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Spinal hematoma is an unusual complication in central regional anesthesia (1). In the literature it is called in most cases an event associated with various disorders: hematologic, systematic diseases or anticoagulant therapy. Regardless of the underlying pathology, the effects of this complication can become dramatic, ranging from lower back pain to neurological deficit to varying degrees. We present the case of a patient who has suffered post-suffering spinal subdural hematoma without predisposing risk factors. Presentation of the clinical case

Patent 79 years with a history of HTA in the treatment of ARA II and amlodipine, harmful anaemia in the replacement treatment of vitamin B12 and primary prevention of SAS 300 mg. which was suspended 5 days before the intervention. In 2001, she underwent surgery to replace her right knee under intradural anesthesia with bupivacaine isobara without incident. As for pre-operative analysis, the parameters were within the norm. Enter the subcapital fracture of the left hip to accommodate partial prostheses performing subarachnoid anesthesia with a single paramedic puncture, which turned out to be hematic, but which is refined and injected 10 mg. buivacaine isobara, unaccompanied by paresthesia to accommodate partial prostheses. No other intra-operational incidents. High urPA full motor and sensory recovery. 36 hours after the operation begins with lumbar pain located in the right renal pit. Twelve hours later, she begins with constipation, valued by the Internal Medicine Service, which diagnosed her with a paralytic ileus box, installing treatment with a rectal tube, nasogastric and serum therapy. Within a few hours, he was again appreciated for citing the inability to move his legs, as well as reduced sensitivity. Sensitive paraparesis 0/5 objected to the abolition of osteotendinos reflexes (ROT) and sensory level in D10. Before the clinic, a bolus 1500 mg of methylprednisolone and subsequent infusion was prescribed, thinking about the possibility of an epidural hematoma, taking into account the previous regional anesthesia. The image testing was requested by dismembering the MRI, performed by the Radiology Service because of the planted prosthesis, and decided to perform the lumbar spine of the spine CT, which showed hypermenedness, fusiform morphology and slightly lateral image on the right in the anterior epidural space in relation to the acute hematoma, which extends from D12-L5 with a 1.3 centimeter sharpness with a noticeable reduction of the spinal cord. At all times, coagulation times and other parameters of hemostasis were this conclusion is decided by neurosurgery surgically unpacking hematomas. In the surgical act it was the opposite that the hematoma was subdural predominantly of the previous place, which did not allow extraction by the aspiration, and also objected to the tense and cyanotic dura mater, as well as the edematized medullary cone. The solid hematoma content was partially removed, and the graft was placed in the dura mater incision area for the impossibility of the seam by edema. She joined the UCI for post-operative control, having been discharged without noticeable incidents in the 48 hours remaining of paraparesia. During his time on the floor he continued his paralytic picture of ileus. In the face of the situation, no improvement was received in the specialized unit of wounded medical nurses for intensive rehabilitation. Discussion

The regional spinal anesthesia is considered a safe method, but not without complications, some life-threatening. Spinal hematoma, although rare, is an adverse event of lumbar puncture and is a cause for concern, as it can lead to permanent neurological deficit. Its incidence is not reliably established, in the revised literature most often collected extradural hematoma, followed by subdural and subaracoid (2). In a prospective study in France assessing the incidence and characteristics of serious complications associated with regional anesthesia, about 100,000 regional anesthetics were collected within 5 months, resulting in trauma after puncture and neurotoxicity of anesthesia being the most common cause of neurological complications. However, this study showed that the frequency of severe complications with this type of anesthesia was very low. Cheney et al analyzed the records of the pen's claims to determine the role of nerve damage in regional anesthesia. Of the 4,183 revised claims, 670 (16%) were not claims. refer to neuro-neurological injuries associated with anesthesia. The frequency of hemorrhagic complications is unknown, but very rare. Horlocker in the citation review refers to the fact that the incidence in the literature should be less than 1/150,000 epidural anesthesia and less than 1/220,000 intradural (3). Vandermeulen et al. (3) and Tyagi et al. (4) indicate in their series of patients (81 cases in total) that the most common cause of bleeding, defined as subdural hematoma, is due to a large percentage (between 68% and 54%). to blood clotting disorders. These blood clotting disorders were secondary to some malignant hematological processes (leukemia, haemophilia, thrombocytopenia, cryoglobulinemia, hemorrhagic diathesis, police, etc.). The second most common cause of hematoma is iatrogenic At least 33%; that was due to the difficulties faced in the foolish puncture or return of CSF. Epidural anesthesia, especially in catheterization, is more traumatic than BSA, and is therefore more prone to bruising. In our environment there is a 2002 review that records a total of 16 cases of bleeding after regional anesthesia, with 5 cases of subdural hematoma. A type of regional anesthesia will also curb the incidence of neurological complications. Thus, 70% of Vandermeulen and Tyagi series patients are associated with epidural anesthesia, and 23% are associated with intradural anesthesia. In Castillo with 18 patients 8 cases (40%) epidural anesthesia, 9 (45%) intradural anesthesia and 1 cerebrosal epidural combination. The neurological clinic usually begins in the first 24-48 hours with lumbar pain followed by engine deficits in the lower extremities and sensitivity disorders. In our case, the imaging test (reverse-lumbar CT) indicated the epidural location of the hematoma. Domenicucci et al in its review include that image test of selecting MRI for early diagnosis and determining the exact location of the hematoma to be confirmed by the surgical examination. As excessively avened factors for the production of spinal hematoma, we have hbpm introduced for thromboprophylaxis that do not increase the risk if safety intervals are observed from the time the drug is scheduled for the technique performed. Patients receiving oral anticoagulation should discontinue it 3-5 days before the technique and perform analytical monitoring to test INR on the day of the intervention. On the other hand, anti-thrombocyte remedies such as dienopyridins should be discontinued at least 7-10 days before (5). The usual recommendation for sympathetic hematoma is the implementation of urgent decompressive laminectomy, surgical decompression 8 hours after the onset of symptoms is associated with the worst prognosis, in addition to various corticoid models added to the treatment. While intervention is indicated in severe and progressive cases, it may be less serious and slow-paced and sender cases allow for a wait-and-see attitude under strict clinical and radiological control. Castillo et al. note that cases of hematoma with cerebrosal compression secondary neuroaxial anesthesia, found in Spain, numerically important in many cases after subacnoid anesthesia, often associated with known and preventable risk factors.0Bibliography1.- Castle J, School F, Gallart L, Montes A, Samse E, Castagno J, Santeuri X. Safety as a result of proper preoperative assessment and training. Rev. Espe Anesthetiol Reanim 56(1): 16-20. (PubMed) No, no, no. 2.- Domenicucci M, Ramieri A, Paolini S, Russo N, Occhiogrosso G, Di Biasi C, Delfini R. Spinal subarachnoid bruising: our experience and literature review. Neurosurgical law vol.147 issue July 7, 2005. P.741-50. (PubMed)3.- Vandermeulen E, Van Aken H, Vermelen J. Anticoagulants and spinal epidural anesthesia. Anest Analg 1994; 79(6):1165-77. (PubMed) No, no, no. 4.- Pulls A., Bhattacharya A. Central Neuroc blocks and anticoagulation: an overview of current trends. Eur J Anesthesia 2002: 19: 317-29. (PubMed) No, no, no. 5.- Horloker TT, McGregor DG, Matsushige DK, Schroeder DR, Besse JA. Retrospective review of 4,767 consecutive spinal anesthesia: complications of the central nervous system. Perioperative results of the group. Analg, Anest. 1997 Sea; 84(3):578-84. (PubMed) No, no, no. Intracranial subdural hematoma (US), as a complication of epidural anesthesia procedures, is very rare, although there are cases described in the medical literature. After puncturing dura mater in these methods there is a risk of GS, which may be due to cerebrosal fluid hypotension syndrome (CSF). The symptoms of this table are associated with the massive effect and shift of structures, depending on the age of the patient, his location, size, speed of institution, preconditions and compression of intracranial structures. Differential diagnosis between CSF hypotension syndrome and HS from intracranial hypertension can become difficult, avoiding early diagnosis. We present the case of a 27-year-old postpartum who developed GS secondary epidural anesthesia procedures given to her during childbirth. 27-year-old woman, without any prior interest. Go to the emergency room of our hospital in 2 consecutive cases, 4 days after the uncomplicated eutoxic delivery, in which epidural anesthesia was injected with levupevavainan by puncturing the needle Weiss type 18 in the intervertebral space L2-L3, front-occipital headache clinic, which appears and aggravates in the two-legged and improves with decubital. You have blood pressure (PA) 148/73mmHg and heart rate (FC) 70 bpm. Physical examination has not revealed any pathological data, and there is no marfanoid, leptosomatic habit, no hyperlacia of joints or skin. There is no neurological examination of the hearth. The patient is prescribed with painkillers and anti-inflammatory drugs. In the following weeks he presents a postural headache that does not make it impossible for him to carry out the activities of everyday life, and that gives or improves with the pain treatment prescribed. About a month after the month, to our service is an intense headache that has lost its postural character, which does not improve with conventional painkillers and that is accompanied by vomiting and a restrained state of anxiety and arousal. PA 136/86mmHg and FC 37 lpm. Neurological examination did not reveal pathological findings. Hemogram, biochemistry, coagulation, venous and systematic urine gasometry without findings. In ECG, sinus bradycardia stands out, without other conclusions. Normal chest X-ray. Extensive left-wing fronto-tepro-parietal HS is observed in cerebral CT, with a significant effect of the mass displacement of the middle line and the ventricle system 14 mm to the right (Figure 1). Discussing the case with the neurosurgical service of the reference hospital, the question of its transfer is decided. While in intensive care awaiting transfer, he suffers from a sharp deterioration in consciousness, with Glasgow 3 and anisocoria, so a decision was made to sow, relax, orioterache indulgence, mechanical ventilation and the introduction of drugs against brain swelling. Upon arrival at the reference hospital, the bruises are urgently evacuated with the implementation of two tripeno holes, one anterior parietal and one left posterior parietal, causing a high-pressure hematoma. With a serogematic debit of abundant drainage and Glasgow 15, it has a good evolution, brain control CT performed, where the re-separation of the brain parenchyma and the recovery of the middle line (Figure 1) is objective and finally discharged to monitor your attending physician and neurologist area, pending consideration in external consultations of neurosurgery. HS is defined as the collection of blood that accumulates in the space between dura mater and arachnoid at the cranial level, with post-traumatic etiology being the most common. The first descriptions of chronic SS were made by Vepfer in 1658 and Morgani in 1761, while Virkhov in 1857 considered non-traumatic etiology in the so-called internal hemorrhagic groin-imeningitis1.2 Trotter1-3, in 1914, theorized with the possibility that its origin was in the rupture of small thin veins located in the arachnoids. HS as a complication of epidural anesthesia methods is a rare complication, with a prevalence between 1/500,000 and 1/1,000,000.4.As the most common complication of epidural anesthesia procedures is the headache of dural postpunction (CPPD), in relation to CSF hypotension syndrome, due to extasia of it in the lumbar puncture, which leads to a decrease in intermission pressure. As a characteristic of CPPD, the postural nature of CPPD stands out, appearing or deteriorating about 15 minutes after the beep and improvement similar time with decubital. in accordance with the diagnostic criteria of the International Headache Society (2004, ICHD-II)5.6. Studies show that the duration of symptomatology does not go beyond about 5 days in most cases5. When the decrease in CSF pressure is suddenly performed, the displacement of brain structures can lead to rupture of intracranial subdural veins, which can cause HS7-10. Many authors have associated lumbar puncture techniques and the material used at the beginning of GS as a complication, given that the larger diameter, type of pencil tip and needle bezel are used more predisposed to vascular injury1. However, the type of needle used does not appear to be the solution to the problem as cases of GS secondary epidural anesthesia techniques with fine rut1-5 needles have been reported. It should be noted that in the acute phase of the GS, as a result of the increase in brain volume, increased intracranial pressure (PIC) is caused by that in the advanced phases of the brain subjects the stem of hypoperfusion and ischemia, causing an increase in the activity of the sympathetic and parasympathetic autonomous nervous system, which tries to increase the volume of heartbeat and PA to levels that exceed the pressure exerted on the brain stem, in order to overcome the vascular resistance to cerebral blood flow generated by the increased PIC11.15 This physiological reaction to PIK height is called the Cushing effect and is clinically characterized by a triad of high blood pressure, bradycardia and mis-breathing, signs of poor prognosis. In the case of our patient, bradycardia was detected without any time with high blood pressure or irregular breathing. The duration of the headache for one week after the lumbar puncture for epidural anesthesia, the disappearance of its postural nature and the appearance of a related neurological focal point should put us on notice of the existence of acute intracranial pathology, whose symptoms will no longer depend on CSF hypotension as a typical symptoms of CPPD, but intracranial hypertension, mass effects and intracranial hypertension. HS.

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