Neurological Sciences (2019) 40:2179–2181
https://doi.org/10.1007/s10072-019-03809-3

LETTER TO THE EDITOR

Widening the spectrum of secondary headache: intracranial hypotension following a non-aneurysmal subarachnoid hemorrhage

Valentina Oppo[1] · Giovanni Cossu[1] · Simona Secci[2] · Maurizio Melis[1]

Received: 7 November 2018 / Accepted: 26 February 2019 / Published online: 7 March 2019

Abstract

Background Intracranial hypotension has been associated with a wide spectrum of neurological conditions including chronic non-aneurysmal and acute aneurysmal subarachnoid hemorrhage.

Case A 59-year-old man presented with a non-aneurysmal subarachnoid hemorrhage in a perimesencephalic pattern after a mild physical exertion. In the course of the disease, a magnetic resonance imaging of head and spine displayed intracranial hypotension that resolved spontaneously.

Discussion Long-standing intracranial hypotension has been reported as the cause of chronic subarachnoid hemorrhage and a single case of intracranial hypotension as the consequence of intracranial pressure fluctuations after acute aneurysmal subarachnoid hemorrhage has been described.

This is the first description of intracranial hypotension caused by acute non-aneurysmal subarachnoid hemorrhage. We hypothesize that blood in the subarachnoid space could have determined a spine cerebrospinal fluid leak through intracranial pressure fluctuations or mechanical action, causing arachnoiditis and possibly a dural tear.

Keywords Subarachnoid hemorrhage · Perimesencephalic · Intracranial hypotension · Spinal epidural hematoma

Dear Editor,

Intracranial hypotension (IH), whether spontaneous or due to an eliciting, mostly traumatic, event, consists of orthostatic headache with specific radiological findings due to low cerebrospinal fluid (CSF) volume [1], caused by CSF leaks from the spine, mostly at thoracic or lower cervical level [1, 2]. A pre-existent dural weakness can directly lead to CSF leak or render the dura more vulnerable to trivial trauma or there may be a disposition due to connective tissue disorders [1].

IH is associated with a spectrum of neurological conditions that is constantly broadening [1], including chronic non-aneurysmal and acute aneurysmal subarachnoid hemorrhage (SAH) [3–6]. Complications of IH can be severe and potentially life-threatening [1]. We describe a case of IH occurring after non-aneurysmal SAH, aiming to explore the possible connections between these two clinical entities.

A 59-year-old man was referred to the emergency department for explosive, bilateral occipital, throbbing headache accompanied by nausea and vomiting occurring during physical exertion (amateur soccer play).

His medical history included active smoking, hypertension, and previous surgery for bilateral Dupuytren's contractures.

Head computed tomography (CT) scan performed urgently revealed a SAH in a perimesencephalic distribution (Fig. 1). Subsequently, CT angiography, magnetic resonance imaging (MRI), and digital subtraction angiography (the latter performed twice, at onset and after 3 weeks) did not reveal a vascular malformation. Therefore, a diagnosis was made of non-aneurysmal perimesencephalic SAH (PMSAH).

Seven days after admission, he complained of severe back pain and leg pain in a radicular distribution; therefore, a spine MRI was performed, revealing a subacute subarachnoid hematoma and arachnoiditis at lumbar level, and a dorsal thin epidural hematoma extended from D7 to D11 level (Fig. 2E).

At day 9, symptoms were unchanged (the patient still complained of throbbing occipital headache without postural features); therefore, a second MRI was performed: It showed

✉ Valentina Oppo
valentinaoppo@gmail.com

[1] Neurology Service and Stroke Unit, Department of Neuroscience, AO Brotzu, Cagliari, Italy

[2] Radiology Department, AO Brotzu, Cagliari, Italy

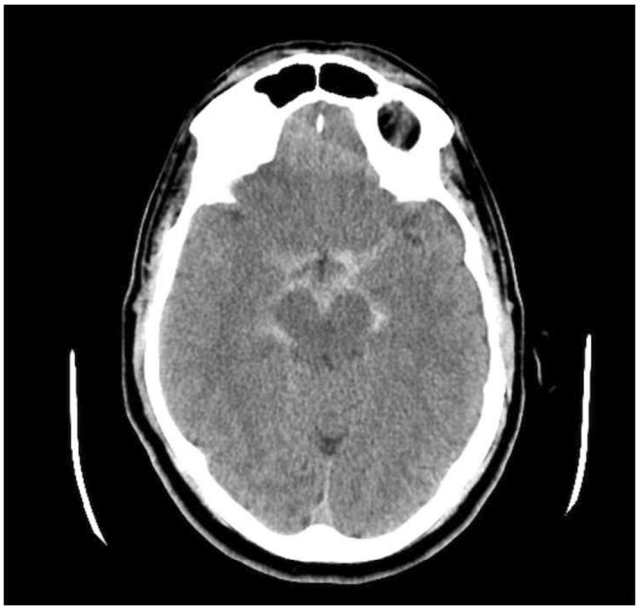

Fig. 1 Head CT scan performed at onset showing SAH in a perimesencephalic distribution

prominent bilateral subdural fluid collections (that had not been detected on MRI at onset, Fig. 2A), diffuse meningeal enhancement and flattening of the optic chiasm, consistent with IH (Fig. 2B, C, and D).

The patient was treated with oral nimodipine for prevention of cerebral vasospasm and intravenous saline solution for 3 weeks.

Headache and back pain resolved in 3 weeks, a brain MRI performed 23 days after the admission showed complete resolution of SAH and of the radiological picture of IH (Fig. 2F). The patient was discharged 25 days after admission, asymptomatic.

We present a case of IH occurring in the clinical course of PMSAH, which is defined as SAH with a characteristic distribution (the blood clot is located anterior to the pons or midbrain with or without extension around the brainstem, into the suprasellar cistern or into the proximal sylvian fissures), negative cerebral angiogram and a milder course [7].

Several cases of recurrent non-aneurysmal SAH in the posterior fossa leading to superficial siderosis [3, 4] and a few cases of acute diffuse non-aneurysmal SAH [5] have been reported in association with IH. The proposed pathogenic mechanism is recurrent bleeding from friable vessels at the site of dural tear [3] or occlusion or rupture of cerebellar bridging veins, stretched due to brain sagging [3, 5]. In all these reports, SAH was the presumed consequence of a long-standing condition of low-volume CSF stretching the vessel walls. On the contrary, our patient had no history of orthostatic headache or other clinical features of IH, and the radiological picture of IH appeared after PMSAH. Therefore we considered IH as the

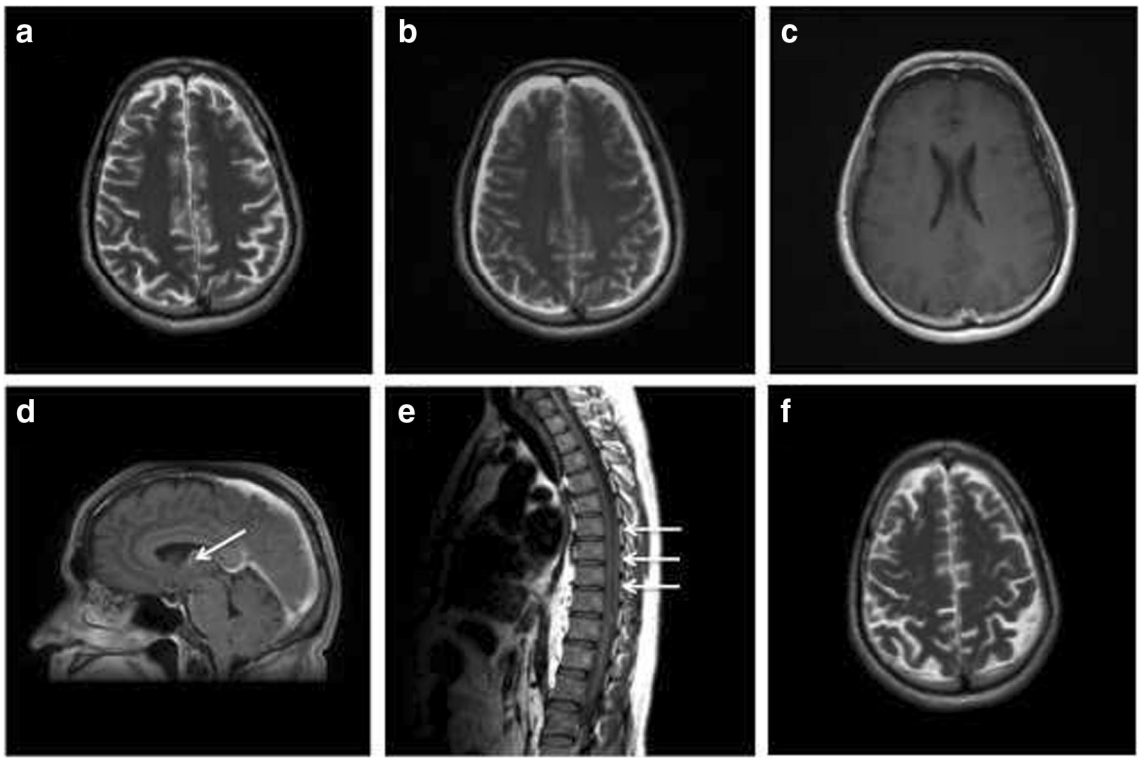

Fig. 2 T2 head MRI performed at day 1 (A); T2 (B) and enhanced T1 (C, D) head MRI performed 9 days after admission showing bilateral subdural fluid collections (B), diffuse meningeal enhancement (C), and flattened optic chiasm (D, arrow); spine MRI (E) revealing thin epidural hematoma extended from D7 to D11 levels (arrows); T2 head MRI performed at day 23 (F)

consequence of PMSAH. One case of IH complicating the postoperative course of an aneurysmal SAH is reported: it was hypothesized that intracranial pressure (ICP) fluctuations due to SAH caused the rupture of a perineural cyst and consequently the low CSF volume syndrome [6].

In our case, we hypothesize a similar pathogenic mechanism: the large subarachnoid blood amount could have determined a spine CSF leak through ICP fluctuations [6], or through mechanical action (causing arachnoiditis and possibly a dural tear). The presence of an epidural fluid collection at thoracic level is consistent with a CSF leak [1, 2]. The subacute subarachnoid hematoma and arachnoidits at lumbar level were instead interpreted in the setting of circulation of blood down in the spinal axis, which is a known phenomenon after aneurysmal SAH [8].

As an alternative explanation, PMSAH and IH could share the same triggering event or predisposing connective tissue disorder [1]. Future studies could better clarify further pathogenic link between SAH and IH.

To our knowledge, this is the first reported case of IH consequent to non-aneurysmal SAH, which widens the clinical spectrum of cerebrospinal fluid leakage syndromes. Physicians should be aware of this possible complication that may hamper the recovery after a PMSAH, especially in those patients with the largest extent of subarachnoid blood collection.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

References

1. Mokri B (2014) Spontaneous CSF leaks: low CSF volume syndromes. Neurol Clin 32(2):397–422
2. Schievink WI, Maya MM, Jean-Pierre S, Nuño M, Prasad RS, Moser FG (2016) A classification system of spontaneous spinal CSF leaks. Neurology 87(7):673–679
3. Schievink WI, Maya MM, Nuño M (2011) Chronic cerebellar hemorrhage in spontaneous intracranial hypotension: association with ventral spinal cerebrospinal fluid leaks. J Neurosurg Spine 15(4): 433–440
4. Webb AJS, Flossmann E, Armstromg RJ (2015) Superficial siderosis following spontaneous intracranial hypotension. Pract Neurol 15(5): 382–384
5. Schievink WI, Maya MM (2016) Diffuse non-aneurysmal SAH in spontaneous intracranial hypotension: sequela of ventral CSF leak? Cephalalgia 36(6):589–592
6. Sivakumar W, Ravindra VM, Cutler A, Couldwell T (2014) Intracranial hypotension in the setting of concurrent perineural cyst rupture and subarachnoid hemorrhage. J Clin Neurosci 21(6):1063–1065
7. Flaherty ML, Haverbusch M, Kissela B, Kleindorfer D, Schneider A, Sekar P, Moomaw CJ, Sauerbeck L, Broderick JP, Woo D (2005) Perimesencephalic subarachnoid hemorrhage: incidence, risk factors, and outcome. J Stroke Cerebrovasc Dis 14(6):267–271
8. Schievink WI (1997) Intracranial aneurysms. N Engl J Med 336(1): 28–40