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Intraparenchymal pericatheter cyst: a rare but important manifestation of cerebrospinal shunt failure

Diogo Marques, Filipa Castelão, Ricardo Pimentel Silva, Ana Cristina Rios

Neuroradiology Department, Hospital Garcia de Orta, Almada, Portugal

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To the Editors

Ventriculoperitoneal (VP) shunt is one of the most commonly performed procedures in neurosurgery, and is used in a variety of diseases including hydrocephalus and idiopathic intracranial hypertension (pseudotumor cerebri).

Shunt failure is a frequent problem, with up to 1/3 VP shunts failing within 12 months after placement, and 50% of patients requiring shunt revision during the first six years. Among the most prevalent complications are shunt blockage and infection [1]. Intraparenchymal pericatheter cyst is a rare complication of a shunt procedure, and is mostly seen in children [1, 2].

We present the case of a four-year-old girl with a history of type 1 Chiari malformation, who had been submitted to VP shunt and median suboccipital craniectomy two years before the current admission, at which she had been suffering from persistent generalised headache and recurrent vomiting of three days' duration. On clinical examination, she presented neck stiffness, without any other meningeal signs or focal neurological deficits. Laboratory studies, including inflammatory parameters, were unremarkable. Blood and cerebrospinal fluid (CSF) cultures were sterile.

On admission, head computed tomography (CT) scan showed a large hypodense subcortical area surrounding the proximal (ventricular) catheter of VP shunt (Fig. 1, G). To better characterise this finding, a brain magnetic resonance imaging (MRI) was obtained. This revealed a well-defined, homogenous cystic lesion surrounding the proximal catheter with signal similar to CSF in all sequences (hyperintense on T2-weighted with attenuation on T2/FLAIR, and hypointense on T1), facilitated diffusion, and adjacent extensive vasogenic oedema. No enhancement was observed on T1 post-gadolinium sequence. This lesion was associated with moderate locoregional mass effect (Fig. 1, A-F). A retrospective analysis of the initial brain CT showed that the proximal catheter was disconnected from the valve of the VP shunt, suggesting shunt failure (Fig. 1, H).

Based on the clinical and radiological features, the diagnosis of intraparenchymal pericatheter cyst due to VP shunt dysfunction was established. Shunt revision was performed, with gradual clinical improvement over the following days. Follow-up CT scan after two months revealed near-complete resolution of the intraparenchymal pericatheter cyst, without symptomatic recurrence.

The occurrence of pericatheter CSF oedema and intra-parenchymal pericatheter cyst has been associated with CSF shunt dysfunction, due to outflow increased resistance or obstruction [1, 3, 4].

The exact pathogenesis of pericatheter collections remains unclear. The pressure differential between the intraventricular CSF and brain parenchyma is thought to be the main reason for CSF flow into the parenchyma. Two possible pathways in the formation of pericatheter CSF collections have been hypothesised: either via the fenestrations on the proximal segment of the proximal (ventricular) catheter, or via backwards flow along the channel that was created during its insertion (focal weakness in the ependymal surface) [2–5]. It appears that CSF is not able to flow back to the ventricular system due to a one-way valve mechanism, leading to the gradual development of

Address for correspondence: Diogo Marques, Neuroradiology Department, Hospital Garcia de Orta, Avenida Torrado da Silva, 2805–267 Almada, Portugal; e-mail: diogomarques1993@gmail.com

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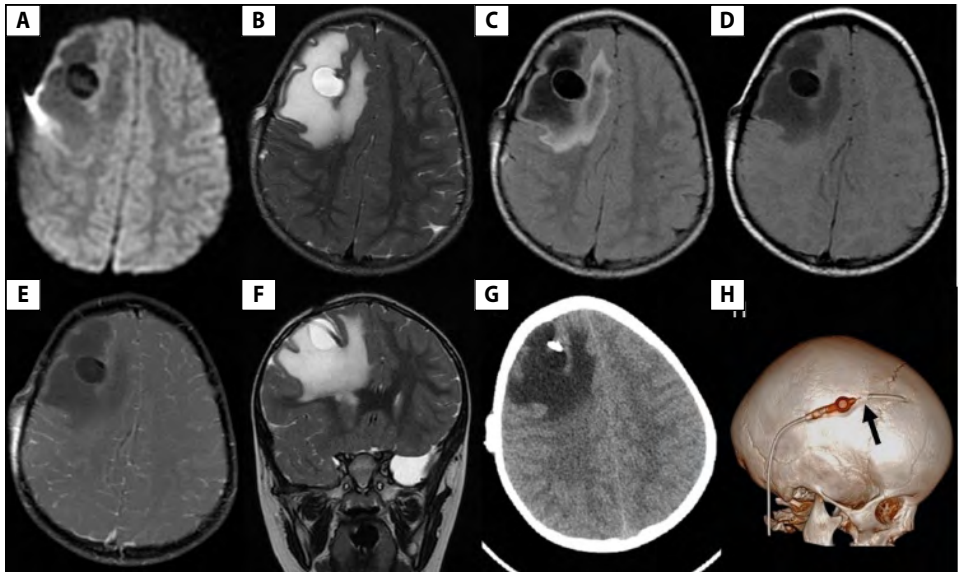

Figure 1. Brain magnetic resonance imaging (MRI) study in axial [DWI (A), T2 FSE (B), T2/FLAIR (C), T1 SE (D), T1 FatSat after intravenous gadolinium injection (E)] and coronal [T2 FSE (F)] views, demonstrating a homogenous, well demarcated cystic intraparenchymal pericatheter lesion with signal similar to CSF in all MRI sequences, without diffusion restriction, and associated with surrounding frontal vasogenic oedema. No enhancement was observed on post-gadolinium T1 fat-saturated sequence €. On axial non-enhanced head computed tomography (CT) (G), there was a large hypodense subcortical area adjacent to proximal catheter compatible with finding presented on MRI. 3D CT bone reconstruction (H) depicted disconnection between proximal catheter and valve of ventriculo-peritoneal shunt (*black arrow*)

pericatheter oedema and cyst (with or without ventriculomegaly) [4]. Pathologically, this lesion is a pseudocyst, given the absence of an epithelialised wall [2].

These intraparenchymal cysts are often preceded by pericatheter CSF oedema. The progression of CSF oedema to a cyst depends on various physiological factors such as the acuteness of shunt obstruction, the tautness of the ventricular wall, the degree of pericatheter gliosis, and the compliance of brain parenchyma [4]. Brain consistency at the time of the shunt might explain the higher incidence in the paediatric population, whose parenchyma is softer and more compliant [2, 4]. Astrogliosis often develops along the shunt catheter regardless of prior pericatheter haemorrhage or local radiotherapy, promoting pericatheter cysts [5].

Brain MRI plays an important role in differential diagnosis, mainly with tumours, infections (cerebritis and/or abcess) and ischaemic strokes. On MRI, these pericatether lesions have thin-wall cysts with CSF signal in all sequences, often associated with low attenuation and high T2/FLAIR signal in the surrounding parenchyma, consistent with vasogenic oedema. The lack of enhancement after contrast injection and diffusion restriction can be very useful in differential diagnosis [1–3, 5]. Additionally, in some cases, VP shunt nuclear scintigraphy (usually with Indium-111 disodium pentetate tracer) can be performed to evaluate the patency and function of the VP system [1].

Management of these patients remains controversial. Shunt revision is recommended, even though there have been a few cases in the literature treated with a conservative surgical approach (cyst aspiration).

Pericatheter cyst is a rare VP shunt complication, and is more commonly seen in children. The pathogenesis remains unclear but is thought to be multifactorial. Imaging studies, such as MRI and CT, have an important role to play in the diagnosis of these lesions, improving the management of these patients.

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