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Case Reports

Ventriculoperitoneal shunt malfunction caused by proximal catheter fat obstruction

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ABSTRACT

Ventriculoperitoneal (VP) shunt placement is the mainstay of treatment for hydrocephalus, yet shunts remain vulnerable to a variety of complications. Although fat droplet migration into the subarachnoid space and cerebrospinal fluid pathways following craniotomy has been observed, a VP shunt obstruction with fat droplets has never been reported to our knowledge. We present the first reported case of VP shunt catheter obstruction by migratory fat droplets in a 55-year-old woman who underwent suboccipital craniotomy for removal of a metastatic tumor of the left medullocerebellar region, without fat harvesting. A VP shunt was inserted 1 month later due to communicating hydrocephalus. The patient presented with gait disturbance, intermittent confusion, and pseudomeningocele 21 days after shunt insertion. MRI revealed retrograde fat deposition in the ventricular system and VP shunt catheter, apparently following migration of fat droplets from the fatty soft tissue of the craniotomy site. Spinal tap revealed signs of aseptic meningitis. Steroid treatment for aseptic "lipoid" meningitis provided symptom relief. MRI 2 months later revealed partial fat resorption and resolution of the pseudomeningocele. VP shunt malfunction caused by fat obstruction of the ventricular catheter should be acknowledged as a possible complication in VP shunts after craniotomy, even in the absence of fat harvesting.

1. Introduction

Ventriculoperitoneal (VP) shunt placement is the mainstay of treatment for hydrocephalus in both children and adults [1–3]. However, VP shunting remains vulnerable to a variety of complications [4,5] and VP shunt malfunction remains the most frequent reason for shunt revision [6–8].

The migration of fat droplets into the subarachnoid space after craniotomy [9–11], spinal trauma [12–14], baclofen pump insertion [15], and rupture of intracranial [16–20] or intraspinal canal dermoid cysts [21,22] has been previously described. Recently, two cases of obstructive hydrocephalus due to Sylvian aqueduct obstruction with fat droplets after craniotomy were reported [23,24].

We present a unique case of fat migration into the ventricular catheter of a VP shunt, along with signs of aseptic meningitis and shunt malfunction.

2. Case report

A 55-year-old woman with a medical history of metastatic renal cell carcinoma was referred to the Department of Neurosurgery for headache, nausea, and gait disturbance, which had worsened in the past month. MRI revealed a heterogeneously enhancing mass located in the left medullocerebellar region, with slight fourth ventricle compression (Fig. 1). Surgical removal of the tumor was performed via left paramedian incision and left suboccipital craniotomy. Gross total removal of the lesion was achieved under neurophysiological monitoring; no fat graft was used for closure. The postoperative course was uneventful. Postoperative CT scan was unremarkable and demonstrated a slight decrease in ventricular dilatation. At discharge, 7 days after surgery, the patient's preoperative neurological symptoms had resolved and there was no new neurological deficit. Histopathology confirmed metastasis of renal cell carcinoma.

The patient returned to the emergency room 1 month later, with confusion and gait disturbance and a large pseudomeningocele in the surgical site. Cerebrospinal fluid (CSF) examination revealed slight hypoglycorrhachia of 36 mg/dL (*versus* an expected level of 66–77 mg/dL given the patient's blood glucose level of 111 mg/dL), with hyperproteinrrhachia of 2324 mg/L (normal level <450 mg/L), with no microorganisms on microscopy, and negative culture. The sample was bloody, precluding a polymorphonuclear leukocyte count. Head CT scan without contrast after emergency room readmission demonstrated ventricular enlargement with transependymal edema, with no apparent CSF pathway obstruction. Fat droplets were noted in the superior aspect of the lateral

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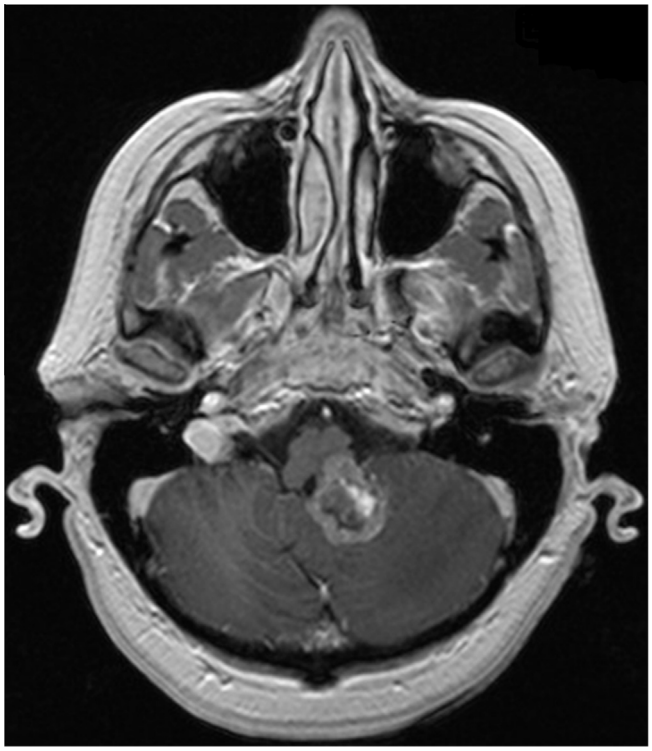

Fig. 1. Axial T1-weighted gadolinium-enhanced MRI showing a heterogeneously enhancing mass located in the left medullocerebellar region, with slight fourth ventricle compression, in a 55-year-old woman with a history of metastatic renal cell carcinoma.

ventricles (Fig. 2). Despite the fat droplets in both lateral ventricles, a right frontal Strata II programmable shunt (Medtronic, Minneapolis, MN, USA) with opening pressure of 1.5 (corresponding to 7.0 cm H_2O prone and 8.5 cm H_2O standing) was inserted without complications. The patient's symptoms completely resolved and the pseudomeningocele disappeared 2 days later, and she was discharged in a good condition.

The patient returned to the emergency department 21 days after the second discharge, with intermittent episodes of confusion, gait disturbance, and a large occipital-suboccipital pseudomeningocele. New CSF examination revealed similar parameters (glucose 50 mg/dL, protein 1425 mg/L, negative culture, polymorphonuclear leukocytes 10 cells), and bacterial meningitis was again ruled out. T1-weighted sagittal MRI performed at this admission demonstrated fat particles in the left Luschka foramen, lateral ventricles, and ventricular catheter, suggesting retrograde fat particle migration from the surgical site through the ventricular system with deposition into the ventricular catheter. An occipital-suboccipital pseudomeningocele due the VP shunt malfunction was evident (Fig. 3). Attempts to reduce shunt opening pressure to a minimum did not improve the pseudomeningocele or reduce the size of the ventricles. Steroid treatment for aseptic "lipoid" meningitis was initiated with dexamethasone 4 mg/day for 2 weeks.

At 1 month follow-up, the patient had no neurological deficits and complete resolution of the pseudomeningocele. At 2-month follow-up, T1-weighted sagittal MRI revealed partial absorption of the earlier fat deposition in the lateral ventricles and partial resolution of fat obstruction in the ventricular catheter (Fig. 4).

3. Discussion

We present a case of VP shunt malfunction due to obstruction of the ventricular catheter by fat droplets that had migrated from soft tissue in the craniotomy site.

We hypothesize that the patient developed partial shunt obstruction leading to development of the pseudomeningocele, which the led to CSF pressure elevation. Along with this, the patient also developed signs of aseptic meningitis. The clinical signs, confusion and ataxia, may be attributed to both aseptic meningitis and VP shunt malfunction. To the best of our knowledge, this is the first report describing fat droplet migration into a VP shunt causing catheter obstruction. Fat droplets most likely entered into the fourth ventricle through the left foramen of Luschka, passing through the Sylvian aqueduct into the third ventricle, then through the foramen of Monro into the lateral ventricles, and thus to the proximal catheter. The upward flow of CSF into the VP shunt and low fat density may have facilitated fat droplet migration into the catheter.

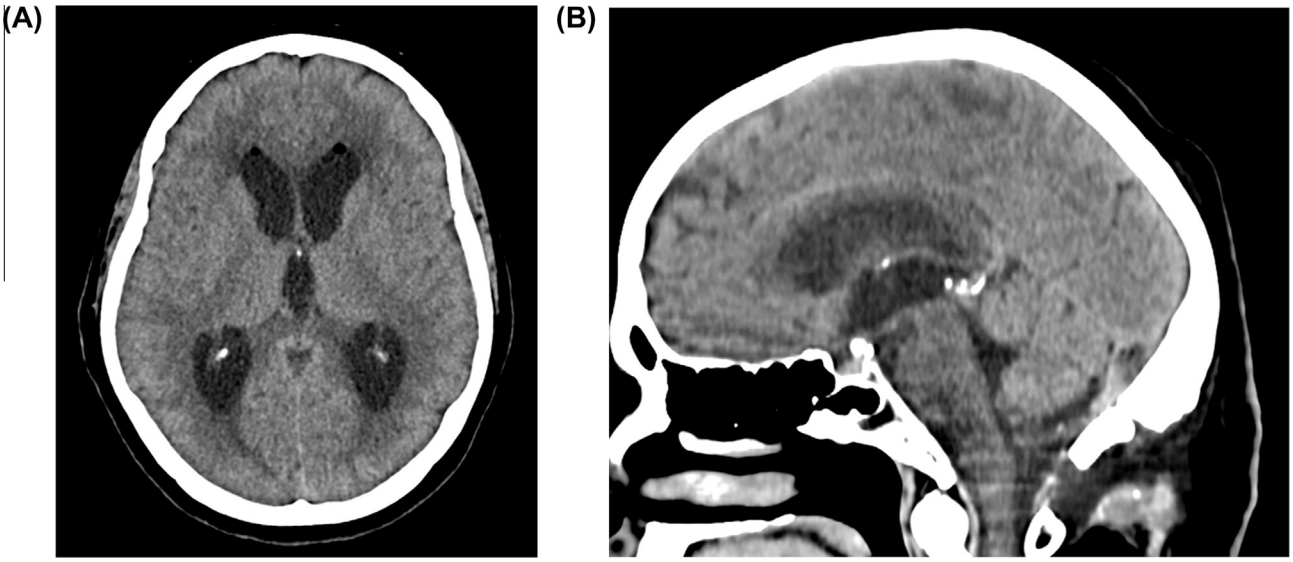

Fig. 2. (A) Axial and (B) sagittal head CT scan without contrast after emergency room readmission demonstrates ventricular enlargement with transependymal edema, with no apparent cerebrospinal fluid pathway obstruction. Fat droplets are noted in the superior aspect of the lateral ventricles.