

Hypothalamic dysfunctions as a late consequence of surgical opening of the lamina terminalis. A controversial hypothesis

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Abstract

BACKGROUND: Opening of the lamina terminalis is often used in surgery of the optico-chiasmatic region. Consequently, alteration of cerebral-spinal fluid (CSF) dynamics can occur after this manoeuvre, thus potentially translating into clinical complications. Herein, we describe 2 cases in which clinically relevant hypothalamic dysfunctions developed after few days opening of the lamina terminalis both patients showed mild to moderate preoperative hydrocephalus which improved postoperatively.

CASES DESCRIPTION: In a patient with ruptured aneurysm of the basilar bifurcation, opening of the lamina terminalis was performed prior to acute-stage clipping. On postoperative day 7th, the patient developed significant subdural hygroma, mild disturbances of consciousness and increase of ADH concentration. These clinical features resolved only following subdural hygroma drainage and ventricular-peritoneal shunting. One previously operated patient in whom the lamina terminalis had been opened to remove a sizeable parasellar tumour showed a similar post-operative course. In this patient, sole subdural hygroma drainage was not an effective treatment, and the patient died subsequently for complications related to long-standing, though mild, hypothalamic dysfunction.

CONCLUSIONS: Our experience may suggest that hypothalamic dysfunctions should be reminded as a possible, although rare, complication following the opening of the lamina terminalis. This clinical condition, if not properly managed, may contribute to trigger severe life-threatening complications.

Abbreviations:

ADH	- Antidiuretic Hormone
CSF	- Cerebral-Spinal Fluid
CT	- Computered Tomography
GH	- Growth Hormone
GSC	- Glasgow Scale
LH	- Luteinising Hormone
MRI	- Magnetic Resonance Imaging
PRL	- Prolactine
SAH	- Sub Arachnoid Heamorrhage
SIADH	- Sindrome of Inappropriate Antidiuretic Hormone
TSH	- Thyroid Stimulating Hormone
VP-shunting	- Ventricular Peritoneal Shunting

INTRODUCTION

Opening of the lamina terminalis is a widely used manoeuvre during surgery for lesion involving the anterior hypothalamic region (Colby *et al.* 2010; Komotar *et al.* 2009; Kim *et al.* 2006; Andaluz & Zuccarello 2004). Whilst it is as a rule mandatory in cases of tumors close to and/or invading the anterior wall of the third ventricle, such as giant pituitary adenomas and craniopharyngiomas, surgical opening of the lamina terminalis is an option in aneurysm surgery, which is however recommended by many experienced surgeons (Mortini *et al.* 2011; Colby *et al.* 2010; Kim *et al.* 2006; Spallone 2004; Samii & Tatagiba, 1997; Konovalov & Gorelyshev 1993; Yasargil 1990).

Recent papers have convincingly suggested that this simple surgical manoeuvre can be effective in preventing and/or at least reducing the risk of postoperative hydrocephalus following aneurysmal SAH (Komotar *et al.* 2009; Kim *et al.* 2006; Tomasello 1999; Yoshimoto 1998; Tetsuro *et al.* 1998). However no mention has been made of the possible negative consequences of this manoeuvre, which undoubtedly alters the intracranial cerebrospinal fluid (CSF) dynamics.

We have managed successfully a case of high-pressure unilateral subdural hygroma which we reasonably considered as a consequence of opening of the lamina

terminalis following acute-stage clipping of a top basilar aneurysm. A week after surgery, this case showed a moderate impairment of the anterior hypothalamus, which required proper management. A similar clinical event had been noted in the past in one case operated on by the senior author (AS), which, however, did not show the same successful clinical outcome, also due to the lack of proper consideration of the possible causative factors. These cases are reported hereunder in some detail.

CASE REPORTS

Case 1

A 64 year-old woman was admitted in May 2006 for a coma-producing SAH. This was likely to be a recurrent haemorrhage since the patient reported having suffered from severe neck pain lasting a couple of days, two weeks before. On admission she was grade IVa (Hunt-Hess' scale), and GCS 9. Emergency diagnostic work-up (CT, MRI, angio MRI and cerebral angiography) (Figure 1, A–C) showed a top-basilar aneurysm causing severe (Fisher grade 3) SAH and moderate hydrocephalus (Figure 2). Also, considering the presence of hydrocephalus as a possible causative factor for decreased consciousness, early surgery for aneurysm was scheduled and performed uneventfully 48 hours later, using a



Fig. 1. MRI angiography (A), CT angiography with volume rendering (B) and digital subtraction angiography (C) demonstrate an aneurysm of the basilar artery at the bifurcation.



Fig. 2. CT scanning shows diffuse SAH and moderate hydrocephalus, with early enlargement of the temporal horns.

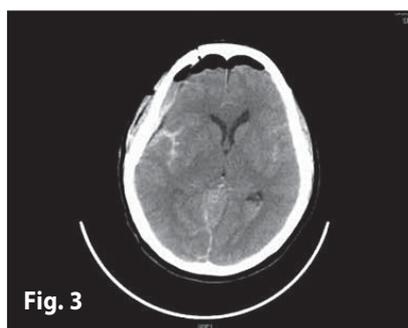


Fig. 3. Postoperative CT scanning shows resolution of the preoperative hydrocephalus.

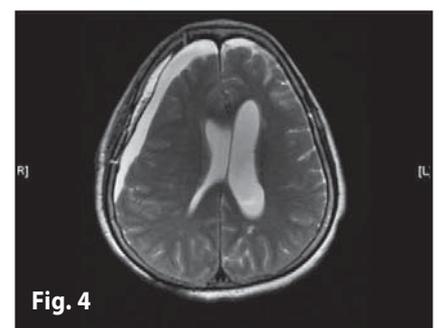


Fig. 4. Postoperative MRI gives evidence of a significant right subdural fluid collection.

right transcavernous approach. The lamina terminalis was opened for CSF release as well as for treatment of hydrocephalus. This helped in relaxing the brain and having access to the basilar bifurcation lateral to internal carotid artery (ICA), which had been carefully mobilized following release of the proximal dural ring. Aneurysm clipping was easily performed after proper dissection of the two large perforators.

In the next days there was a steady, progressive improvement in the neurological parameters, and post-operative CT scanning showed resolution of the hydrocephalus (Figure 3).

Patient was grade 3a (Hunt and Hess Scale) and GCS 13. On the 3rd post-operative day (5th post-SAH), a progressive left hemiparesis was detected, and Flair-MRI showed an area of hyperintensity in the right upper mid-brain. This was interpreted as a consequence of post-operative vasospasm and not from a perforator compromise, since no hemiparesis had appeared after clipping of the aneurysm. Accordingly, Triple-H therapy was applied, with some clinical response. Three days later, disturbance of consciousness appeared, accompanied by only slightly increased urinary output (2000 cc/day) and mildly decreased urinary concentration. Appropriate therapy was applied, without evident response. Control MRI demonstrated some resolution of the hyperintense lesion showed by the previous MRI (Figure 4), but also demonstrated the presence of a right subdural fluid collection of significant entity. This had also been found present in a previous post-operative examination, though in a less significant proportion.

It was hypothesized that the subdural CSF collection could play a major role in the clinical worsening. Being aware of unhappy experiences with previously treated patients (one of which will be reported as the following case), we elected to have the patient undergo a cardiac-gated MRI in order to evaluate the CSF circulation in the operated area. This examination (Figure 5) suggested increased CSF egress from the lamina terminalis towards the basal cistern of the right side.

We speculated that simple draining of the hygroma would not be sufficient to resolve the post-operative complication because it would not act on the causative factor itself. Moreover, we hypothesized that increased flow at the level of the surgical opening of the lamina terminalis, demonstrated by the MRI, could somehow be causally related to the clinical worsening. To this purpose, we monitored daily urinary and plasmatic Na⁺, osmolarity, as well as plasma levels of ADH, TSH, LH, GH and PRL in particular the ADH was assayed using the method described by Robertson et al (Robertson *et al.* 1973). There was a steady progressive increase in the ADH plasma concentration, which correlated well with decrease of consciousness. We considered that the apparent resolution of the hydrocephalus did not mean a real resolution of the disturbed CSF circulation, and decided to treat the patient with a VP shunting, together with draining of the subdural hygroma.

This was performed on June 3rd, 18 days following the SAH, 15 days following surgery. CSF opening pressure was increased (19 cm² H₂O).

VP shunting was deliberately inserted contralaterally in order to allow placing of a subdural external "closed system" drain for a few days after draining the subdural fluid collection. Immediately following surgery, there was a steady decrease up to normalization of the plasma level of ADH. Progressively there was a normalization of the plasma and urinary levels of Na⁺, which matched with a progressive general improvement of the consciousness, and a slow, but also progressive, improvement of the left motor deficit (Figure 6).

Post-operative cardiac-gated MRI (Figure 7) demonstrated normalization of the abnormalities of the CSF circulation showed by the pre-shunting examination. The patient was sent for rehabilitation programme and continued to show progressive clinical improvements. When last seen, 3 months post-operatively, she showed a mild (Grade 4) left hemiparesis, but was able to walk with a cane.

Control CT scan was normal, except for a small area of low density in the right upper mid brain (Figure 8).

Case 2

A 71-year-old man was admitted to our department in 2001 for management of a giant (4 × 5 cm) intra-suprasellar tumour extending along the clivus and causing mild-to-moderate hydrocephalus. Due to its predominant extension along the clivus, a subcranial approach, as described by Raveh et al (Raveh *et al.* 1993), was chosen for surgical removal of the mass which was expected to be a clival chordoma.

In fact, a soft tumor was rather easily removed with regular suction from both the intradural suprasellar, and the subdural extradural clival region, up into the lower third of the clivus. Opening of the lamina terminalis was mandatory in accomplishing macroscopical total removal of the mass.

Rather surprisingly, the tumor was found, histologically, to be a non-functioning pituitary adenoma.

On the first post-operative day the patient was conscious although mild decrease of the urinary concentration prompted correction with intranasal vasopressin in order to prevent diabetes insipidus. This was not needed later on.

The patient was neurologically and mentally intact after surgery and ambulatory on the fifth post-operative day. However, his mental brightness was observed to decrease on the 7th post-operative day, together with a decrease in serum Na⁺ level (from 140, to 136 early postoperatively, to 125 mEq/l). Serum electrolyte abnormalities were corrected, however, the clinical status did not improve as expected in the presence of normal urinary output.

A control CT scanning (Figure 9 A–B) showed a bilateral subdural hygroma, which was drained four days later, on the 15th postoperative day. Clinical



Fig. 5. A-B Cardiac-gated MRI clearly suggests increased CSF egress from the lamina terminalis on the right side. Whiter nuance indicates increased CSF flow. Note the definite difference in color between the right and the left paramedian regions. The available GE software did not allow quantitative CSF flow analysis

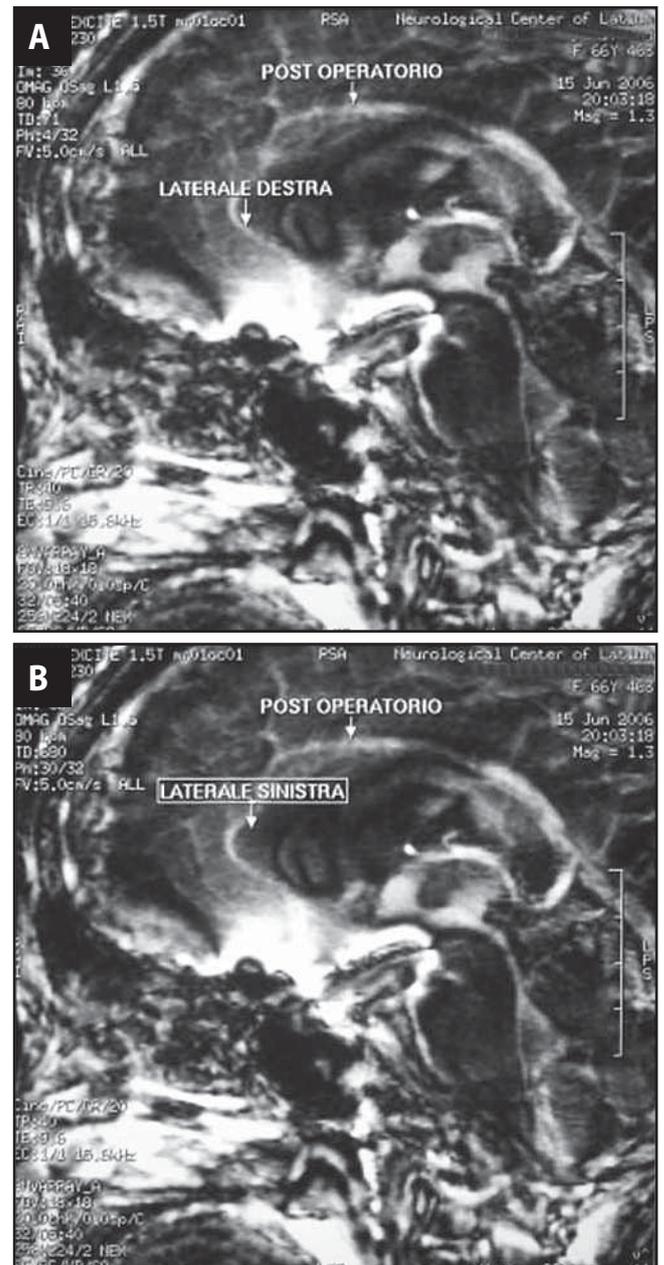


Fig. 7. A-B Control cardiac gated MRI indicates normalization of the CSF circulation at the level of the anterior based cisterns.

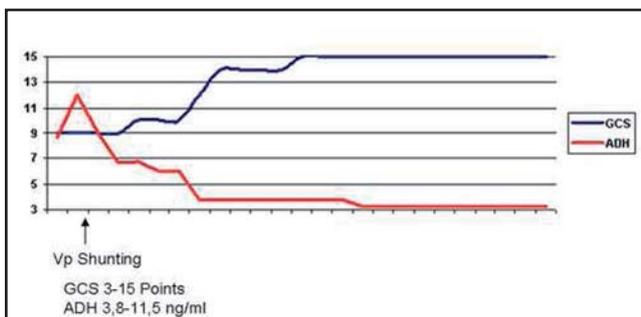


Fig. 6. Graphic representation of dynamic in the ADH plasma concentration correlated to GCS.

improvement was very mild if any, and control MRI scan (Figure 10) showed persistence and/ or reaccumulation of the hygroma. Eventually serum Na⁺ was observed to increase progressively up to 150 mEq/l, while only mild decrease in urinary output and only mild response to appropriate therapy. The level of consciousness never improved significantly; the GCS fluctuating between 10 and 11. Again, the electrolyte impairment was corrected without clinical improvement.

The patient died suddenly on the 23rd post-operative day for presumed pulmonary embolism. Permission for autopsy was denied.

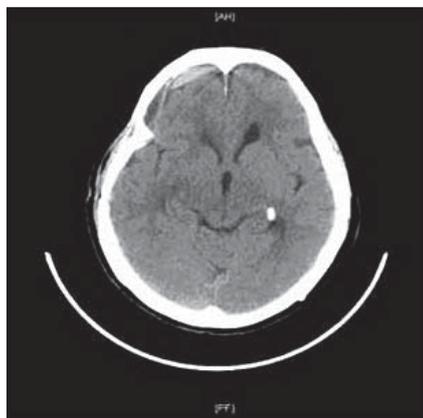


Fig. 8. Control CT scanning shows disappearance of the right subdural fluid collection and the hydrocephalus. An area of decreased density is evident in the right upper midbrain.

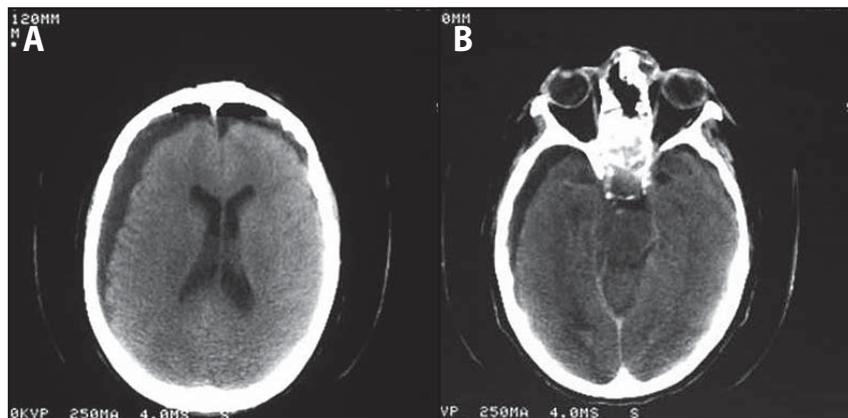


Fig. 9. Postoperative control CT scanning shows bilateral subdural fluid collection (A), and near-total tumor removal (B).

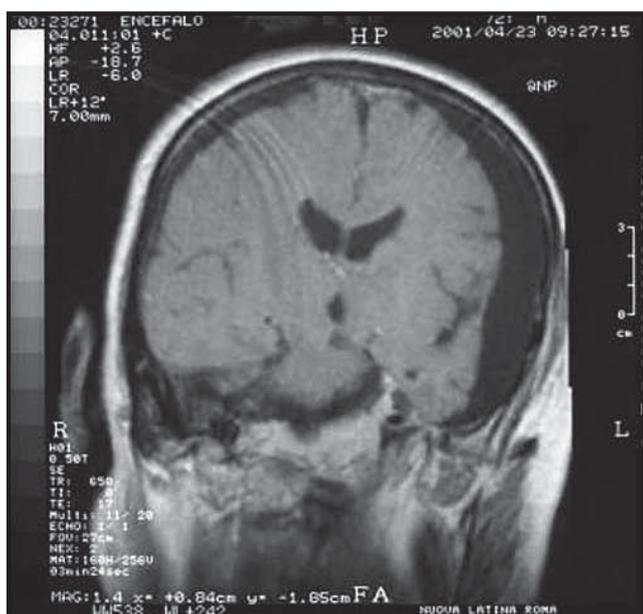


Fig. 10. Postoperative MRI gives evidence of reaccumulation of the subdural hygroma.

DISCUSSION

Surgical fenestration of lamina terminalis

Opening of the lamina terminalis is a technical manoeuvre which is often used in surgery for lesions of the optico-chiasmatic region. In cases of tumors affecting the anterior wall of the third ventricle, this manoeuvre is an obligatory step for proper and safe dissection close to the anterior hypothalamus. In aneurysm surgery, it helps to attain brain relaxation, although this can also be achieved satisfactorily with other technical manoeuvres, such as extensive opening of the basal cisterns as well as proper use of modern neuroanesthesiological techniques. Also, it may reduce the risk of postoperative shunt-requiring hydrocephalus (Komotar *et al.* 2009; Kim *et al.* 2006; Komotar *et al.* 2002; Tomasello *et al.* 1999; Yoshimoto *et al.* 1998; Tetsuro *et al.* 1998).

However, opening of the lamina terminalis certainly alters the intracranial dynamics of the CSF, although it should not globally affect the ICP. As a rule, this event does not have clinical relevance. However, in some clinical setting, as suggested by the present experience, such an alteration may lead to significant symptoms and complications requiring proper management. In other words, in the presence of certain facilitating factors, the alternative way for CSF egress from the ventricular system, created by the surgical fenestration of the lamina terminalis, can cause visible complications, such as subdural hygroma (Yoshimoto *et al.* 1998; Tetsuro *et al.* 1998), and at the same time it might perhaps play a role in the occurrence of more subtle, though not negligible complications, such as mild but persistent signs of hypothalamic dysfunction (Kreitschmann-Andermahr *et al.* 2004) which may in turn favour more serious, fatal ones in two of the present cases, complications as a result of a vicious circle.

Hypothalamic dysfunction

Clinical signs of hypothalamic functional impairment are not uncommon after surgery in the region of the lamina terminalis as well as following SAH (Yoshimoto *et al.* 1998; Tetsuro *et al.* 1998). They are usually considered to be the result of damage of hypothalamic nuclei due either to surgical trauma and/or to local vascular insufficiency. In this case the time course of clinical signs of hypothalamic dysfunctions was correlated closely with the development of the subdural hygroma. CSF-gated MRI demonstrated increased CSF egress from the third ventricle on the right side, where the postoperative subdural hygroma had developed. This situation normalized following VP-shunting as demonstrated by the control CSF-gated MRI a couple of weeks later. Not unexpectedly, the subdural hygroma showed a progressive though slow improvement, matching clinical and, mostly, neurohormonal improvement. As far as this latter is concerned, there was a strict temporal correlation between insertion of the VP shunt and

normalization of the blood concentration of ADH, a fact which would suggest a causal relationship between these two events. CSW syndrome is a well-known complication of SAH and consequent vasospasm occurring mainly in the presence of midline aneurysm. Several pathophysiological processes have been implied in its occurrence, including also a not well defined “natriuretic peptide”. This might also have been the case in the patients here reported. Case 1 showed the typical time course of clinically relevant post SAH vasospasm. However the clinical course suggested two different peaks of symptoms: the one of hemiparesis reaching its most relevant clinical expression within the first week after SAH, and then showing some improvement; that of disturbances of consciousness and hypothalamic dysfunction, which reached its peak remarkably later and strictly correlated with increased ADH blood concentration suggested rather a SIADH. Case 2, studied much less thoughtfully, showed a time pattern of progressive hypothalamic dysfunction, (initially CSW syndrome and later on possibly SIADH related unrelated to the surgical trauma but closely correlated with the development of post operative subdural collection).

In this case surgery for subdural hygroma was not effective, and the mild impairment of the hypothalamic functions and of the consciousness never resolved completely, a fact that certainly played a role in the unfavourable outcome of the patient.

Pathophysiological hypothesis

The present cases were operated on for different pathologies with different surgical approaches. However they showed, as a common feature, the presence of preoperative hydrocephalus as well as the opening of the lamina terminalis performed at surgery. We speculate that the presence of preoperatively altered intraventricular CSF pressure, perhaps combined with some postoperative impairment of other physiological mechanisms of CSF reabsorption – such as subarachnoid blood and/or debris due to aneurysmal SAH and/or previous intracranial surgery – might have represented those “facilitating factors” which we mentioned above. The present cases scenario has created the premises for the intraventricular CSF to be shifted under increased pressure towards the subdural space, causing a difficult-to-resolve subdural hygroma. At the same time, hypothalamic disturbances, also reported by other studies with aneurysmal SAH in which the lamina terminalis was opened (Yoshimoto *et al.* 1998; Tetsuro *et al.* 1998) might also have been caused by the following factors:

- a. increased flow at the level of the anterior part of the third ventricle, affecting the functions of the neighbouring region
- b. marked increase in intraventricular pressure, in the presence of a “normal” ventricular system.
- c. a combination of both

It must be taken into account that the blood brain barrier is either underrepresented or practically absent at the level of supraoptic and paraventricular areas in the anterior hypothalamus (Dolenc 2003; Aroussi *et al.* 2000), a fact which renders these specific areas vulnerable in certain pathological conditions. A good example of this is the occurrence of significant neurohormonal abnormalities after brain trauma, which could persist as late as one year or more after the traumatic event (Jovanovic *et al.* 2010; Kreitschmann-Andermahr *et al.* 2004).

We suggest that the presence of modifications of the local chemico-physical characteristics of anterior hypothalamic environment, as it could happen in the presence of blood, due to SAH and/or previous intracranial surgery, may trigger a process of local cytotoxicity, which would render these cells particularly vulnerable to any change of the local environment including perhaps changes of the local hydrodynamics (Yoshimoto *et al.* 1998; Tetsuro *et al.* 1998). This process would potentially affect the cell membrane and interact with their selective ion permeability, eventually causing hypothalamic insufficiency. Perhaps this suggested pathophysiological process might represent, or somehow interact with, that “natriuretic factor” (peptide), that was suggested in the past to be the causative factor of CSW syndrome

Hyponatremia is a manifestation of a variety of disorders and a side-effect of diuretics (Glover & Clayton 2012; Rastogi *et al.* 2012; Tarantini *et al.* 2007; Kurtumus *et al.* 2006). Alternatively, it may be only manifestation of an underlying misdiagnosed disorder (Kurtumus *et al.* 2006; Sata *et al.* 2006) but also could act as worsening element in this process.

Out of the present cases, we studied thoroughly only case 1. Previous experience with similar cases has represented the reason for proposing, and consequently evaluating, a pathophysiological hypothesis. As stated above, we speculated that resolution of hydrocephalus in case 1, demonstrated by postoperative CT scan, did not necessarily mean resolution of the disturbances of intraventricular CSF physiology (Raimondi & Tomita 1982), and decided accordingly to implement also a ventricular peritoneal shunting. To this purpose, we want to stress that the intraventricular pressure was well above the range (190 mm Hg) as measured during the VP-shunting, in spite of the apparent resolution of hydrocephalus following surgery. Also, impaired CSF dynamics at the level of the anterior basal cisterns normalized following ventricular extrathecal CSF diversion, and subdural hygroma –though slowly– resolved. Most relevant, as stated above, resolution of the neurohormonal abnormalities was diagnosed on the basis of both clinical criteria and, most important, by monitoring of the normalization of the ADH plasma concentration, promptly followed VP shunting, a fact which in our opinion gives support to our admittedly controversial hypothesis. In fact, the other case which we managed

previously and who did not undergo a VP shunting never showed a resolution of their, mild-to moderate, signs of hypothalamic dysfunction. We are aware that other explanations might exist for the occurrence of the hypothalamic dysfunction in the present cases, such as for example delayed vasospasm. However we want to stress the close correlation between VP shunt insertion, normalization of ADH plasmatic levels, normalization of CSF dynamics and clinical improvement, which would indicate that altered CSF hydrodynamics could play, in fact, a causative rule.

CONCLUSION

Opening of the lamina terminalis is not a consequence-free procedure, since it alters significantly the physiological CSF dynamics. The present experience would perhaps suggest that the intracranial altered CSF physiology created by the fenestration of the lamina terminalis might, although occasionally, negatively interact with the anterior hypothalamic functions (of the supra-optic and paraventricular areas in particular). In such cases we strongly suggest placing a VP shunt for CSF diversion, maybe in addition to the surgical drainage of the subdural fluid.

We hope that reporting our experience with the present cases will focus the attention towards the possible occurrence of these complications which are likely to be extremely rare, but not unlikely to be underrepresented and/or misdiagnosed.

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