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Herpetic Meningoencephalitis Occurring After Decompressive Craniectomy in Severe Head Trauma

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ABSTRACT

Viral meningoencephalitis in general and herpes in particular is an extremely rare complication after neurosurgery. We report the case of a 26-year-old patient, admitted for management of a severe head injury with refractory intracranial hypertension who underwent decompressive craniectomy which was complicated by herpetic meningoencephalitis treated with aciclovir for 21 days with a successful outcome at 12 months.

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KEYWORDS:
Head trauma, Herpes simplex virus, Meningitis, Neurosurgery.

INTRODUCTION

Herpes simplex virus (*HSV*) meningoencephalitis after neurosurgical procedures is rare. It has been associated with the resection of central nervous system (CNS) tumors [1]. The diagnosis is difficult, responsible for a therapeutic delay with a high morbidity and mortality rate [2]. Rapid diagnosis and management are essential while ruling out other infections [3]. The film array is a rapid test with a sensitivity of 95% and a specificity of more than 99% for *HSV* identification in cerebrospinal fluid (CSF) and is therefore accepted worldwide as a standard diagnostic tool [4].

CASE PRESENTATION

A 26-year-old patient without any notable pathological history was admitted to the emergency room of provincial hospital for management of a severe trauma following a fall from the third floor causing loss of consciousness. The initial examination found an unconscious patient with a GCS of 7/15 (E 1, V1, M5), who was hemodynamically and respiratory stable. The patient was intubated, ventilated and sedated on neurological criteria and then transferred to our university hospital for specialized and multidisciplinary management.

On admission to the emergency department of our university hospital, the patient was intubated, ventilated and sedated, pupils in tight miosis, with a blood pressure of 150/75 mmHg and a heart rate of 85 bpm, saturated correctly under protective mechanical ventilation in volume-controlled mode with a FiO2 of 50%. A body scan was performed (figure 1), showing, on the craniofacial level, left temporal intraparenchymal hematoma, right temporal extradural hematoma, bilateral parenchymal contusions, left parietal sulcal meningeal hemorrhage, and diffuse cerebral edema, associated, on the thoracic level, with foci of alveolar hemorrhage and bilateral basal inhalation. Biology revealed: hemoglobin at 12.3 g/dl, hyperleukocytosis at 12180 elements/mm3, CRP at 140 mg/l, platelets at 209,000 elements / mm3, PT at 100%, normal renal and hepatic functions, and a slightly disturbed rhabdomyolysis. After monitoring (arterial, central venous and urinary catheters, repeated transcranial doppler (TCD) and gasometries), the patient was put under deep sedation, osmotherapy, noradrenaline with MAP target and ACSOS prevention. In front of the refractory intracranial hypertension to neuro-resuscitation measures (pathological transcranial doppler and aggravation of diffuse cerebral edema at the control brain CT), The patient was transported to the operating room where he underwent a left fronto-parieto-temporal craniectomy with hematoma evacuation, duraplasty and flap feeding, then the patient was transferred to the neuro-resuscitation department for further management.

On day 7, the patient developed an infectious problem with a febrile plateau at 39°C and a worsening of the infectious workup with white blood cells (WBC) at 17,000 elements / mm3 and a CRP at 242 mg/l. A complete infectious workup

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(blood cultures, cytobacteriological urine study, protected distal sampling and lumbar puncture) was performed with a purulent CSF leading to the initiation of Vancomycin and Meropeneme at meningeal dose. Lumbar puncture (LP) came back positive with WBCs at 170/mm³ with lymphocytic predominance (82%), hyperproteinorachy at 1.33 g/l (0.15-0.45), normoglycorachy at 1.3 g/l with concomitant glycemia at 1.08 g/l. All other samples were negative. On day 9, in the absence of clinical and biological improvement (WBC at 25600 elements/mm3, CRP at 292 mg/l and a control LP showing WBC at 1220/mm³ and proteinorachy at 1. 6 g/l), the negative culture of the initial LP and a brain scan eliminating an empyema or an intracranial collection, a multiplex PCR was carried out coming back in favour of *HSV1* meningitis, hence the patient was put on antiviral drugs (aciclovir) with a good clinical (apyrexia) and biological (WBC at 16,700 elements/mm3 and CRP at 116 mg/l) evolution.

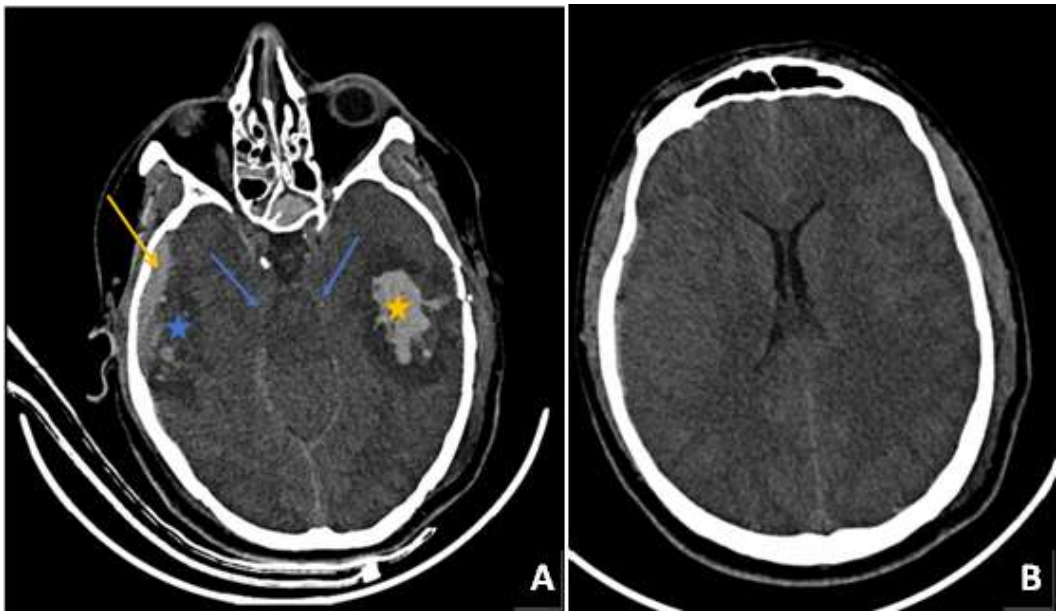

Figure 1. A) - Left temporal intra-parenchymal hematoma (yellow star), heterogeneous containing air bubbles within, measuring 40 x 18 x 31 mm in diameter (APxTxH), surrounded by a hypodense collar related to peri-lesion edema,

- Right temporal extradural hematoma (yellow arrow) and Temporal oedemato-hemorrhagic Focal Points of Contusion (blue star) bilaterally,
- Central Involvement (blue arrows),

B) Effacement of the cortical sulci without SB-SG dedifferentiation, with collapsed lateral ventricles related to the onset of diffuse cerebral edema.

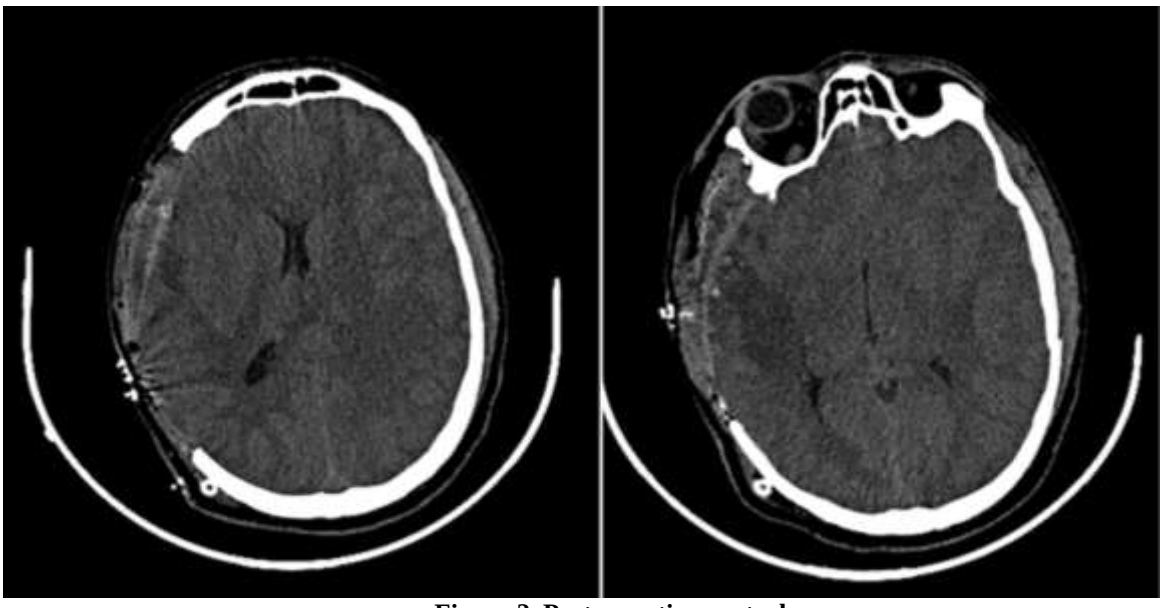

Figure 2. Postoperative control

Table 1: Clinico-biological evolution through ICU stay.

		J0 Admission	**J7**	**J9**	**J15**	**J21** ICU discharge
Heart rate (bpm)		85	140	138	90	75
Respiratory rate (cpm)						
Temperature (°C)		37.2	**39**	**40**	37.5	36.8
Hemoglobin (g/dl)		12.3	10.8	10.3	10.6	10.1
WBC (e/mm³)		12180	**17000**	**25600**	16700	12260
Platelets (e/mm³)		209000	280000	342000	398000	425000
PT (%)		100	98	100	97	95
Urea (g/l)		0.3	0.25	0.23	0.28	0.38
Creatinine (mg/l)		7	8	7	6	7
CRP (mg/l)		140	**242**	**292**	116	58
Bicarbonate (mmol/l)		20	15	16	22	21
Lumbar puncture	WBC/mm³	-	**170** lymphocytic (82%)	**1220**	34	-
	Protéinorachie g/l	-	**1.33**	1.6	0.9	-
	Glycorachy (g/l)	-	**1.3**	-	-	-

On day 15, a LP was performed, showing WBCs at 34/mm³ and a protein count of 0.9 g/l. A control brain scan showed regression of the initial post-traumatic injuries. The duration was 10 days for antibiotic therapy and three weeks for aciclovir. The patient had received mechanical (upon admission) and pharmacological thromboprophylaxis (started 48 hours after craniectomy with a satisfactory control CT scan), as well as nutritional support throughout his ICU stay. He had received a weaning tracheotomy with sedation cessation and respiratory and motor physiotherapy. He woke up with a good neurological recovery (GCS 15, good cough reflex and no sensory-motor deficit). After a positive leakage test and bolus of corticosteroid therapy, the patient was decanted on day 20 and transferred 24 hours later to neurosurgery department. The 12-month follow-up found a conscious patient with a GCS of 15/15 without sensory-motor deficit.

DISCUSSION

Life-threatening infections that occur after neurosurgery are usually of bacterial origin and include bacterial meningitis, brain abscesses, and subdural empyema [3]. Immediate postoperative viral encephalitis is extremely rare [5], and *HSV* is the most common cause of viral and fatal non-epidemic encephalitis.

The *HSV* encephalitis is considered to involve the reactivation of a latent infection localized in the cranial nerve ganglia at the level of the sensory ganglia, in particular the trigeminal ganglion [6], or to result from invasion of the CNS through the olfactory tract [7]. The question that arises is whether reactivation of the viral agent occurs in response to stress (surgical/post-traumatic) or following surgical manipulation of brain tissue or following severe head trauma as in our case. Factors that may promote reactivation of viral infection have been proposed, including corticosteroid therapy, chemotherapy, trauma, immunosuppressed state, high viral load, low total dose of acyclovir (especially in children under 2 years of age), short-term treatment, bacterial infections (including meningitis and *Pneumococcus* infection), exposure to ultraviolet radiation, and menstruation [8]. Two types of reactivation are distinguished (inflammatory and necrotic) with different clinical manifestations. In inflammatory reactivation, the clinical presentation involves a leukoencephalopathy without convulsions, which is progressive and characterized by the presence of choreoathetosis and behavioral disorders [9]. In contrast, in necrotic reactivation, the clinical features are comparable to those of the first clinical symptoms of meningoencephalitis (convulsions and/or status epilepticus, fever and disturbed consciousness) [10].

There are some interesting features of herpetic encephalitis in post-neurosurgery that emerge from a review of cases reported in the literature. The average time reported for the onset of encephalitis after surgery was about 6 days [11] and 7 days in our case. The main symptoms described were fever and altered consciousness [11]. But the signs and symptoms are not specific, which makes diagnosis difficult and management late, which is why mortality can reach 70% in the absence of treatment and 30% in those who are properly treated. However, the morbidity in those who survive varies between 30 and 39% [4].

In neurosurgery, when a postoperative infection is suspected, LP is mandatory, and subsequent analysis and culture of the CSF is paramount to screen for CNS pathogens [2]. Because of the diversity of these pathogens responsible for meningitis and/or encephalitis, multiplex PCR techniques allowing to simultaneously search for different bacterial, viral, parasitic or mycotic pathogens are techniques of the future despite their high cost for the moment. For example, the Film Array Meningitis/Encephalitis Panel kit (BioFire Diagnostics) allows the simultaneous detection of 14 pathogens responsible for the majority of meningitis and encephalitis (Table 1). These very rapid techniques (about 1 hour) are well adapted to emergency situations [12]. Due to its sensitivity of 95% and specificity of more than 99% for the identification of HSV in CSF [2], multiplex PCR has been shown to be the most sensitive for the diagnosis of herpes necrotizing encephalitis and can avoid brain biopsy [13]. In contrast, a brain biopsy is required to confirm the diagnosis of herpetic inflammatory encephalopathy in which PCR results are usually normal [11].

An elevated level of interferon α (produced by lymphocytes as part of the humoral immune response within the CNS, and its production precedes the antiviral antibody response) in the CSF is another useful diagnostic marker [11].

Magnetic resonance imaging (MRI) is sensitive in these cases, but it is not specific. In cases of necrotic reactivation, there is an increase in radiographic findings, including evidence of high density in the temporal region. However, the findings may be normal in the early phase of infection and may not even be readable in the postoperative setting. Diffuse MRI changes away from the surgical site are strong indicators of encephalitis [11].

Treatment consists of the earliest possible administration of high-dose aciclovir at a recommended dosage of 10 mg kg-1 every 8 hours for 21 days. A shorter duration of treatment (10 days) seems to be associated with a significant relapse rate. Some authors suggest that a control LP should be performed between days 10 and 14 and that aciclovir should be continued until day 21 only if the PCR is positive. A retrospective, non-randomized study of 45 patients evaluating corticosteroid therapy in combination with aciclovir suggests a beneficial effect of corticosteroids; however, this has not yet been validated by controlled trials. Relapses under well-conducted treatment have been described in adults, but are rare [14].

CONCLUSION

After neurosurgery, viral encephalitis is a rare but serious complication that should be suspected in cases of unexplained postoperative fever with consciousness disorders, especially in patients with a history of encephalitis. Bacterial origin should be excluded by CSF analysis and multiplex PCR should be performed to confirm or affirm the diagnosis of viral encephalitis. The potentially serious complications should guide the physicians involved in the management of these patients.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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