

Dissecting spontaneous cerebrospinal fluid collection

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ABSTRACT

Hydrocephalus is a common condition in the pediatric population known to have many causes and presentation patterns. We report from the analysis of 2 cases the existence of a new complication of pediatric hydrocephalus. Naming this entity “dissecting intraparenchymal cerebrospinal fluid collection”, we advance a hypothesis regarding its pathophysiology and discuss its clinical implications and management.

Introduction

Hydrocephalus is a common problem in the pediatric population, affecting 1.1 in 1000 infants [16]. With a myriad of causes harboring different pathophysiology, it comes to no surprise that this condition can present in many forms and contexts. Based on two patients, we report, to our knowledge, a previously unreported entity complicating hydrocephalus in the pediatric population. We put forward a hypothesis for their formation and confront it to the current literature regarding similar entities. The unique clinical implications of this condition are also discussed.

Methods

After approval from our institution’s ethics committee, the medical files, operative reports and imaging of 2 cases of pediatric hydrocephalus complicated with a dissecting intraparenchymal cerebrospinal fluid (CSF) collection were retrospectively reviewed. Data collection focused on neurological signs and symptoms, operative findings and imaging characteristics of these collections. Furthermore, all existing brain imaging for the 2 cases were extensively reviewed and correlated with radiological reports.

Results

Case 1

A preterm male neonate was born at 31 weeks of gestation from an uncomplicated vaginal delivery. Serial transfontanel ultrasounds revealed a Papile grade 2 germinal matrix hemorrhage (ventricular hemorrhagic spillage without hydrocephalus) on the right side 4 days

after birth (Fig. 1). Magnetic resonance imaging (MRI) done at 56 days showed slightly dilated ventricles and a small arachnoid cyst on the left temporal pole (Fig. 2). No other abnormalities were detected. Given the reassuring neurological exam showing no sign of intracranial hypertension (normal cranial perimeter, reactive neonate, and depressible anterior fontanelle), a clinical and radiological follow-up was carried out. However, the patient was lost to follow-up. At 24 months, the patient was brought in for repeated vomiting, ataxia and irritability since 4 days. Of note, a delay in language acquisition has been diagnosed in the past months. Emergent MRI revealed communicating hydrocephalus and a new fluid collection along the left superior temporal gyrus in communication with the lateral ventricle (Fig. 3). The patient was operated on the day after for a ventriculo-peritoneal shunt (VPS). During surgery, opening pressure of the ventricular system was measured to be very high (above 40 mmHg). The patient improved steadily in the immediate post-operative period, with resolution of the vomiting episodes and irritability. On last follow-up 1 year after surgery, there was diminution both the hydrocephalus and the intraparenchymal temporal collection’s size (Fig. 4). The patient presented no neurological deficit except for a persisting delay in language development.

Case 2

A post-term female neonate was born at 41 weeks of gestation from an uncomplicated vaginal delivery. She presented at 1 month with macrocrania, splayed cranial sutures, tense anterior fontanelle and slight general loss of tonus. She also harbored a discrete paresis of the upper left extremity. Emergent CT scan revealed a triventricular hydrocephalus sparing the fourth ventricle related to aqueduct stenosis. The CT also showed a right frontal collection in communication with the lateral ventricle (Fig. 5). A right sided VPS was carried out on the

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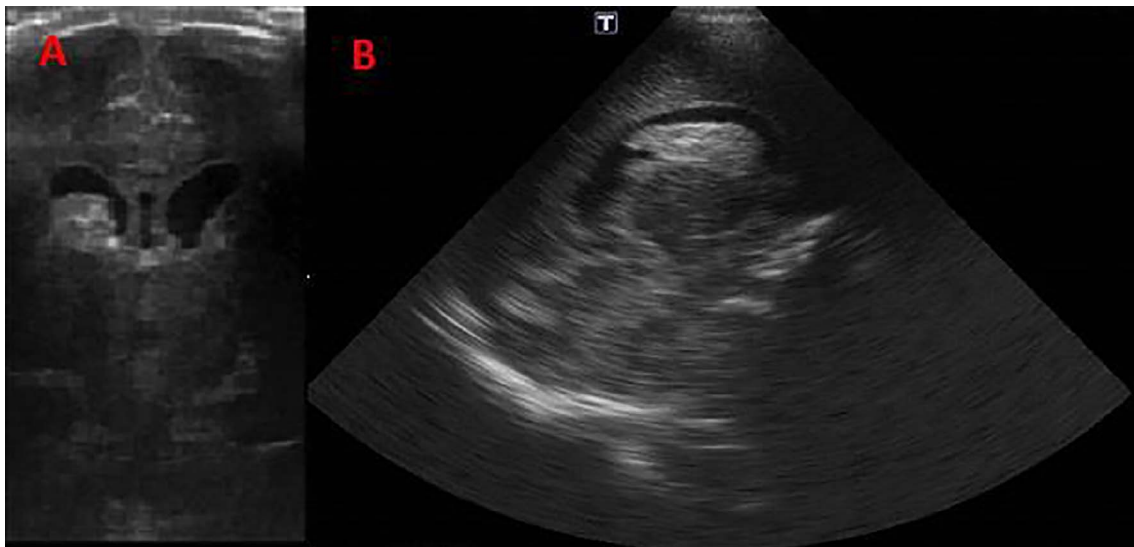


Fig. 1. Transfontanelle ultrasound of case 1 performed 4 days after birth showing a Papile grade 2 germinal matrix hemorrhage with ventricular spillage and normal ventricular size. (A) Coronal view. (B) Sagittal view.



Fig. 2. T2 weighted brain MRI of case one done at 56 days of life showing slightly enlarged ventricles and a small arachnoid cyst on the left temporal pole (arrow). (A) Axial view centered on the frontal horns of the lateral ventricles. (B) Axial view centered on the temporal horns of the lateral ventricles. (C) Coronal view.

same day as the radiological exam to treat the hydrocephalus and resulting intracranial hypertension (Fig. 6). The patient improved in the immediate post-operative period with normalization of the cranial perimeter and re-approximation of the cranial sutures. Distal catheter malfunction warranted a shunt replacement 2 weeks later. Last follow-up at 4 months of age was reassuring with normal cranial perimeter and no clinical sign of intracranial hypertension. A slight upper left extremity weakness was however still present. MRI done at the same time confirmed the diagnosis of aqueduct stenosis and showed a diminution of the size of the ventricles as well as the right frontal collection (Fig. 7).

Discussion

We present 2 cases of parenchymal CSF collections accompanying hydrocephalus in pediatric patients. The cause of hydrocephalus was in one case the blood deposit from an intraventricular hemorrhage and aqueduct stenosis in the other. Although intraparenchymal CSF collections are a known entity, this new unreported pathology differs from

those already known in literature. Table 1 summarizes the other causes of parenchymal CSF collection in the pediatric population [1–3,5,6,10–12,14–16]. We suggest that the CSF collections reported in the current article are a consequence of the hydrocephalus and elevated pressure on the ventricular wall, dissecting the wall with subsequent creation of the periventricular collection. Hence, the term “dissecting cerebrospinal fluid collection” or “dissecting CSFoma”, would be appropriate to name this new entity.

Many elements in our 2 cases support this hypothesis. First, imaging in case 1 predates the appearance of the CSFoma, ruling out a pre-existing congenital collection or porencephaly. Even though a small ipsilateral middle fossa arachnoid cyst was present, the CSFoma did not come in contact with the later. Furthermore, among the main differential diagnosis for this condition would be porencephaly secondary focal necrosis in the periventricular white matter (periventricular leukomalacia, PVL). PVL, documented both in preterm and term infants, is thought to be the result of an *in utero* ischemic insult [9,15]. PVL could give rise to periventricular collections both by an *ex vacuo* dilatation of

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