

Multidisciplinary management and outcome in subdural empyema - a case report

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Rezumat

Managementul multidisciplinar și consecințele în empiemul subdural – prezentare de caz

Empiemul subdural cerebral (ESC) reprezintă o colecție intracranială septică ce apare în principal ca o complicație a sinuzitei, otitei sau mastoiditei. Deși este destul de rar, ESC necesită o atenție deosebită în vederea diagnosticării precoce, imagistica cerebrală fiind obligatorie în fiecare caz suspiciat. Orice întârziere în derularea tratamentului duce către comă și exitus. Autorii prezintă un caz despre un băiat, care a dezvoltat sepsis sever, edem cerebral important, colecție fină de lichid subdural cu minimă deplasare a liniei mediane. Ulterior IRM-ul cerebral a evidențiat creșterea colecției subdurale cu mărirea deplasării liniei mediane ce a necesitat intervenție chirurgicală de urgență. Colecția s-a dovedit a fi empiem. Alte constatări ale IRM cerebral au fost pansinuzita, mastoidita și tromboflebita de sinus transvers. Recuperarea a fost însoțită de antibioterapie de lungă durată, tratamentul epilepsiei secundare și kinetoterapie pentru deficitul neurologic. Pacientul a dezvoltat de asemenea și chiste cerebrale care au expansionat creierul prin breșa osoasă postoperatorie și care au necesitat

intervenție chirurgicală atât pentru îmbunătățirea deficitului neurologic cât și pentru aspectul estetic. Gestionarea acestui caz a necesitat o colaborare strânsă între infecționist, neurolog, neurochirurg, microbiologist și radiolog.

Cuvinte cheie: infecții ORL, supurație intracraniană, urgență chirurgicală, chist intracerebral, cranioplastie, abord multidisciplinar

Abstract

Cranial subdural empyema (SDE), a localized septic intracranial collection, occurs mostly as a complication of sinusitis, otitis or mastoiditis. Although relatively rare, SDE requires an increased attention for early recognition, cerebral imagery being mandatory in any suspected case. Any delay in treatment can lead to coma with a fatal outcome. The authors report the case of a young boy, who developed a severe, important cerebral edema, thin subdural collection with minimal displacement of the median line. Repeated cerebral MRI showed an enlarged subdural collection with higher median line shift and imposed rapid surgical intervention. The collection proved to be empyema. Other findings on MRI are pansinusitis, mild mastoiditis and transverse sinus thrombophlebitis. The recovery was accompanied by the need for long course of antibiotherapy, secondary epilepsy treatment and kinetotherapy for hemiparesis. The patient also developed intracerebral cysts expanding the brain through the postoperative cranial defect which later needed surgical intervention, for both neurological and esthetic improvement. The management of

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the case implied strong interdisciplinary collaboration between infectionist, neurologist, neurosurgeon, microbiologist and imagist.

Key words: ENT infections, intracranial suppuration, surgical emergency, cerebral cysts, cranioplasty, multidisciplinary approach

Introduction

Subdural empyema (SDE) is an intracranial collection of purulent material situated between the dura mater and the arachnoid mater. SDE accounts for a medium of 20% of all cases of intracranial abscesses (ranging from 10% to 40% according to different studies) (1, 2, 3, 4). SDE can lead to cerebral edema, hydrocephalus (8), cerebral infarction from thrombosis of the cortical veins or cavernous sinuses or from septic venous thrombosis of contiguous veins in the area of subdural empyema (9).

The predilection for young ages (10-40 years old) is explained by the higher incidence of ear and rhino-sinuses acute infections during this period of life (6, 10). The infection disseminates from paranasal sinuses and less frequently from the middle ear or mastoid cells, spread via cerebral thrombophlebitis or rarely by direct erosion of a sinus wall or mastoid bone (6, 7, 11).

SDE treatment is primordially surgical and constitutes a major emergency because of the high risk of death by mass effect. The surgery protocol is complex and implies the complete discharge of the pus and the entirely removal of the purulent membranes developed on brain and meninges surfaces (6).

The introduction of antibiotics, the discovery of computed tomography (CT) and later magnetic resonance imaging (MRI), the mainstays of the imaging diagnosis of SDE (14, 15) have decreased the mortality rate (5) but now patients survive with neurological deficits.(10).

Case report

A 15 years old boy, was addressed by emergency department to infectious diseases clinic as acute meningo-encephalitis reporting suffering in the last 3 days from headache, cervical pain, drowsiness, nausea, vomiting and fever and also a minor head injury, 3 days old, apparently because of dizziness. Native cranial CT scan revealed a hypodense fluid collection over the right hemisphere of 5,2 mm, important cerebral edema with midline shift of 4,2 mm (Fig. 1) and also pansinusitis. Considering the general status of the patient and the CT characteristics, the neurosurgeon decided not to operate at this stage. Neurological exam: Glasgow Coma Scale of 10, left hemiparesis, including facial paresis, neck stiffness and tonic-clonic contractions of the right leg. Lab findings showed leukocytosis with neutrophilia, thrombocytopenia ($56000/\text{mm}^3$)



Figure 1. Right hemisphere hypodense fluid collection with minimal midline shift

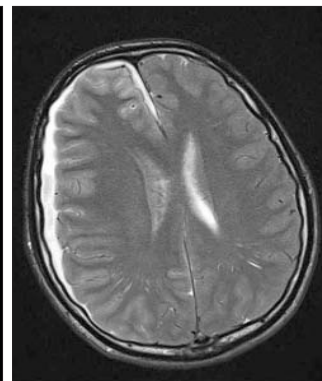


Figure 2. T2 weighted image revealing a high signal fluid collection midline shift

prolonged prothrombin time and inflammatory syndrome (CRP 162 mg/L).

He received cerebral edema treatment and Meropenem + Vancomycin for the severe sepsis. In the next hours, the patient became anisocoric and a gadolinium-enhanced MRI revealed an increase in right fronto-temporo-parietal and interhemispheric fluid collection, with low signal in T1 and a high signal in T2 weighted images (Fig. 2), triggering a 9 mm midline shift. The patient was rapidly transferred to the Neurosurgery Clinic.

Emergency surgery was performed with a wide decompressive frontal-temporal-parietal craniectomy and the surgical drainage of the SDE. Intraoperative the right hemisphere was covered by a thick layer of pus, white brain adherent membrane and small amounts of gas underlining bacterial presence (Fig. 3). After complete drainage of the pus and the removal of the white adherent membrane, the brain presented intense hyperemia (Fig. 4). About 60 ml of pus was removed, and the lab isolated three anaerobic bacteria

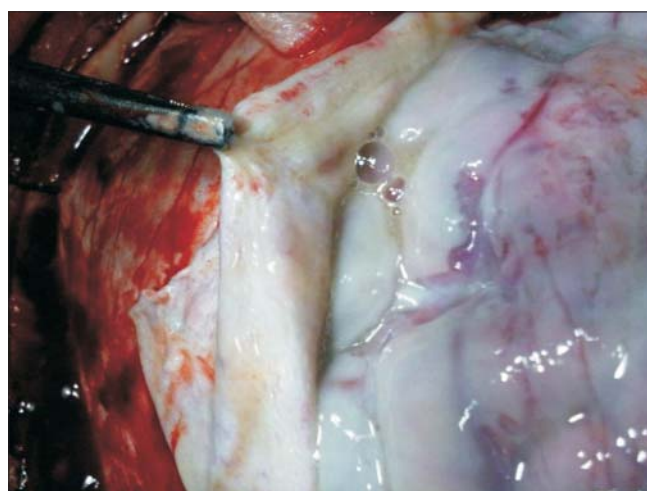


Figure 3. Pus covering the right hemisphere and gas visible emerging from subdural empyema



Figure 4. Hyperemic brain surface after removal of SDE

(*Fusobacterium varium*, *Fusobacterium mortiferum*, *Propionibacterium propionicum*).

He received treatment with Meropenem, Linezolid, Metronidazole and Chloramphenicol, for one week, then Linezolid and Meropenem till 3 months along with anticoagulants and anticonvulsants. The cerebral contrast enhanced MRI, 3 weeks postoperative showed right sigmoid and transverse sinus thrombosis, inflammatory changes in right mastoidian cells, pansinusitis, the swelling of the brain through the craniotomy with the presence of liquid filled cysts and a minimal residual fluid collection over the right hemisphere and interhemispheric.

One month after surgery he was transferred to the Infectious Disease Clinic for treatment and recovery as no further neurosurgical treatment was necessary at that moment.

The motor deficit was partially recovered. He continued to have episodic tonico-clonic seizures with onset in the left leg, some with secondary generalization which were controlled with anticonvulsant therapy. He was discharged 2 months later, continuing the anticonvulsant therapy.

The 6 months and 1 year cerebral CT scan revealed enlargement of the cerebral cysts (Fig. 5). The patient began accusing persistent head aches and transitory nausea.

1 and a half years after the first surgery, cranioplasty was performed, using a custom made PEEK implant. The intracerebral cysts were drained along with the puncture of the lateral ventricle (through the floor of one of the cysts) in order to reduce the brain enough to fit the implant. Postoperative CT scan showed complete cysts drainage (Fig. 6).

The patient had a very good postoperative evolution, with neurological improvement of the left facial hemiparesis, minor left hemiparesis improvement, no more headaches or nausea and also a good esthetic improvement (Fig. 7).

Discussions

The diagnosis of meningo-encephalitis (recent fever and neck stiffness as meningitis signs and hemiparesis, seizures,

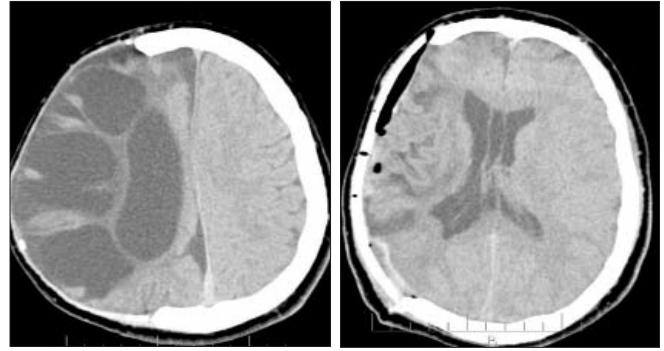


Figure 5. Intracerebral cysts, 1 year after SDE surgery

Figure 6. Post cranioplasty surgery cerebral CT scan



Figure 7. Preoperative brain swelling and postoperative outcome

altered mental status as encephalitis signs) with important cerebral edema, sustained the medical treatment – broad-spectrum antibiotherapy with a good penetration in the CSF and cerebral substance and depletive treatment – Mannitol and Dexamethasone.

The encephalitis' signs imposed an imagistic exam for searching localized collections with surgical disposal.

After the first imagistic exam (native CT scan), the crucial question was about the cause of the midline displacement, because of the high risk of encephalic herniation, which requires fast intervention – surgical decompression. In the opinion of the first neurosurgeon and the 2 imagists who initially examined the patient, respectively the CT scan, the very thin subdural effusion didn't explain the midline shift. On the other hand, the cerebral edema, even if important, was "uniform" and could not explain the asymmetry. Could it be an intraparenchymal mass undetectable at CT scan? The answer was given by the second imagistic exam, consisting in MRI with contrast medium, which excluded focal brain signs and showed the increasing subdural collection, pointing this as cause for cerebral structures displacement.

The second arising problem was if the effusion was a hematoma or an empyema? A minimal decompression can be sufficient for a hematoma, but in the case of an empyema, the

surgery should be more complex and difficult. A subdural hematoma (eventual infected in a second time) after falling, was ruled out, because the clinical exam didn't show any signs of cranial trauma and the history of the illness started before he fell. Without contrast medium, a subdural hematoma cannot be differentiated from an empyema, on CT scan (16). The distinction on MRI it is also debatable (16). The clinical course as a severe sepsis and the proximal pansinusitis favored the presumption that there was a purulent collection.

What was the starting point of the empyema in that case? Probable the acute pansinusitis, even the impairments were especially on the left side. The propagation of the infection could take place by contiguity but also via venous intracranial system (the patient had intracranial right thrombophlebitis). In the literature, the sinusitis are the most common cause of the subdural empyema overall and the frontal location particularly indicate the sinuses (6, 7). An otomastoidian origin could be poorly sustained by the MRI findings (minor changes on right mastoidian cells) and the right temporal-parietal location of the empyema (6, 7). A dental origin could be suspected because of the association of the 3 anaerobes which have an oral habitat (6).

The surgical intervention became imperative, at the onset of anisocoria, despite the severe thrombocytopenia. Even though imaging can accurately localize the fluid collection allowing it to be evacuated by burr hole placement, the surgical team opted for a wide right frontal-temporal-parietal craniectomy because it improves the outcome in SDE by allowing wide exposure, adequate exploration, and better evacuation of subdural and interhemispheric purulent material (17, 18).

The bone flap from the F-T-P craniectomy showed signs of contact with the purulent collection and it was decided to discard it.

Despite of the large and thoroughly intervention, the next MRI showed 2 inter-hemispheric residual collections that necessitated a long antibiotic treatment.

SDE is known to cause venous sinus thrombosis with cerebral infarction (5, 19). Our patient was diagnosed 3 weeks after surgery with right sigmoid and transverse sinus thrombosis. On the same CT scan intracerebral cysts were developing. The question here is: did the venous sinus thrombosis have a part in the formation of such cysts?

The complexity of the case required multidisciplinary collaboration between infectionist, neurologist, neurosurgeon, microbiologist and imagist.

Conclusions

Broad-spectrum antibiotic treatment should be administered from the moment of diagnosis in order to achieve the best possible outcome.

Emergency surgery must be performed with no delay, along with medical treatment is not effective. Wide craniotomy should be the surgical method of choice to ensure the full drainage of the purulent fluid and to obtain a good decompression.

In the presence of venous sinus thrombosis, the possibility of brain cysts formation should be taken into consideration. Cranioplasty using custom made implants or preserved bone flaps must be attempted as soon as possible to avoid traumatic brain injury and to prevent cerebral cysts from evolving, in case they exist, which could lead to further neurological deficits.

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