To coagulate or not to coagulate. That is the question.

COAGULAR O NO COAGULAR. ESA ES LA CUESTIÓN.

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DECISION-MAKING IS A COMPLEX ISSUE ESPECIALLY IN SEVERE MULTI-PATHOLOGICAL PATIENTS, WHOSE PROCESSES INTERACT WITH EACH OTHER AND WITH OTHER TREATMENTS, MAKING IT IMPOSSIBLE TO FOLLOW REGULAR PROTOCOLS. IN THIS CASE, THE PATIENT SUFFERED FROM A CRANIOENCEPHALIC TRAUMATISM AND A PULMONARY THROMBOEMBOLISM. IN THE CASE OF PULMONARY THROMBOEMBOLISM, AN ANTICOAGULATION PROPHYLAXIS IS RECOMMENDED WHILE IN THE FORMER IT IS CONTRAINDICATED. MOREOVER, COMPLICATIONS MAY APPEAR, APPARENTLY RELATED OR NOT TO MAIN DISEASES. IN THESE SITUATIONS, IT IS ESSENTIAL TO ESTABLISH PRIORITIES AND HAVE A VAST KNOWLEDGE OF PATIENTS' PATHOLOGIES AND THE POSSIBLE COMPLICATIONS THAT MAY EMERGE. FIRSTLY, OUR AIM WAS THE RESOLUTION OF THE BRAIN TRAUMA DUE TO THE MORTAL RISK OF BRAIN HERNIATION. STRAIGHTAWAY WE RELIEVED THE SEVERE SUBSEQUENT COMPLICATIONS OF THE MAIN PROCESSES, NAMELY, REFRACTORY INTRACRANIAL HYPERTENSION, ACUTE RESPIRATORY FAILURE AND THROMBOCYTOPENIA. ONCE THE PATIENT WAS STABLE, PULMONARY THROMBOEMBOLISM TREATMENT COULD BE SET AND FOLLOWED AFTER CLINICAL IMPROVEMENT AND DISCHARGE.

KEYWORDS: PE, TBI, MANAGEMENT OF THE POLYPATHOLOGICAL PATIENT.

PALABRAS CLAVE: TEP, TCE, ABORDAJE DEL PACIENTE PLURIPATOLÓGICO.

Introduction

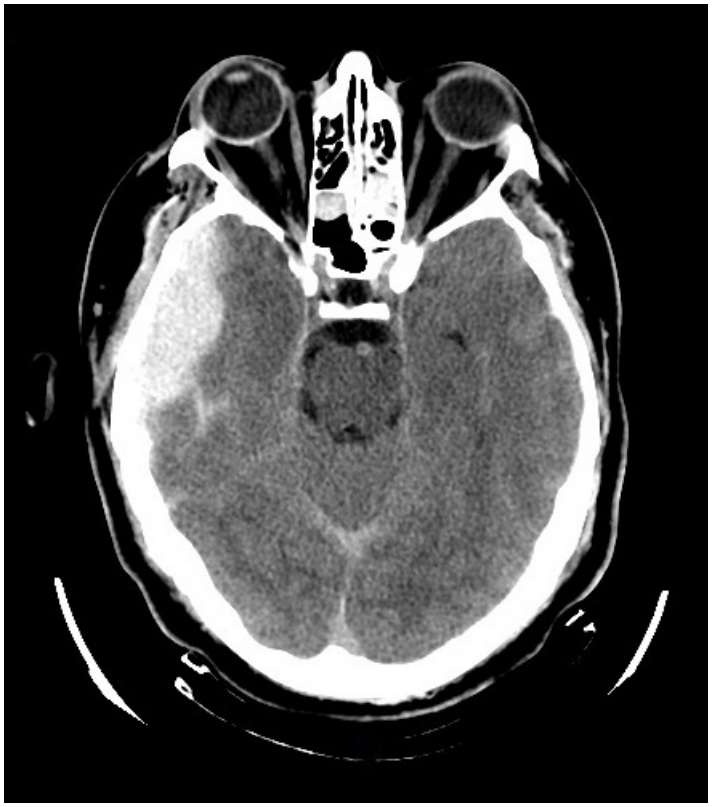
Due to the super specialization of medicine, its holistic conception from a physiopathological point of view is hard to achieve. With this clinical case, we offer an example of the decision-making in a multi-pathological patient and the complexity it may involve, like for example, those admitted in an Intensive Care Unit (ICU). The strategy followed in this Department would be to establish a hierarchical list of problems according to their potential mortal risk, to then provide them with a treatment. All of this taking into account the possible complications and interactions between treatments and concomitant processes. Thus, we combine both support and etiological treatments. Thereby, if the patient's physiological reserve allows it and his or her processes evolve favorably, support measures are reduced until his or her full recovery.

In this case, one of the main pathologies that will be introduced is pulmonary thromboembolism (PTE). This is a life-threatening syndrome produced by vascular obstruction in the pulmonary vasculature. Frequently, it is a consequence of deep venous thrombosis (DVT). As a matter of fact, the thrombus formation factors are the same in both cases, namely hypercoagulability, endothelial injury and alterations in blood flow (1). Its non-specific clinical presentation may vary between dyspnea, cough, pleuritic or substernal chest pain and tachypnea, tachycardia, hypotension, fever or cyanosis; depending essentially on the embolus size and the previous cardio-respiratory status of the patient (1). Moreover, it could involve cardiogenic shock or syncope (with possible associated falls) and their consequences. These falls could imply other symptoms whose significance may depend on the location and the intensity of the traumatism. In case of severe brain trauma with epidural or subdural hemorrhage, it might be associated with oedema and mass effect. Since the overall volume of the cranial vault cannot change, an increase in the volume of one of its components, or the presence of pathologic ones, necessitates the displacement of other structures, an increase in intracranial pressure –intracranial hypertension (IH)– or both. One of the most hazardous displacements is brain herniation which might involve loss of consciousness, pupillary dilation and further neurologic deficits, or even coma and death (2).

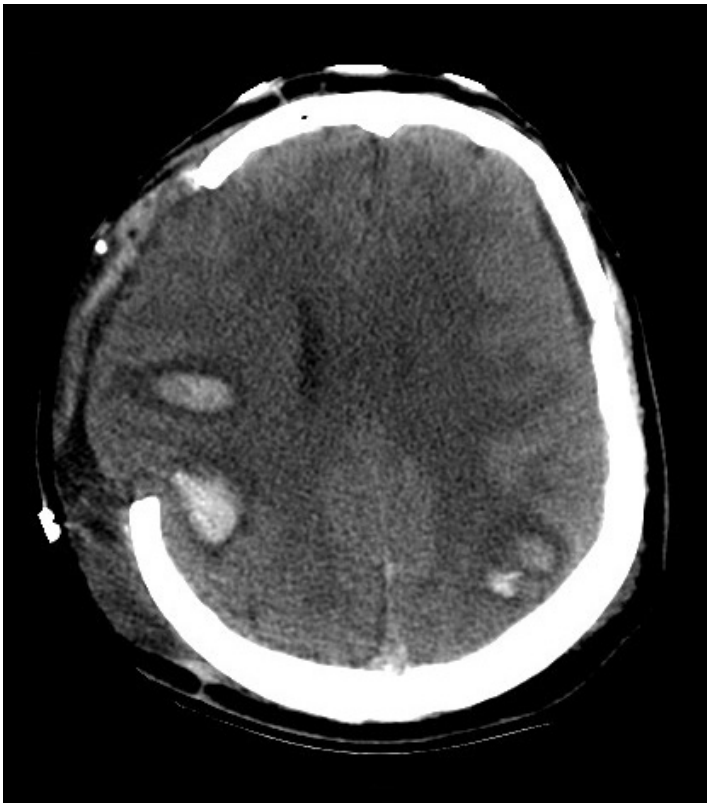
Case report

A 42-year-old male is admitted in ICU due to a fainting episode and consequent fall, which caused him a brain trauma, followed by epileptic seizures, vomits –with probable lung aspiration– and coma.

The medical record reveals that he is obese and that he smokes. His family states that the patient presented anxiety, oral and upper limbs paresthesias 2 hours before the accident. Moreover, he experienced weakness and dyspnea



▲ **Figure 1**. *Head CT scan. Right epidural hematoma in temporal lobe.*



▲ **Figure 2**. *Head CT scan. Right decompressive hemicraniectomy and bilateral acute intraparenchymal hemorrhages.*

in the previous 2 days. His Glasgow Coma Score is 4 and he has isochoric reactive pupils. Etiological differential diagnose of syncope includes neurological, cardiac and vascular diseases. Interview, examination and basic complementary test (an abnormal simple thoracic radiology and an ordinary electrocardiogram) show the necessity of a CT scan test. Cranial Computed Tomography (CT) shows a right epidural hematoma shifting the mid-line, whereas computed tomography pulmonary angiography (CTPA) shows a thrombosis of some of the branches of the left and right inferior lobar arteries.

After setting sedoanalgesic, antibiotic, antiepileptic and stomach protection treatment, we try to improve respiratory function with orotracheal intubation and invasive mechanical ventilation adding a greater oxygen inspiratory fraction and positive end-expiratory pressure (PEEP). Furthermore, a decompressive craniectomy is performed meanwhile the hematoma is evacuated. Nevertheless, the patient suffers intraparenchymal hemorrhages, so we carry out a second contralateral craniectomy in order to control IH.

Referring to the respiratory condition, a bronchoaspiration pneumonia and bilateral atelectasis produce a severe hypoxemia that forces us to optimize the antibiotic and respiratory measures. Despite the treatment, the patient contracts a multidrug resistant *Acinetobacter baumannii* (MDRA) pulmonary infection so we set him in prone ventilation and change the empiric antibiotic therapy to a directed one

The study of the pulmonary thromboembolism (PTE) is not conclusive; besides, the ECO-doppler rules out existence of deep vein thrombosis, thus implanting an inferior vena cava filter is dismissed. Days after, a control TC reveals triangular alveolar opaque areas (Hampton's hump) and bullaes, suggesting pulmonary infarction. This deterioration caused by prolonged mechanical ventilation entails a percutaneous tracheostomy. Just before providing heparin, we notice a severe drug-induced thrombocytopenia. It improves after removing all the suspicious drugs and adding corticosteroids.

After increasing the number of platelets, the PTE is treated with fondaparinux and the patient progressively gets better and is discharged to the Physical Medicine and Rehabilitation Department.

Discussion

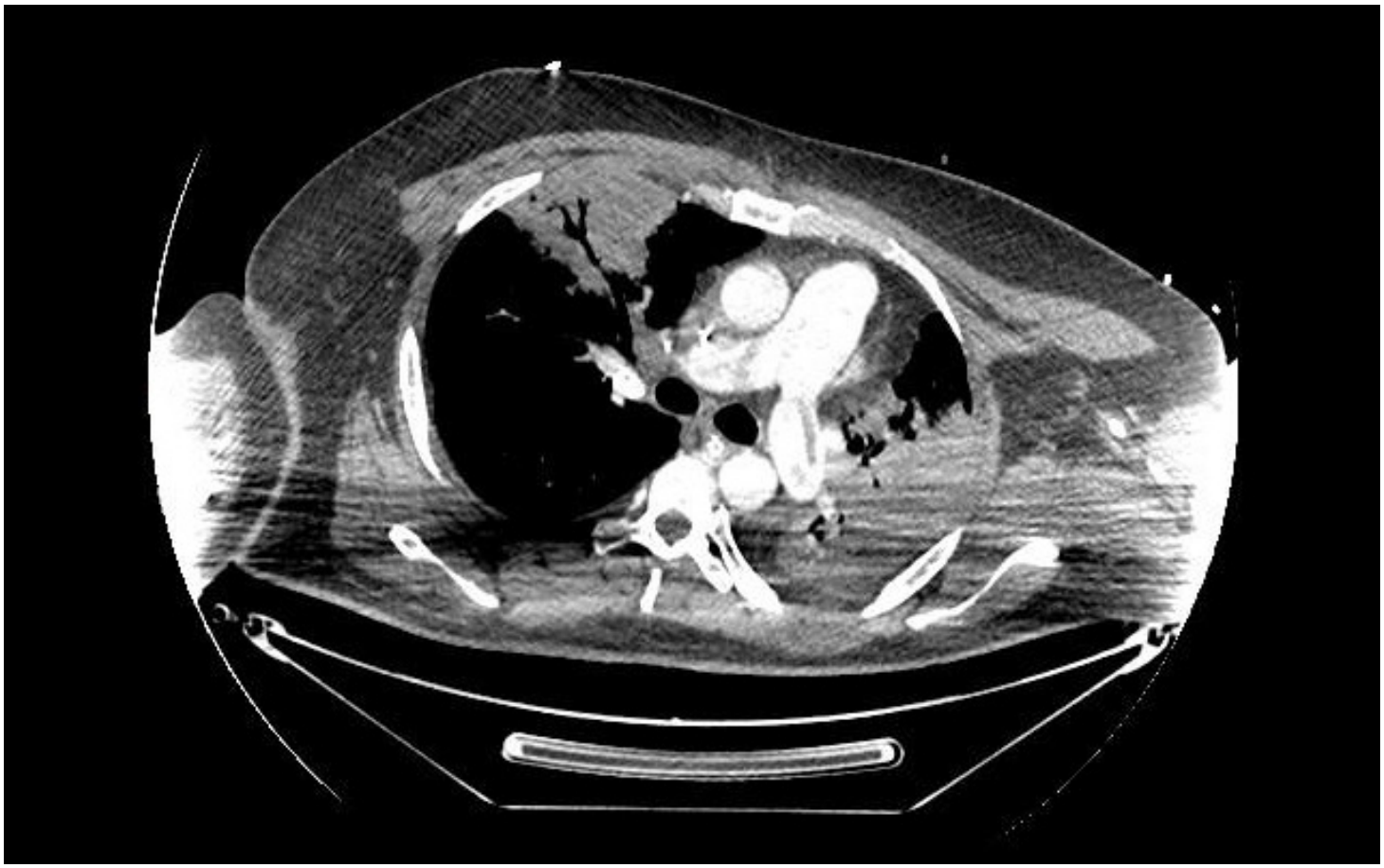
This case poses a controversy in medical decisions especially because of the interaction of each specific treatment with these complex concomitant diseases. The torpid progress and the narrow therapeutic range make this case worthy of discussion.

Effective management of severe brain trauma involves the evacuation of the underlying epidural or subdural haematoma but also the avoidance of IH (3). IH is a severe condition that can lead to patients' death by herniation. Medical treatment of increased intracranial pressure should include sedation, permissive hyperventilation, hypothermia, drainage of cerebrospinal fluid if necessary, and osmotherapy with either mannitol or hypertonic saline (3). Decompressive craniectomy might seem an aggressive measure, but it is the elective choice when IH becomes resistant to medical treatment. Although complications are extremely rare, this procedure might develop some potentially lethal ones, such as the emergence of new brain hemorrhages (4). Secondary brain hemorrhages to decompressive craniectomy are thought to be caused by rapid brain shift that can produce remote bleeding due to the shear stress to the brain vessels (4).

Regarding the PTE, the diagnostic strategy starts assessing the clinical probability of PTE and is followed by either D-dimer testing or CTPA (5). Our patient scored from 9 to 11 points in Padua Prediction Score for Risk of venous thromboembolism, according to which anticoagulation prophylaxis is indicated if the risk measured is over 3 points. The results in critically ill patients who have a high risk for DVT and PTE, demonstrate they should receive pharmacologic prophylaxis unless contraindicated (6). However, any anticoagulation, either with heparin –unfractionated heparin (UFH) or low molecular weight heparin (LMWH)– warfarin, fibrinolytic agents or thrombin inhibitors may all lead to intraparenchymal hemorrhages as a complication (7), hence their employment should be avoided not only because of the underlying IH, but also because of the tissue damage on account of mass effect.

As our priority is to preserve brain integrity, we have to avert progression of PTE using alternative measures to heparin, such as pneumatic intermittent compression. Furthermore, due to the severe refractory hypoxemia of our patient, we have to improve his respiratory function using different measures as antibiotic prophylaxis, bronchial secretions aspirations, lung protective ventilation strategy using small tidal volume with PEEP, alveolar recruitment maneuvers, postural changes and prone ventilation. As the patient is obese, prone ventilation improves atelectasis in dependent zones of the lung and in combination with lung recruitment method increases SpO2 (8).

Compounding to patient's severe respiratory and neurological condition is the gradual developing of



▲ **Figure 3**. *Pulmonary CT scan. Hampton's hump and left atelectasis.*

thrombocytopenia. After discharge different causes as inflammatory illness o congenital disorders, the most common diagnose is drug induced thrombocytopenia (9). This could be caused by several drugs that were part of patient's treatment as levetiracetam (anticonvulsant), tigecycline (antimicrobial used to treat MDRA) and IV metamizol. The frequency of drug-induced thrombocytopenia in acutely ill patients has been reported to be approximately 19–25% (9). Generally, platelet count falls within 7 or more days after starting a new drug. When the drug is stopped, the platelet count rises rapidly within 1–10 days of withdrawal (9). After this episode of thrombocytopenia, TEP treatment with fondaparinux is more suitable than heparin due to heparin ability to caused immune-mediated thrombocytopenia.

In concurrence with the pulmonary infection, the patient has many risk factors that make him susceptible of being infected with MDRA, such as mechanical ventilation, stay in an ICU for a considerable time period, a severe illness, recent surgery and many invasive procedures as well as multiple intravenous lines, monitoring devices, surgical drain, and indwelling urinary catheters (10). Typically, treatment of *A. baumannii* infection includes aminoglycosides (amikacin) in combination with a beta-lactamase-stable beta-lactam (piperacillin) often along with beta-lactamase inhibitor (tazobactam) or imipenem (10). In case of MDRA, which is resistant to at least all penicillins and cephalosporins, fluroquinolones and aminoglycosides; carbapenems are the treatment of choice (10). Nevertheless, it is suggested that an antibiotic therapy should always be guided by *in vitro* susceptibility profile of the organism, due to the multiple and rapid mechanism of resistance (10). In this case, we set the suggested treatment by the 2014 clinical practice guidelines, but we directly change empirical treatment with meropenem, linezolid and levofloxacin to those that the antibiogram indicated as effective (tigecycline and colistin).

The successful outcome supports the effectiveness of the medical decisions for future similar situations. It is important to consider our established priority, in order to deal with all the processes that the patient suffers, and also to have an ample knowledge of these so as to be able to foresee the possible complications and anticipate to them.

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