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RESEARCH ARTICLE

HEMATOMA OF THE PSOAS MUSCLE IN A PATIENT ON ANTICOAGULANTS: CASE REPORT

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Abstract

Psoas hematoma is one of the most serious complications of anticoagul ant therapy. It occurs mainly in patients treated with Heparin or Warfarin, either in cases of overdose or even during properly managed treatment. Clinically, it presents with intense pain, muscle paralysis, and sensorimotor deficits along the course of the femoral nerve, which are difficult to identify in intensive care patients. Treatment is mainly conservative; surgical management has specific indications and requires correction of hemostasis disorders, which justifies the use of percutaneous drainage. Managing anticoagulants or deciding whether to continue them in such situations is a therapeutic challenge.We report the case of a 78-year-old patient admitted for management of a head injury, anticoagulated for a pulmonary embolism, who developed a vitamin K antagonist (VKA) overdose with a psoas hematoma during hospitalization.

Introduction:-

Anticoagulants are medications used mainly for the preventive or curative treatment of thromboembolic disorders. Their use requires rigorous monitoring because, even when properly supervised, patients on anticoagulants are prone to complications of varying severity. Hemorrhage is the principal complication of this therapy, and it has been reported that each year, approximately 1–7% of patients on anticoagulants experience hemorrhagic complications [1].Psoas hematomas are rare but serious complications that occur not only in anticoagulated patients but particularly in individuals with hemophilia [1]. Clinical manifestations vary depending on the degree of femoral nerve compression. Treatment may be conservative or surgical. In such complications, the decision to stop or maintain anticoagulant therapy represents a major challenge and highlights the importance of balancing risks and benefits.We report the case of a 78-year-old man admitted for management of a head injury, anticoagulated for a pulmonary embolism, who developed a VKA overdose complicated by a psoas hematoma during hospitalization.

Case Report:

We report the case of a 78-year-old patient admitted for management of a cranial impact trauma. Initial clinical evaluation found a Glasgow Coma Scale score of 12/15, symmetrical reactive pupils, no sensorimotor deficit, blood pressure of 120/09 mmHg, heart rate of 86 bpm, respiratory rate of 30 breaths/min, and oxygen saturation (SpO2) of 89% on room air. After stabilization, a full-body CT scan revealed a 7 mm subdural hematoma associated with minimal subarachnoid hemorrhage and pulmonary contusion areas, without additional traumatic abnormalities.

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Three days after trauma, the patient developed neurological deterioration requiring ventilatory support. Control brain CT showed stable lesions; brain MRI revealed diffuse axonal injury. The patient received intensive care support, and low-molecular-weight heparin (LMWH) prophylaxis was initiated on day 7 post-trauma to prevent thromboembolic disease. Neurological improvement followed, and the patient was extubated on day 17 after trauma.During hospitalization, the patient developed increased oxygen requirements without signs of pulmonary infection, along with supraventricular tachycardia. A chest CT angiography revealed a left segmental pulmonary embolism with right heart chamber dilation but no echocardiographic signs of acute cor pulmonale. Therapeutic anticoagulation with enoxaparin 6000 IU every 12 hours was initiated, resulting in good respiratory improvement. On day 7 of anticoagulation, and after stabilization of cerebral lesions, a transition to acenocoumarol was started, with the INR remaining within the therapeutic range for the first five days.On day 5 of the transition, the patient developed anemia, with hemoglobin dropping to 7.5 g/dL from 11 g/dL. INR was 9. Clinical examination revealed abdominal compartment syndrome (intravesical pressure of 20 mmHg and oliguria) without external bleeding. After symptomatic treatment (discontinuation of anticoagulants, blood transfusion, vitamin K administration), an abdominopelvic CT scan showed a left psoas muscle hematoma measuring 11×9×25 cm (Figure 1). A drain was placed near the renal fossa, yielding 300 cc of hemorrhagic fluid. The patient improved with cessation of bleeding, normalization of intra-abdominal pressure, and improvement of laboratory parameters (INR, hemoglobin, renal function). Follow-up ultrasound showed a reduction in hematoma size, and iso-coagulant prophylactic anticoagulation was resumed.

Discussion:-

Psoas hematoma is a rare but potentially severe complication that may occur after trauma, anterior iliac crest bone graft harvesting, hip arthroplasty, but mainly in hemophiliacs and patients on anticoagulants [1]. Heparin and warfarin are the drugs most often implicated [2]. It has also been reported with antiplatelet agents [3]. This complication has previously been described only in isolated case reports and small series, so its incidence remains poorly known [4]. Retroperitoneal hemorrhage incidence, however, has been reported at 1.3–6.6% in patients receiving therapeutic anticoagulation, compared with 5.5–10.4% in hemophiliacs [3]. Anticoagulant-related psoas hematomas may occur even without overdose. Although generally unilateral, rare cases of bilateral hematomas have been reported [1].

Clinically, psoas hematoma causes intense pain, muscle dysfunction, and sometimes nerve paralysis, most often affecting the femoral nerve due to its anatomical pathway. Symptoms vary from iliac fossa abdominal pain to neuralgic irradiation into the thigh, with variable degrees of motor and/or sensory deficit from femoral nerve compression, or even subischemic presentation in the lower limb. Rapid or voluminous hematomas may result in anemia or hemorrhagic shock [5].The particularity of our case lies in the hematoma's presentation as an abdominal compartment syndrome complicated by anuria and anemia. These conditions are common in intensive care and can delay diagnosis, especially when pain cannot be verbalized due to neurological impairment.

In the absence of surgical indications—namely compressive hematoma with neurological signs [5]—treatment is usually conservative, consisting of bed rest, analgesia, and correction of coagulation disorders [6]. Hemorrhagic complications from anticoagulants pose a considerable challenge, especially when discontinuation risks worsening the primary disease. In this case—segmental pulmonary embolism versus major hemorrhage—the decision to stop anticoagulation was debated. However, due to overdose and an INR of 9, temporary discontinuation with blood products and vitamin K was chosen. Many case reports describe clinical improvement after stopping anticoagulants, reversing coagulopathy, and nonsurgical management [1,3,7]. Continuation of anticoagulation has been documented in only one case, under strict clinical and INR monitoring.In our patient, management required surgical intervention due to compartment syndrome and renal impairment. Percutaneous decompression was chosen as the preferred approach—an increasingly attractive alternative to conventional surgery thanks to advances in ultrasound and CT imaging.

Conclusion:-

Psoas hematoma is a rare complication of anticoagulant therapy and may occur even during well-managed treatment. Our case illustrates the diagnostic difficulty in intensive care settings, where key symptoms may be absent, and highlights the therapeutic challenge of managing anticoagulants in patients who may also present major thromboembolic events.

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