

Case Report

Arachnoid Cyst Burst in Presence of Contralateral Nomura IV Subdural Hematoma: After Effects of Traumatic Kickback and Intracranial Pressure

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Abstract

Intracranial arachnoid cysts are benign fluid-filled sacs located between the brain and the arachnoid membrane. Their rupture is an uncommon event, often associated with trauma or an underlying pathology. Here, we present a case of a 61-year-old woman with a ruptured arachnoid cyst coinciding with a contralateral subdural hematoma. The patient manifested a persistent headache, balance disorders and right facial paresthesia. Brain CT study showed a right temporo-polar cyst (known to the patient) and a right parietal chronic Subdural Hematoma (cSDH). Although the kickback may contribute to bursting or weakening of cyst integrity, the CT-density pattern of the cSDH, according to Nomura classification is to be considered associated with increased fibrinolysis activity and potentially with parenchyma compression. This case highlights the importance of considering arachnoid cyst rupture as a potential indirect consequence of head trauma kickback, particularly in the presence of contralateral intracranial hemorrhage.

Keywords: Arachnoid Cyst Break; Subdural Hygroma; Chronic Subdural Hematoma; Nomura Classification; Head Trauma Kickback; Local Intracranial Hypertension; Diffuse Intracranial Hypertension; Cerebral Compliance

Abbreviations

SDG: Subdural Hygroma; CSF: Cerebrospinal Fluid; cSDH: chronic Subdural Hematoma; IAC: Intracranial Arachnoid Cyst

Introduction

Each intracranial expansive lesion can lead to hypertension and parenchyma compression.

Increased endocranial pressure can be distinguished into local and diffuse [1]. Local forms although starting on one side may nevertheless involve, when compliance mechanisms are no longer compensating, the contralateral hemisphere as well and become global. The chronic subdural hematoma (cSDH) is a collection of old blood in the subdural space, generally post-traumatic and due to bridging veins tear [2,3]. Its expansion is not only associated with the rebleeding but also with osmotic mechanisms triggered by hemoglobin-degrading proteins [4]. When it results in parenchyma compression and midline shift, it may represent neurosurgical emergency. Nakaguchi classification can be used to analyze the morpho-radiological pattern of blood collection, correlating with the risk of postoperative recurrence [5]. In some cases, it can be applied Nomura classification, which correlates fibrinolytic activity and expansion tendency with CT-density degree [6].

Intracranial Arachnoid Cysts (IACs) are Cerebrospinal Fluid (CSF) collections that develop subdural and consist of arachnoid membrane walls. They can be congenital and secondary (post-traumatic, post infective) [1]. Severe genetic pathologies are related with IAC such as Down Syndrome, mucopolysaccharidosis, neurofibromatosis, polycystic kidney disease (ADPKD), acrocallosal syndrome and Aicardi syndrome [7-15]. One of the most common hypotheses is the splitting of the arachnoid membrane during embryogenic development of subarachnoid cisterns [8]. Being most asymptomatic the diagnosis is like incidental findings in 25% of cases and the treatment choice is conservative management [16-20]. When symptomatic these can cause headaches, seizures, visual disturbances, gait disturbances, nausea, vomiting, developmental delays (in children) and changes in behaviors. Although they can arise at any site of the skull, the main localization is the middle fossa (34%), the posterior fossa or retro-cerebellar (33%) and the cerebral convexity (14%) [2]. Galassi classification divides middle temporal fossa cysts according to the extent of the mass effect into three types:

- Type I: small lens-shaped cyst communicating with subarachnoid space and limited to the anterior portion of middle cranial fossa
- Type II: sparsely communicating with subarachnoid space, displacing the temporal lobe when extends along the Sylvian fissure
- Type III: large cyst not communicating with subarachnoid space, that can occupy whole middle fossa, displacing multiple lobes up to determine midline shift [21]

Symptomatic cysts in younger age may be associated with hydrocephalus. Approximately 6% of arachnoid cysts present a rupture which can occur spontaneously or because of trauma [22].

Case Description

A 61-year-old woman comes to the emergency department following a head injury three months earlier and onset of headache. The patient underwent a brain CT study, which documented the presence of a right temporo-polar arachnoid cyst (already known to the patient). One month before admission, due to the persistence of the headache, balance alterations and paresthesia in the right hemiface, she returned to the emergency department, repeated the brain CT study, which documented the presence of a minimal layer of left parietal cSDH and an extensive hemispheric layer of liquid-like density on the right, in communication with the known arachnoid cyst. At the time of admission, the patient did not remember any head injury with epicenter in the right temporo-parietal region. The patient had a positive family history of hydrocephalus pathology, in fact her brother had undergone ventriculo-vesical shunt surgery. The patient in anamnesis reported sleep disorder, arterial hypertension and gastro-oesophageal reflux disease. Patient therapy consisted of pantoprazole, olmesartan medoxomil and bisoprolol. In her remote history she reported two previous miscarriages, a left breast lump and surgical removal of two uterine polyps. The patient on admission has a GCS 15/15, multiple oscillations on the Romberg test and headache in the nuchal and vertex area [23-30].



Figure 1: This image shows the right temporo-polar arachnoid cyst.

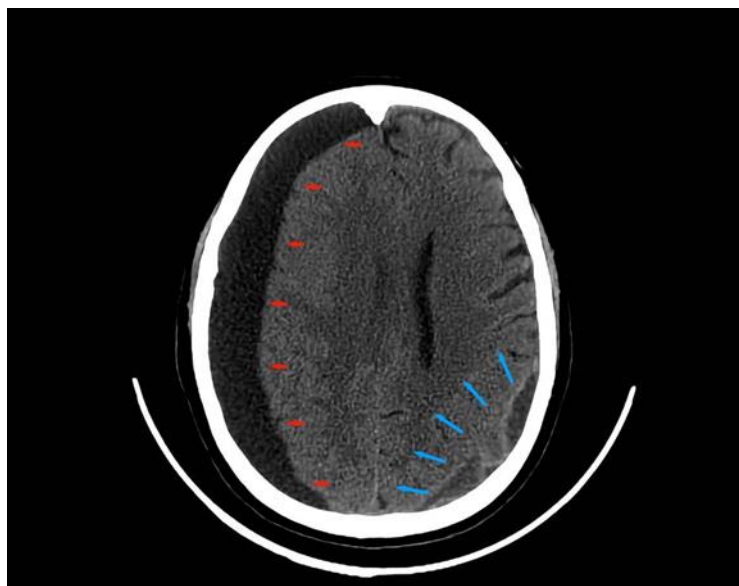


Figure 2: This image indicates the presence of the left chronic subdural hematoma and the right hygroma. The red arrows indicate the compression exerted by the hygroma, while the blue arrows indicate the compression vector forces determined by the cSDH.



Figure 3: This image shows a septum of the primitive cyst.

Discussion

Post-traumatic intracranial injuries starting in one hemisphere may directly or indirectly involve the contralateral hemisphere. Some intraparenchymal hemorrhages by destroying periventricular structures may reach the ventricles or contralateral hemisphere through central structures such as the thalamus or corpus callosum. Acute rapidly growing subdural hematomas may open into the arachnoid space and cause subarachnoid hemorrhage with hemoventricle and global irritation of the cerebral cortex. Hemorrhagic lesions characterized by an acute phase are with greater risk of expansion leading to a rapid increase in endocranial pressure. Chronic lesions, however, may increase due to osmotic phenomena or due to rebleeding [29]. Both types can initially result in an increase in local endocranial pressure that if not resolved in time can become diffuse and affect ventricle-parenchymal structures overall [14,18]. Use of anticoagulants can result in an increased risk of membranes rupture, which are often characterized by important neoangiogenesis and lead to risk of acute bleeding with increased volume of SDH thus parenchymal compression. Patient in our case never used antiplatelet or anticoagulant drugs but the presence of a layered and membranous hematoma is to be correlated with increased fibrinolytic activity during which degradation of fibrinogen and fibrin

occur. Hematoma growth is related not only to an increase in hematic content (rebleeding) but also to an increase in molecules with high osmotic power involving fluid recall and SDH expansion [19,24]. Mixed-density and layering type cSDHs (respectively 4th and 5th grades in Nomura classification) are those characterized by higher hyperfibrinolytic activity, with greater risk of rebleeding and osmotic expansion [22]. When a CT-morphological pattern cannot be captured, Nomura's classification focusing on CT-density can be used. In fact, this classification, unlike the better-known Nakaguchi's, analyzes the correlation between blood density pattern and fibrinolysis degree and, through the D-Dimer degradation activity, the tendency for expansion and re-bleeding [21,22]. Surgery of hematomas with such features progressed over the years and to date is associated with a significant decrease in the risk of recurrence, especially when integrating the middle meningeal artery embolization with procedures of hematoma draining by neuroendoscopy, the latter technique in fact allowing better visualization of the subdural space facilitates in membranes coagulation and aspiration of acute clots [7]. It's probable that at an early stage the osmotic growth secondary to hyperfibrinolytic activity of hematoma resulted in an increase of endocranial pressure and midline thrust, leading to compression of left lateral ventricle, as was evident in brain CT study [18]. However, the contralateral presence of an arachnoid cyst may have attenuated the midline dislocation by dampening the compression onto anatomic structures [17]. De Brito Henriques and colleagues described the case of a 10-year-old boy with hygroma ipsilateral to a temporal IAC and a contralateral fronto-parietal acute SDH exerting mild compression [11]. Once the cerebral compliance mechanisms run out, vector forces of intracranial hypertension starting from the left subdural space (cSDH) and directed into the contralateral hemisphere could have resulted in excessive crushing of the cyst walls to bursting it with overflow into the subdural space [6,13]. Most hygromas that do not cause symptomatology can be observed and treated conservatively, those as in the case described in which compression symptomatology appears require surgical treatment (subdural hygroma drainage by craniotomy or subduro-peritoneal shunt) [26,30]. According to literature, the lesions associated with a lower probability to increase endocranial pressure, since they are generally associated with stationary behavior, are IACs and Subdural Hygroma (SDG). There are, however, cases in which these lesions by a supply mechanism called "ball-valve" may undergo expansion. The valve-like mechanism could originate from the median wall of the cyst that corresponds to the peri-encephalic arachnoid above pia mater of temporal cortex. Superficial and cortical cysts are treated by fenestration or shunt. However, in recent years neuroendoscopy is also having considerable use for sylvian or cortical cysts, although the main indications were the treatment of suprasellar, quadrigeminal and posterior cranial fossa cysts. The most difficult to endoscopically attack are the temporobasal cysts. Endoscopic fenestration has proven to be an effective and safe technique with a low rate of permanent complications [10,23]. The abrupt rupture of the cyst wall contributed on the one hand to fill the subdural space with CSF (hygroma) but also to create a possible communication breach with the arachnoid space that promoted the filling and expansion of the hygroma itself [30]. In view of the proximity of cyst middle wall to the temporal lobe, arachnoid breach could have connected the subdural space with the Sylvian cistern, with continuous hygroma replenishment [20]. SDGs are characterized by a fluid content that may be clear, sometimes blood-tinged or xanthochromic, due to the rupture of inflammatory and vascularized membranes (hematohygroma) [8]. Therefore, as cSDH, they can be distinguished into acute and chronic, but unlike the latter hygromas do not possess a true capsule and the inner fluid can have different chemical-physical profiles [30]. The patient owned an arachnoid cyst known for decades, likely congenital and such chronicity could result in qualitative and quantitative alteration of the fluid, both chemically and physically. Several studies in the literature state that the filling fluid would not be CSF itself but a CSF-like fluid with variable cellular-protein content of different molecular weights (from 9.3 kDa to 479.3 kDa) with both acidic and basic behavior [3,4]. Although cases of spontaneous cyst rupture have been described given the dynamics of the head injury with direct impact to the left temple, a first explanation is that the recoil (kickback or impulse) may have caused the cyst rupture or otherwise weakened the walls [16]. In the later stages, pressure vector forces to the opposite side still participated in the opening in the subdural space. Since cyst walls, especially older cysts, possess their own microvascularization abrupt rupture of the cyst results in the passage of blood into the CSF that fills the subdural space [8,27].

Conclusion

Rupture of a large arachnoid cyst in the subdural space may be mistaken for a post-traumatic hygroma if wall residues are not found, especially after localized head trauma. When the impact occurs in the opposite cranial region the true reasons for the hygroma constitution may be difficult to interpret. However, dynamics of head trauma inherent to "impact-impulse" and intracranial pressure gradients caused by the expansion of a contralateral subdural hematoma, may help to find an explanation regarding mechanisms that led to cyst rupture and CSF overflow into the subdural space.

Conflict of Interests

Author declares that there is no conflict of interest for this paper.

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