CASE REPORT

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Brain metastases as a cause of malignant cerebrospinal fluid ascites: case report and review of the literature

Yin Yee Sharon Low^{*1}, John Thomas¹, Wei Keat Wan² & Wai Hoe Ng¹

Practice Points

- Obstructive hydrocephalus is a known complication of brain metastases.
- The insertion of a ventriculo-peritoneal shunt is usually the recommended treatment of choice for such patients, if surgery for the causative lesions is not a viable option.
- Common complications of ventriculo-peritoneal shunts include local distal occlusion of the peritoneal catheter with resultant blockage, shunt infection, disconnection and implant failure.
- Malignant cerebrospinal fluid ascites as a result of ventriculo-peritoneal shunting is an extremely rare event; the limited number of cases reported in literature are usually pediatric patients with primary brain tumors or germ cell tumors.
- Successful management of malignant cerebrospinal fluid ascites cited in the literature usually includes abdominal paracentesis to relieve the ascites and conversion to a ventriculo-atrial shunt.
- However overall, shunts result in an improved quality of life and increased survival time for the majority of patients with hydrocephalus secondary to brain metastases, despite the possibility of this rare complication. Hence, it is still considered as a reasonable management technique for hydrocephalus secondary to inoperable brain metastases.

SUMMARY The development of cerebrospinal fluid ascites after a ventriculo-peritoneal shunt operation is an extremely rare complication. There have been only sporadic case reports and small series reviews published in regards to this condition. They are usually found in the context of primary brain tumors, usually affecting the pediatric population. We present an unusual case of a patient with known metastatic breast carcinoma to the brain who develops malignant cerebrospinal fluid ascites after a ventriculo-peritoneal shunt insertion. This is the first known adult case of a metastatic breast primary to the brain causing tumor dissemination via a ventriculo-peritoneal shunt.

Presentation of case

A 48-year-old female with a previously treated history of metastatic invasive ductal carcinoma of the breast to the liver and lungs was admitted

for nonspecific symptoms of nausea and vomiting. She had completed six cycles of paclitaxel and carboplatin chemotherapy the year before. The last computed tomographic (CT) scan of

¹Department of Neurosurgery, National Neuroscience Institute, 11, Jalan Tan Tock Seng, 308433, Singapore

²Department of Pathology, Singapore General Hospital, Outram Road, 169608, Singapore

*Author for correspondence: Tel.: +65 6357 7191; Fax: +65 6357 7120; sharon.yy.low@gmail.com

her chest, abdomen and pelvis was conducted 3 months prior to her admission. This demonstrated that both primary and metastatic lesions had reduced in size postadjuvant therapy. No new discerning lesions were noted in the scan. Clinical examination did not demonstrate any obvious focal neurological deficit. She was alert and cooperative, with a full Glasgow Coma Score. Fundoscopy demonstrated no evidence of papilledema.

A postgadolinium contrast T₁ MRI of her brain was ordered. This showed multiple small enhancing nodules in the posterior fossa and to a lesser extent in the supratentorial cerebrum. Marked vasogenic edema in the cerebellum had caused significant compression on the fourth ventricle and aqueduct with resultant obstructive triventricular hydrocephalus. There was no gross abnormality seen in the sellar and skull base (Figure 1). The fluid-attenuated inversion recovery sequence showed evidence of transependymal cerebrospinal fluid (CSF) absorption (Figure 2). The overall impression was that of brain metastases causing obstructive hydrocephalus.

■ Initial operation

The patient underwent a ventriculo-peritoneal (VP) shunt insertion. During surgery, the CSF was noted to be clear but under high pressure upon ventricular cannulation. The remainder of the surgery was otherwise uneventful. A postoperative CT brain scan showed resolution of the hydrocephalus with the tip of the ventricular catheter *in situ* (Figure 3). In addition, her initial symptoms of nausea and vomiting resolved postoperatively. Although the intraoperative

Figure 1. Preoperative coronal T₁-weighted MRI with gadolinium contrast.

CSF results did not show any evidence of raised protein or infection, its cytology reported a few atypical cells suspicious for metastatic carcinoma. Immunostaining with cytokeratin marker AE1/3 decorated a few positive cells. No immunostain for GCDFP-15 and PR were done as there were few cells present for evaluation (Figure 4). The patient was subsequently discharged home and completed outpatient palliative whole-brain radiotherapy, which consisted of a total of 30 Gy over ten sessions.

■ Readmission & management

The patient was readmitted approximately 6 weeks after discharge. According to her family, she was noted to have poor appetite, marked lethargy and reduced cognition for approximately 3 days. However, there were no other constitutional symptoms, such as fever, chills or rigors. The patient denied any complaints of headaches, nausea or vomiting. Upon assessment, she was alert but slow in responding. Her surgical wound sites were well healed and the shunt valve was noted to be compressible and refilling well. However, her abdomen was found to be remarkably distended, although soft and without evidence of tenderness, guarding or palpable masses. Bowel sounds were present and of normal caliber. In addition, there was no clinical evidence of jaundice or hepatic failure, and her serum liver function tests were unremarkable.

A repeat CT brain scan showed recurrence of the previously resolved hydrocephalus with the previously noted VP shunt catheter *in situ*. The cerebral metastatic lesions were noted to be stable (Figure 5). The shunt valve was smoothly tapped under asepsis, and the CSF was sent for cell count and cultures. The results were negative for infection. A repeat CSF cytology showed the presence of malignant cells again. CT of the abdomen and pelvis reported that the markedly distended abdomen was due to newfound ascites. The peritoneal end of the VP shunt was positioned in the right-middle quadrant of the abdominal cavity without localized abscess formation, omental plugging or visceral injury. There were no new discernible enhancing peritoneal masses or lesions, as compared with her previous CT conducted 3 months prior (Figures 6 & 7).

■ Outcome

In view of the new clinical and radiological findings, the impression was that of the distal VP shunt catheter being obstructed due to the

high intra-abdominal pressure from the ascites. An abdominal drainage catheter was inserted under radiological guidance and a large volume of ascitic fluid was drained. The catheter was capped after initial drainage to allow the amount to be drained to be closely monitored in the ward. The yellowish fluid had 45 leukocytes/ μ l and a protein level of less than 1 g/dl. This result correlated with the patient's CSF total protein of 0.3 g per liter. The calculated serum-ascites albumin gradient was less than 1.1 g/dl, indicating that the ascites was not likely secondary to increased hepatic portal pressure. Fluid cultures did not show any bacterial growth after being cultured in the microbiology laboratory for 7 days. However, peritoneal fluid cytology demonstrated malignant cells, consistent with metastases from a primary breast carcinoma (Figure 8). After the transabdominal drainage procedure, the patient became more alert and interactive with her family and medical team, without any further intervention to the VP shunt. However, owing to the extent of her primary disease, conservative management was pursued and she eventually passed away a few months later.

Discussion

VP shunting has proven to be one of the most common and effective methods for relieving hydrocephalus in the history of neurosurgery [1–3]. The peritoneal lining, comprising of specialized mesothelial cells that provide rapid lymphatic drainage from the peritoneal cavity into the subadjacent lymphatic lacunae [4], enables it to be particularly effective for rapid absorption from the peritoneal cavity. However, the insertion of shunts of any kind is associated with a spectrum of known possible side effects. The VP shunt, which is currently the most widely used type of CSF-diversion shunt, is comparatively less prone to cause complications in general. The risk of a VP shunt-related complication has been variably quoted from 1 to 59% across the literature [5–7]. The most common cause of peritoneal shunt malfunction is a local distal occlusion of the peritoneal catheter with resultant CSF flow disruption [8,9]. Other common intra-abdominal complications arising from VP shunts are shunt infection, disconnection, blockage and equipment failure [9–11]. Less common complications include incisional hernia, subcutaneous CSF collection, peritoneal pseudocyst formation,

Figure 2. Preoperative axial fluid-attenuated inversion recovery sequence MRI showing evidence of transependymal exudation of cerebrospinal fluid along both frontal horns.

intestinal perforation, tube migration into the thoracic cavity, navel and scrotum and CSF ascites [9,11,12]. VP shunt blockages may manifest as either local abdominal signs or raised intracranial pressure [10].

Figure 3. Postoperative computed tomographic brain scan showing ventricular tip of ventriculo-peritoneal shunt *in situ* with resolved hydrocephalus.

Figure 4. Cerebrospinal fluid cytology smear showing a few atypical cells with pleomorphic nuclei.

■ The VP shunt as an iatrogenic route for metastasis

In general, ascites usually result from a concurrent illness such as cirrhosis, congestive cardiac failure, nephrosis or disseminated carcinoma-tosis [13]. CSF ascites, however, is an extremely unusual event. The cited risk of this occurring is approximately 2% [14], and its etiology has been found to be unclear in more than 50% of patients [15].

There have been a few theories cited in the literature for the probable cause of CSF ascites. The most common hypothesis is the impaired absorption of CSF via the peritoneum [15,16]. Ascites has been more commonly found to have followed abdominal surgery or previous shunt reconstruction. Under such circumstances, there is a reactive postoperative inflammatory response. This is associated with an increase in leukocytes, impairment of lymphatic flow and a subsequent increase in intraperitoneal protein concentration

due to impaired absorption across the peritoneum, causing ascites. As a result, lymphatic obstruction causes diminished return of fluid to the vascular compartment [17].

The other mechanisms proposed that may contribute to CSF ascites include high protein content of the CSF [18–20], overwhelming increases in CSF production [21–24], tumor-secreted vascular permeability factors [19], serosal inflammation secondary to infective and/or immune causes [16,25], and tumor peritoneal dissemination [25–31]. These factors will be discussed accordingly, with specific focus on the latter in accordance to our case report.

There are authors who hypothesize that high protein content in the CSF can result in an altered absorptive state [20]. In these cases, the increased protein content of the shunted CSF may result in a reversed osmotic effect that causes an accumulation of ascitic fluid [32]. The absorptive capacity of the exit pathways for protein molecules can conceivably be overwhelmed by a massive increase in the number and/or size of the protein molecules. Hence, obstruction of the lymphatic system by cellular debris will likely contribute to ascitic fluid accumulation [11]. In addition, as time goes by, the CSF contents may cause a subclinical inflammatory reaction in the peritoneum, worsening its loss of absorptive capacity [18]. However, if this theory holds, patients with increased protein in the CSF will inevitably develop CSF ascites. In reality, this is not necessarily true for all patients who have VP shunts. Studies conducted over the years have shown that an elevated CSF protein content does not significantly increase the risk of shunt complications [33,34].

In cases of villous hypertrophy of the choroid plexus or choroid plexus papilloma, there may be massive overproduction of CSF, beyond the normal absorptive capacity of the peritoneum, resulting in the accumulation of ascitic fluid [11,21–23,35–37]. In order to resolve this issue, some authors report successful resolution of ascites after re-siting the VP shunt to a ventriculo-atrial shunt [11,16,21,23,35,37].

In their study of CSF ascites in children with optic pathway gliomas, Gil *et al.* found that the incidence of ascites for their series (four out of 12 patients who underwent VP shunting) was not associated with infection, tumor metastasis, size of tumor or multiple shunt revisions [19]. Rather, the authors identified tumor-secreted vascular permeability factors from the primary

Figure 5. Repeat computed tomographic brain scan after readmission.

intracranial disease as a significant contributor to the development of CSF ascites [19].

Despite these cited factors as probable causes for CSF-related ascites, in our patient, the CSF protein level was within the normal range, there was an absence of infection and the nature of the high volume of ascites was transudative.

■ Tumor peritoneal dissemination

The literature cites primary brain tumors and germ cell tumors as common sources of tumoral CSF seeding from VP shunts. However, ours is the first case report of intracranial brain metastases leading to CSF ascites after a VP shunt.

Extraneural metastases of primary brain tumors can occur through both blood and lymphatic vessels [31]. This mode of spread is similar to other types of human cancers, including carcinoma of the breast. The first report of iatrogenic tumor dissemination after CSF diversion was in 1954, after a ventriculo-pleural shunt [38]; the first reported case after a VP shunt was in 1963, where a cerebellar sarcoma seeded through the VP shunt into the peritoneal cavity [25].

In general, factors that influence metastatic dissemination of primary brain tumors after a VP shunt are direct surgery on the tumor immediately prior to the shunt, the period of survival owing to the natural history of the primary intracranial disease and immunosuppressive treatments, such as cranial radiotherapy and prolonged corticosteroid therapy [29].

The etiology of peritoneal metastasis from primary brain tumors, especially for optic pathway gliomas and craniopharyngiomas, has been well described in the literature [11,19,31,39–42]. Most of these tumors often cause compression of the cerebral aqueduct, thus necessitating the insertion of a VP shunt and thus potentially leading to a risk of peritoneal metastases by iatrogenic CSF flow into the abdomen [31]. For tumors in close proximity to the ventricles, such as thalamic gliomas, the tumor mass will be widely exposed to the CSF space [42]. During excisional surgery, there can be direct spillage of malignant cells into the CSF and ultimately into the peritoneal cavity via the VP shunt [29]. Furthermore, protein exuded by the mass into the subarachnoid space may cause an elevated CSF protein concentration [42], which in turn, increases the risk of impaired peritoneal absorption, as previously mentioned.

Figure 6. Coronal computed tomographic abdomen scan showing large amount of ascites (arrowhead points to peritoneal tip of ventriculo-peritoneal shunt).

For germ cell tumors, the literature shows that germinoma of the pineal gland is the most common neoplasm metastasizing extraneurally via a VP shunt; other types of CNS tumors include medulloblastoma, pineoblastoma, ependymoma, endodermal sinus tumors, tetratomas, central neurocytoma [43] and malignant melanoma [19,29–31]. Significant examples are described by Saibara *et al.*, Uchino *et al.*, Pallini *et al.* and Cranston *et al.* All cases involve metastasis of

Figure 7. Axial computed tomographic scan of the abdomen showing large amount of ascites (arrowheads point to peritoneal parts of ventriculo-peritoneal shunt).

pineal region and primary CNS tumors into the peritoneal cavity facilitated through a VP shunt system, resulting in the growth of new abdominal masses whose histopathologies are similar to the intracranial primary [44–47]. However, there has been no previous publication whereby metastases from a breast carcinoma are the cause of malignant CSF ascites.

■ Ascites: here, CSF or otherwise?

Ascites in the setting of metastatic breast carcinoma is uncommon in comparison to other cancer types [48–50]. If it occurs, this is usually firstly, secondary to massive liver metastatic infiltration causing portal hypertension [48]. Next, it can be a result of extensive peritoneal spread developing into malignant ascites [51]. However, the histopathological subtype in such cases is frequently invasive lobular carcinoma, where it is known to spread to gastrointestinal viscera, peritoneum, adrenal glands and bone marrow. By contrast, the other subtype (which the patient was initially diagnosed with) – invasive ductal carcinoma – has a higher propensity to spread locally to the lung and pleura [52,53]. The repeat CT abdomen and pelvis upon readmission confirmed her hepatic lesions remained stable without evidence of portal hypertension. More importantly, no new intra-abdominal lesions were seen, which may indicate peritoneal carcinomatosis. Furthermore, both CSF and ascitic fluid were

subjected to a quantitative protein analysis to assess the serum-ascites albumin gradient, to help exclude portal hypertension as a cause of the patient's ascites [54,55].

Hence, based on the patient's primary histopathology, intra-abdominal imaging results, timing of readmission and bodily fluid analyses, the cause of her new ascites was likely secondary to the accumulation of CSF in the peritoneum.

■ Management

In this case, it is clear that the patient has an aggressive carcinoma that was refractory to standard treatment. The malignant cells present in the CSF fail to be eradicated despite whole brain radiotherapy treatment. Hence the poor control of her intracranial disease is likely the most direct factor contributing to tumor dissemination.

In the acute setting, some form of abdominal intervention and CSF drainage to a site other than the peritoneal cavity, rather than just simple removal of the catheter, is recommended [8,16]. For CSF ascites, peritoneal centesis is the recommended treatment modality of choice and this procedure simultaneously provides information confirming the source of the ascites [13,17]. Ascitic fluid can be obtained for analysis of protein, cell count and cultures, especially for patients without evidence of hepato-renal dysfunction on routine blood chemistry [56]. In addition, the shunt should be externalized to allow confirmation that the ascites is truly a result of excessive CSF accumulation [18].

In the long term, if subsequent shunt revision is required, a proposed treatment of choice for VP shunt-related ascites is the conversion of the shunt to a ventriculo-atrial shunt [16,18,35,40], after ensuring there is no infection. Endoscopic third ventriculostomy, where feasible, can be an alternative procedure to manage this problem. There is evidence from the literature that endoscopic third ventriculostomy offers good palliation for patients with obstructive hydrocephalus secondary to cerebral metastases [57,58]. However, VP shunts remain first-line management for this group of patients at our institution; and they too, are able to confer effective palliative measures in the setting of adequate cancer treatment, as illustrated in the recent extensive review by Lee *et al.* [59]. Lastly, ventriculo-pleural shunting may be hazardous because the pleura is also a mesothelial structure and the same problem may occur there, resulting in hydrothorax [35].

Figure 8. Ascitic fluid cytology demonstrating malignant cells.

There have been proposals to consider shunts with filtering devices, but unfortunately filters have been unsuccessful because of the obstruction posed by tumor cells [26,29,30]. Another possible novel modality proposed is a VP shunt valve that has the ability to enable both CSF shunting and intrathecal therapy [59]. Such a device will firstly, overcome mechanical obstruction by neoplastic cells and simultaneously directly deliver tumor-toxic drugs into the CSF. However, in the meantime, it may be advisable to avoid CSF diversionary shunts if possible, either by adequate tumor decompression in order to restore the natural CSF pathway or by temporizing with external ventricular drainage followed by tumor decompression [29].

However, for our patient, such options were not viable. This was because, firstly, she had multiple metastatic lesions that were not resectable; and secondly, a shunt revision was thought to be ill-advised due to the guarded prognosis from her primary disease.

Conclusion

Despite this well-documented but rare complication, for most patients with hydrocephalus secondary to brain metastases, shunts result in an improved quality of life and increased survival time [31,59]. To date, there have been no other reported cases of clinically significant CSF ascites from a VP shunt accumulating in the absence of abdominal pathology, infection, immunological reaction to the shunt itself, malignancy or inflammatory response to CNS proteins from a CNS malignancy [19]. Sterile CSF ascites

complicating VP shunts remains a diagnosis of exclusion [19].

Nevertheless, based on the available literature and the context of this case, VP shunts should be considered as a possible mode of systemic spread of metastatic tumor [31] and may similarly cause such a complication in patients with intracranial malignancies. Therefore, clinicians need to be aware of this entity, especially when managing patients with intracranial tumors. We need to maintain a high degree of suspicion, with careful clinical and radiologic examinations to help diagnose and treat VP shunt-related complications before they progress to more serious conditions [11]. This is especially relevant for patients with already guarded prognosis from a disseminated primary carcinoma, as demonstrated in our case. Any neurosurgical intervention as a means to improve quality of life should be carefully discussed, considered and monitored during their limited time of survival. In conclusion, the spread of metastases via a VP shunt remains a rare event considering the large numbers of such shunts used presently.

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