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Case Report

Acquired Factor VII Deficiency in Cerebral Injury (Case Report)

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Abstract

Factor VII (FVII) is a glycoprotein with a short half-life (4 - 6 hours) in circulation, synthesized by the liver under the dependence of vitamin K.

Severe coagulopathy is a common and serious problem in a neurosurgical patient. As such, liver diseases, vitamin K deficiency, or use of vitamin K antagonists is the cause of acquired deficiency. We could also find rarer causes of factor VII deficiency.

Here we describe a case of acquired factor VII deficiency in cerebral injury in a patient hospitalized in the intensive care unit at the university hospital Ibn Rochd.

Keywords: *Factor VII (FVII); Vitamin K; Cerebral Injury*

Introduction

Factor VII (FVII) is a glycoprotein with a short half-life (4 - 6 hours) in circulation, synthesized by the liver under the dependence of vitamin K.

Severe coagulopathy is a common and serious problem in a neurosurgical patient. As such, liver diseases, vitamin K deficiency, or use of vitamin K antagonists is the cause of acquired deficiency. We could also find more rare cause of factor VII deficiency.

Here we describe a case of acquired factor VII deficiency in cerebral injury.

Case Report

An unidentified elderly male patient was admitted in intensive care unit for consciousness disorder and tonic-clonic seizures.

The initial clinical assessment found an inconscious patient, with a GCS at 8/15, with a breath shortness at 34 cycle/min, an SpO_2 at 85% in ambient air. The patient was stable hemodynamically.

The initial management consisted on oxygen therapy using a high concentration mask and a venous access.

After that, a protective mechanical ventilation was started, and continuous sedation associating Fentanyl and Midazolam was initiated. Management of hemodynamic state required the use of norepinephrine.

Thoracic and cerebral CT was performed:

- Appearance of venous thrombosis of the upper longitudinal sinus
- Low abundance right pneumothorax
- Moderate bilateral pleural effusion with underlying collapse.

A biological assessment was carried out objectifying a decreased prothrombin level, supplemented by a dosage of factor VII which was low. a CRP at 220 mg/L. In addition, the patient had a normal hepatic and renal function and cardiac enzymes.

The patient management steps were the administration of blood plasma and other symptomatic treatment with a good final outcome, and was transferred in other unit.

Discussion

In our case, cerebral venous thrombophlebitis led to the discovery of FVII deficiency.

Acquired FVII deficiency is a rare coagulation disorder whose pathophysiological mechanism is sometimes difficult to elucidate, which explains why no therapeutic protocol has been validated for the treatment. However, the use of fibrinolytics, fresh frozen plasma, prothrombin complex and rFVIIa depends on their availability and the severity of brain damage [1].

The decrease in the level of prothrombin (PT), initially normal, and the normal level of factor V, led us to the diagnosis of acquired FVII deficiency.

Acquired factor VII deficiency is found in patients with discordance between a prolonged PT and normal aPTT. For the diagnostic of this deficiency, coagulation factor activity need to be determined and the decrease of FVII level need to be found [2,3].

A factor inhibitor is suspected when the PT remains prolonged after mixing of patient plasma with normal pooled plasma, a specific FVII inhibitor is suspected based on The Bethesda assay [4].

There are two types of discovery circumstances. In the context of medical pathologies, the determination of FVII is carried out in front of a decrease in PT, not corrected by vitamin K and associated with a normal TCA. The pathologies encountered are then essentially neoplastic and infectious conditions. During trauma, as the case we report, it is the absence of correction of the PT by PFC that is suggestive. This type of deficit has been reported in the context of severe head trauma or in the perioperative period of cardiac surgery [5].

Our patient did not have any other complications secondary to the treatment or to the FVII deficiency. And there is no other evidence in favor of another thromboembolic disease.

Coagulopathy is a common complication of cerebral damage, and findings in clinical series suggest that the degree of coagulopathy correlates with the severity of the cerebral injury. Although frequently seen as a consequence of trauma, coagulopathy may result from non-traumatic lesions of the brain.

Therapy for coagulopathy currently centers on FFP. Management of coagulopathy may require multiple doses of FFP. During this time, the patient cannot receive interventions such as placement of ICP monitors, ventriculostomies, and surgery.

rFVIIa is effective in rapid reversal of coagulopathy in neurosurgical patients when he is used after initial administration of vitamin K and FFP as suggested by Ben Roitberg study.

Ben Roitberg, in his quest to make rFVIIa a first-line treatment in neurosurgical patients needing immediate reversal of coagulopathy, propose a prospective trial, evaluating key issues of time to reversal, the need for repeat doses or additional FFP, any time savings to get to surgery, and the cost of the treatment with rFVIIa compared with FFP [6,7].

Conclusion

The diagnosis of acquired factor VII deficiency in brain injury should be suspected when PT prolongation is found.

Moreover, for a mortality of 13%, the evolution of the patient was satisfactory.

Bibliography

1. Slease RB and Schumacher HR. "Deficiency of coagulation factors VII and XII in a patient with Hodgkin's disease". *Archives of Internal Medicine* 137.11 (1977): 1633-1635.

2. Campbell E., et al. "Factor VII inhibitor". *American Journal of Medicine* 68.6 (1980): 962-964.

3. Delmer A., et al. "Acquired factor VII inhibitor: treatment using high-dose immunoglobulins, corticotherapy and plasma exchange". *Annales de Medecine Interne* 139.1 (1988): 48-50.

4. Delmer A., et al. "Life-threatening intracranial bleeding associated with the presence of an antifactor VII autoantibody". *Blood* 74.1 (1989): 229-232.

5. Bruce DA. "Scope of the problem-early assessment and management". In Rosenthal M, Griffith ER, Bond MR., *et al*. (eds): Rehabilitation of the Adult and Child with Traumatic Brain Injury. Philadelphia: FA Davis (1990): 521-537

6. Arkin S., et al. "Activated recombinant human coagulation factor VII therapy for intracranial hemorrhage in patients with hemophilia A or B with inhibitors. Results of the novoseven emergency-use program". *Haemostasis* 28.2 (1998): 93-98.

7. Muleo G., et al. "The use of recombinant activated factor VII in congenital and acquired factor VII deficiencies". *Blood Coagulation and Fibrinolysis* 9.4 (1998): 389-390.

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