal (VP) shunt surgery. Excessive CSF production (e.g. choroid plexus papilloma and choroid plexus villous hypertrophy), high CSF protein secondary to chronic infection (e.g., tuberculosis), and brain tumours (e.g. optic gliomas and craniopharyngiomas) have all been suggested as contributing factors to the formation of CSF ascites. Peritoneal inflammation as a result of several shunt revisions or some non-specific inflammatory reaction to shunt material has also been explored.

Case Presentation: A 3-year-old girl with lumbar myelomeningocele and delayed CSF ascites following VP shunt is reported. Therapeutic paracentesis was employed to relieve abdominal distension, although recurring accumulation was common. The VP shunt was removed and instead of a Ventriculo-atrial shunt, she underwent Endoscopic Third Ventriculostomy (ETV). CSF ascites gradually disappeared after ETV over a two-week period.

Conclusions: Abdominal paracentesis to relieve ascites and conversion of a Ventriculoperitoneal shunt to a Ventriculo-atrial shunt are commonly used to treat CSF ascites, however Endoscopic Third Ventriculostomy, where feasible, is another alternative treatment that can be performed to treat this condition.

INTRODUCTION

The most frequent procedure for hydrocephalus is a ventriculo-peritoneal (VP) shunt. They have been linked to a variety of issues, including dysfunction, infection, blockage, and migration. Rarely, the patient may develop increasing abdominal distention as a result of cerebrospinal fluid (CSF) accumulation. This is commonly referred to as a pseudocyst. The omentum produces a cyst at the tip of the shunt as a result of inflammation, resulting in a fluid-filled sac. Ascites is caused by a production-absorption mismatch or a non-absorbing peritoneum in very uncommon cases.^[46] Ascites owing to hepatic, renal, or cardiac illness must be distinguished from CSF ascites, which is an abnormal buildup of CSF within the peritoneal cavity^[38].

CSF ascites is a distinct condition in which there is an excessive

Keywords
hydrocephalus,
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accumulation of CSF in the peritoneal cavity due to the peritoneum's inability to absorb it. This incapacity could be the result of a large amount of CSF being produced.^[23] There have been several hypotheses on what factors may lead to the development of CSF ascites. Excessive CSF production (as in choroid plexus papilloma and choroid plexus villous hypertrophy), high CSF protein secondary to chronic infection (e.g. tuberculosis), and brain tumours (e.g. optic gliomas and craniopharyngiomas) have all been suggested as

contributing factors to the formation of CSF ascites. Peritoneal inflammation as a result of several shunt revisions or some non-specific inflammatory reaction to shunt material has also been explored.^[46] After ensuring that there is no infection, the VP shunt associated CSF ascites is treated by converting the shunt to a Ventriculoatrial shunt. Endoscopic Third Ventriculostomy (ETV) is another treatment option that can be done to treat this condition in selective cases.



Figure 1. **A:** CSF swelling at reservoir site of VP shunt with distended abdomen and umbilical hernia due to CSF ascites, **B:** Abdominal incisional scar for VP shunt, **C:** Lumbar Myelomeningocele.

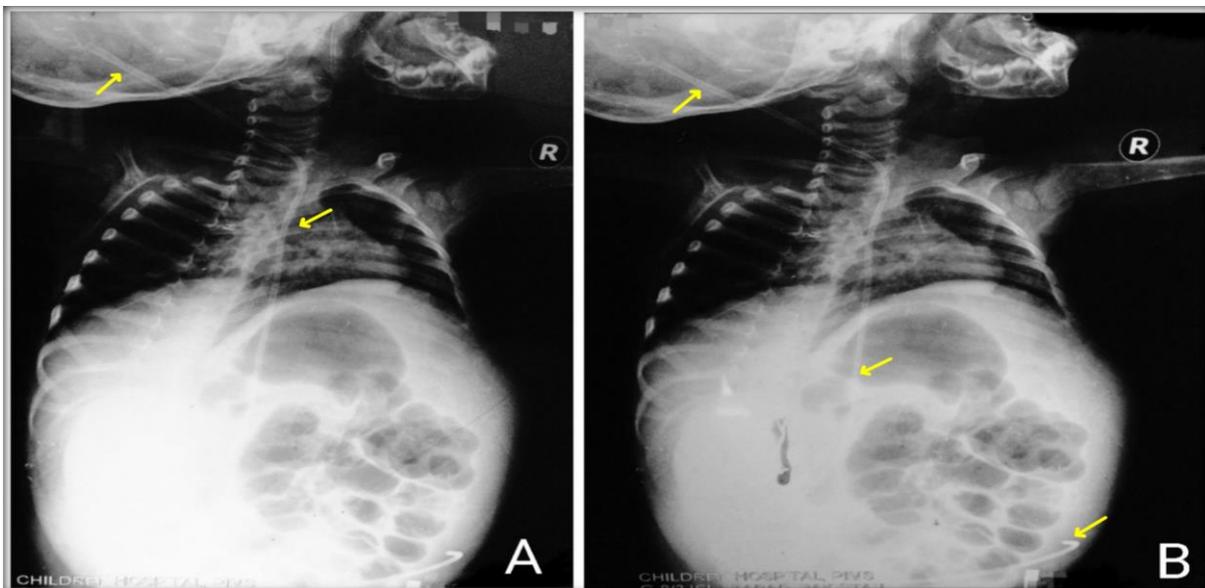


Figure 2. A & B: X-ray shunt series showing VP shunt tubing marked by yellow arrows

Table 1. Detailed literature review of 77 previously reported cases of CSF ascites and their treatment.

S.no.	Author's Name	Year reported	No. of cases reported	Gender/Age	Cause/Type of Hydrocephalus	Treatment for CSF ascites
1	R H Ames ^[1]	1967	1	M/2 years	Congenital hydrocephalus	Ventriculo-atrial shunt
2	D F Dean ^[2]	1972	1	M/1 year	Aqueductal stenosis	Ventriculo-atrial shunt
3	J D Rosenthal ^[3]	1974	1	F/3 years	Suprasellar mass (Astrocytoma)	Ventriculo-jugular shunt
4	Michael J. Weidmann ^[4]	1975	1	F/3 months	Aqueductal stenosis	Multiple Paracentesis followed by Ventriculo-atrial shunt
5	SW Parry ^[5]	1975	1	M/7 months	Communicating hydrocephalus	Ventriculo-atrial shunt
6	J Cummings ^[6]	1976	1	-	Unknown	-
7	R F Lees ^[7]	1978	2	-	Unknown	-
8	J M Noh ^[8]	1979	1	-	Unknown	-
9	S C Ohaegbulam ^[9]	1980	1	-	3 rd ventricle compression by recurrent craniopharyngioma	Spontaneous resolution following Paracentesis
10	A B Adegbite ^[10]	1982	1	F/11 years	Recurrent craniopharyngioma	VP shunt removal leading to resolution of ascites followed by new VP shunt
11	FP Agha ^[11]	1983	1	M/7 years	Suprasellar astrocytoma	Ventriculo-atrial shunt
12	R A Yount ^[12]	1984	4	1-F/6 years, 2-F/20 years, 3-M/4 years, 4-F/3 years	1-Communicating hydrocephalus, 2-Unknown, 3-Optic nerve glioma, 4-Optic nerve glioma	1-Ventriculo-cholecystic shunt, 2-Paracentesis and multiple taps of shunt reservoir, 3-Paracentesis and low-sodium diet, 4-Spontaneous resolution with fluid-restricted diet
13	Gairi Tahull JM ^[13]	1984	1	F/5 years	Communicating hydrocephalus	Ventriculo-atrial shunt
14	DS Rush ^[14]	1985	4	-	-	VP shunt revisions

15	D Madruga Acerete ^[15]	1988	1	F/12 years	Dandy-Walker malformation	-
16	G M Goodman ^[16]	1988	1	F/11 years	Hydrocephalus following lumbar myelomeningocele repair	Ventriculo-atrial shunt
17	S Niikawa ^[17]	1988	1	F/45 years	Post-infectious obstructive hydrocephalus	-
18	F Pérez Peña ^[18]	1990	1	M/17 years	Malformation hydrocephalus	-
19	A Suárez ^[19]	1993	1	M/22 years	Craniopharyngioma	Ventriculo-atrial shunt
20	A West ^[20]	1994	3	1-M/6 months, 2-M/6 years, 3-M/8 months	Optic pathway gliomas	Ventriculo-atrial shunts
21	A Shuper ^[21]	1997	1	F/4 years	Optic chiasm glioma	Ventriculo-atrial shunt
22	Michiko Yukinaka ^[22]	1998	1	F/23 years	Congenital hydrocephalus with spina bifida	Ventriculo-atrial shunt
23	B Chidambaram ^[23]	2000	2	1-F/3 months, 2-M/7 years	1-Congenital hydrocephalus, 2-Optic chiasm glioma	1-Ventriculo-atrial shunt, 2-Ventriculo-atrial shunt
24	Z Gil ^[24]	2001	4	1-M/9 years, 2-M/0.5 years, 3-M/0.5 years, 4-M/4 years	1-Optic pathway glioma, 2-Optic pathway glioma, 3-Optic pathway glioma, 4-Chiasmatic glioma	Ventriculo-atrial shunts
25	BH Lee ^[25]	2001	1	M/68 years	-	VP shunt removal and antibiotics
26	GF Longstreth ^[26]	2001	1	F/28 years	Communicating hydrocephalus	Ventriculo-atrial shunt
27	GY Lee ^[27]	2002	1	F/33 years	Congenital hydrocephalus	Ventriculo-atrial shunt
28	Nayyar Yaqoob ^[28]	2003	1	M/17 years	Tuberculous meningitis	Anti-tuberculous therapy followed by Ventriculo-atrial shunt
29	Raj Kumar ^[29]	2003	4	1-M/9 years, 2-M/2 years,	1-Thalamic glioblastoma,	1-Paracentesis revealed peritoneal mets,

				3-M/4 years, 4-M/8 years	2-Choroid plexus papilloma of 3 rd ventricle, 3-Tuberculous meningitis, 4-Craniopharyngioma	2-Ventriculo-atrial shunt followed by total excision of tumor, 3-Intraperitoneal cyst decompression, 4-Abdominal exploration and VP shunt revision
30	SJ Pawar ^[30]	2003	2	1-M/8 months, 2-M/2 years	Choroid plexus papilloma of posterior 3 rd ventricle	1-VP shunt revision, 2-Ventriculo-atrial shunt
31	Greg Olavarria ^[31]	2005	4	1-M/8 months, 2-M/8 months, 3-F/12 months, 4-F/12 months	Optic chiasmal hypothalamic astrocytoma	1-Ventricular gallbladder shunt, 2-Ventricular gallbladder shunt, 3-Ventricular gallbladder shunt, 4-Ventricular gallbladder shunt converted to Ventriculo-atrial shunt
32	Michael L Diluna ^[32]	2006	1	M/4 years	Arachnoid cyst	Ventriculo-atrial shunt
33	Rajeev Kariyattil ^[33]	2007	5	3 males, 2 females Mean age: 3.7 years	1 Posterior fossa arachnoid cyst, 1 Hemorrhage, 1 Optic chiasm glioma, 1 Craniopharyngioma, 1 Meningomyelocele	4 Ventriculo-atrial shunts, 1 Spontaneous resolution after hypoproteinemia treatment
34	Paik I ^[34]	2010	1	F/21 years	Myelomeningocele	Ventriculo-atrial shunt
35	S Das ^[35]	2010	1	M/7 years	Craniopharyngioma	Ventriculo-atrial shunt
36	N Montano ^[36]	2010	1	F/51 years	Obstructive hydrocephalus due to large Vestibular Schwannoma	VP shunt removal, EVD placement, IV Teicoplanin and multiple ascitic fluid taps
37	WJ Wilma Houtman-van Duinen ^[37]	2011	1	F/29 years	Congenital Cervical Meningocele	Ventriculo-atrial shunt
38	MWANG'OMBE Nimrod Junius ^[38]	2012	1	M/7 years	Aqueductal stenosis	Ascites resolved within two weeks of endoscopic third ventriculostomy
39	Yin Yee Sharon Low ^[39]	2012	1	F/48 years	Brain Mets from Breast Carcinoma with Obstructive hydrocephalus	Peritoneal drain

40	Atakan Comba ^[40]	2013	1	F/6 years	Myelomeningocele	Ventriculo-atrial shunt
41	J Woodfield ^[41]	2013	1	F/1 year	Craniopharyngioma	Ventricular gallbladder shunt converted to Ventriculo-atrial shunt
42	Hira Jamal ^[42]	2016	1	F/37 years	Idiopathic intracranial hypertension	VP shunt removal, antibiotics and Ventriculo-atrial shunt
43	Maheen Siddiqi ^[43]	2017	1	F/16 years	Congenital hydrocephalus	Ventriculo-atrial shunt
44	D Sachan ^[44]	2017	1	M/5 years	Choroid plexus papilloma	Tumor resection and VP shunt removal
45	H Han ^[45]	2017	1	M/20 years	Dandy-Walker Syndrome	Ventriculo-atrial shunt
46	G. Musa ^[46]	2018	1	F/3 years	Communicating hydrocephalus	Ventriculo-atrial shunt
47	AA Khan ^[47]	2018	2	-	-	Endoscopic Third Ventriculostomy
48	Darrick K Li ^[48]	2019	1	F/26 years	Loeys-Dietz syndrome and Congenital hydrocephalus	Multiple therapeutic paracentesis followed by Peritoneovenous (Denver) shunt
49	Saud E. Suleiman ^[49]	2020	1	F/32 years	Hydrocephalus due to brain malformation (corpus callosum agenesis)	Multiple therapeutic paracentesis followed by Ventriculo-atrial shunt
50	George A. Alexiou ^[50]	2021	1	F/60 years	Bilateral frontal meningiomas and Obstructive hydrocephalus	Ventriculo-atrial shunt
51	M Mathew ^[51]	2022	1	F/7 years	Craniopharyngioma	Ventriculo-atrial shunt
52	N Mehta ^[52]	2022	1	M/29 years	Communicating hydrocephalus	Ventriculo-atrial shunt

CASE PRESENTATION

A 3-year-old girl appeared to us as an outpatient with complaints of frequent episodes of vomiting and reluctance to feed for three days, as well as abdominal distention for two weeks. She was born with a lumbar myelomeningocele and congenital hydrocephalus after a normal vaginal delivery at term. At the age of one month, she had a

ventriculoperitoneal shunt placed, but she never had lumbar myelomeningocele repair surgery. She had been doing well since her ventriculoperitoneal shunt surgery until her current presentation. She was lethargic during the physical examination. There was a CSF surge at the VP shunt's reservoir site. Her belly was bloated, and she had an umbilical hernia and rectal prolapse, most likely as a result of excessive

intra-abdominal pressure caused by CSF ascites. (Figure 1; A,B,C). The VP shunt's reservoir was slow. We performed an X-ray shunt series, a Computed Tomography (CT) brain plain, and an abdominal ultrasound. Shunt tubing was found to be totally undamaged on X-ray (Figure 2; A&B). Ventriculomegaly was discovered on a CT scan. An abdominal ultrasound revealed complicated ascites. After a diagnostic paracentesis, the ascitic fluid was revealed to be normal. CSF ascites were confirmed through biochemical analysis. To reduce abdominal distension, therapeutic paracentesis was employed, although recurrent accumulation was common. Instead of a Ventriculo-atrial shunt, the VP shunt was removed, and she underwent Endoscopic Third Ventriculostomy. Following ETV, CSF ascites gradually disappeared over a two-week period.

DISCUSSION

Following the implantation of VP shunts for the treatment of hydrocephalus, a number of abdominal complications have been described. Perforation of the gut, gallbladder, vagina, umbilicus, and volvulus, as well as abdominal CSF encystation and CSF ascites, have all been documented.^[5] Infections^[12,26], malignancies, particularly choroid plexus papillomas and optico-chiasmatic gliomas^[12,20,21,24,31], shunt-disseminated metastasis^[39], and foreign body reactivity to the peritoneal catheter have been linked to CSF ascites.^[23] High protein content of the CSF, particularly in optico-chiasmatic gliomas^[10,20,31]; increased CSF production, as in choroid plexus hyperplasia and papillomas; tumor-secreted vascular permeability factors;^[24] and persistent serosal inflammation are among the different causes reported.^[26,33] To the best of our knowledge, Akdegbite et al. were the first to identify elevated CSF proteins as a probable cause of ascites.^[10]

As little is known about the aetiology of CSF ascites^[4], various mechanisms have been proposed, including subclinical peritonitis, which obstructs lymphatic drainage^[23], elevated CSF protein, which causes peritoneal malabsorption^[10,24,28], and CSF overproduction exceeding absorptive capacity.^[38] Multiple shunt revisions^[2,22], an immunological reaction to vaccination^[2], or shunt degradation^[26] can all cause peritoneal irritation. Chronic illnesses such as tuberculosis^[28] and brain tumours (e.g. optic gliomas and craniopharyngiomas)^[24] have high CSF protein levels. Overproduction of the choroid plexus

papillomas causes ascites.^[30] A diagnosis of CSF ascites is strengthened by comparing the biochemistry of CSF shunt aspirate and ascitic fluid from paracentesis. CSF ascites resolve spontaneously when CSF flow is redirected using a Ventriculo-atrial shunt or ETV.^[38] A previous shunt is linked to a higher rate of ETV failure.^[47] In our situation, ETV was used, and symptoms were resolved within two weeks.

A detailed literature review of 77 reported cases of CSF ascites until March 2022 is given in Table 1. Congenital hydrocephalus, obstructive hydrocephalus, choroid plexus papilloma, craniopharyngioma, and posterior fossa tumour were all common etiological causes. Revision of the VP shunt to a Ventricular-atrial shunt is the treatment of choice for CSF ascites, although revision in choroid plexus papilloma will only relieve ascites with the risk of congestive heart failure and bacteremia. The definitive cure is surgical excision of the papilloma.^[44] Ascites developed and CSF protein levels increased in a craniopharyngioma instance that underwent VP shunt insertion due to hydrocephalus. CSF protein levels were reduced and ascites was alleviated after the recurring tumour was removed.^[10] One patient with ascites and elevated protein levels was treated with a fluid-restricted diet by Yount et al.^[12] According to the literature, the most common treatment overall for CSF ascites is abdominal paracentesis and conversion to a Ventriculo-atrial shunt. The time between VP shunt surgery and the onset of ascites might range from 1 day to 12 years.^[23,38] It appeared in our patient 2.9 years after the VP shunt.

In VP shunting, the peritoneal lining is particularly effective for quick absorption from the peritoneal cavity because it is made up of specialized mesothelial cells that promote rapid lymphatic drainage from the peritoneal cavity into the adjacent lymphatic lacunae.^[39] In dehydrated children, the peritoneum can absorb up to 500 mL of normal saline per 24 hours, and following the osmotic absorption phase, the average fluid absorption rate is 33 mL/h.^[51] CSF ascites is defined as an excessive buildup of CSF in the peritoneal cavity due to the peritoneum's failure to absorb the CSF.^[23] According to literature study, this state of disequilibrium could be caused by primary peritoneal failure, increased CSF volume, increased CSF protein, infections (peritonitis), eosinophilic catheter rejection, immunological response to immunisation, or in

certain cases, no clear cause. CSF ascites has been linked to tumours such as optic gliomas and craniopharyngiomas, possibly due to elevated CSF proteins.^[12, 23, 41] In addition to the probable unique proteins produced by optic gliomas, a disruption in the blood-brain barrier could allow tumour proteins to escape into the subarachnoid CSF, resulting in elevated protein levels and impaired CSF absorption through the arachnoid villi.^[23]

Patients with CSF ascites have an insidious onset, gradual abdominal distention, and no pain. Hepatic, renal, or cardiac dysfunction are not present. The duration between the shunt being placed and the onset of symptoms might range from days to years. To rule out other probable causes of ascites, a thorough study is required. In patients with a history of VP shunt surgery the likelihood of CSF ascites must be kept in mind, which can be validated by an ascitic fluid β_2 transferrin assay. After making the diagnosis of CSF ascites, the treatment includes redirection of CSF flow, preferably Ventriculo-atrial shunt but Endoscopic Third Ventriculostomy is a viable approach in certain circumstances.

CONCLUSIONS

Patients with CSF ascites have an insidious onset, progressive and painless abdominal distention. The time between the shunt insertion and the onset of symptoms might range from days to years. An extensive workup is required to rule out other probable causes of ascites. After ensuring that there is no infection, the VP shunt linked CSF ascites is treated by converting the shunt to a Ventriculoatrial shunt. CSF ascites can be treated effectively by abdominal paracentesis and conversion of the VP shunt to a Ventriculo-atrial shunt, but Endoscopic Third Ventriculostomy is a viable approach in some selected cases.

List of Abbreviations:

CSF: Cerebrospinal fluid
 VP: Ventriculoperitoneal
 ETV: Endoscopic Third Ventriculostomy
 CT: Computed Tomography

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Conflict of interest:

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