

Subarachnoid Hemorrhage in Patient with End-Stage Renal Disease Secondary to Thrombocytopenia

Mayra, Evelyn Quinones Martinez¹, Kim Rodriguez Reling²

^{1,2} Department of Internal Medicine High Specialty Hospital of Veracruz, Sesver, Veracruz, Mexico Av.20 De Noviembre 1074 Ver, Ver

ABSTRACT

This is a case report of a 60-year-old patient with renal disease of 21 years of evolution without renal replacement therapy, as well as systemic arterial hypertension with poor adherence to treatment, began his condition with indifference to the environment, drowsiness, admitted to the emergency room where, thrombocytopenia and in cabinet studies take computed tomography of the skull with report of subarachnoid hemorrhage. The patient presents with thrombocytopenia due to consumption secondary to hemorrhage and platelet dysfunction due to chronic kidney disease stage V, this patient required transfusion support of 5 platelet units and two globular packs, who laboratorially reflected an increase in platelet levels and clinical improvement evaluated with the Glasgow Coma Scale.

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INTRODUCTION

Thrombocytopenia

Thrombocytopenia is defined as a platelet count below 150,000 per μL ¹⁻² With the incorporation of hematology autoanalyzers or electronic (cell) counters to most clinical laboratories, which routinely include platelet counts and new platelet-related parameters in the CBC, the finding of thrombocytopenia is becoming more frequent ³. The prevalence of thrombocytopenia is highly variable, occurring in 0.9% of emergency consultations ⁴ and between 25% and 41% of patients in intensive care units ⁵. Diagnosis will always be by exclusion, in those situations presenting with isolated thrombopenia in the absence of systemic disease or drug exposure and with a normal or hypercellular bone marrow. The blood count will reveal a reduced platelet count, usually between 10-50 per 10⁹/l. Anuclear antibodies will be requested to determine the existence of concomitant connective tissue diseases such as SLE. Bone marrow aspirate will reveal a normal or slightly increased number of megakaryocytes. Emergency treatment will be instituted in the presence of thrombopenia associated with bleeding and will be carried out with prednisone at a dose of 1-2 mg/kg/day/orally. After 3 weeks, a new count will be carried out and if it is acceptable (figures between 80,000-90,000/ μl), gradual withdrawal will be carried out. The platelet count will normalize in 50% of patients, although most of them will suffer a decrease in platelet count once they stop taking

corticosteroids. In the event of major bleeding, in addition to corticosteroids, immunoglobulin infusion at doses of 400 mg/kg/day/5 days or 1 g/kg/day/3-2 days should be used. The mechanism of action of this therapy is through blockade of In the event of resistance to treatment, splenectomy should be performed, with 70-80% responding partially or completely, although in patients over 45 years of age and postmenopausal women, treatment with danazol (200-400 mg/12 h) should be tried beforehand. In those patients refractory to these measures, immunosuppressants should be used: cyclophosphamide in a continuous regimen (2 mg/kg/day) or in boluses (1 g/m²), azathioprine, cyclosporine A, dexamethasone in boluses or interferon alpha. Platelet transfusion is only indicated in the presence of life-threatening ⁶.

Subarachnoid hemorrhage

About 80-85% of subarachnoid hemorrhages (SAH) are due to rupture of spontaneous cerebral aneurysms, 10% non-aneurysmal perimesencephalic hemorrhage (cerebral venous thrombosis, trauma, pituitary apoplexy) and 5% of unknown causes ^{7,8,9,10}. Other less common causes. There are 3 types of aneurysms according to their morphology: dissecting, fusiform (arteriosclerotic) and saccular; being the saccular the most frequent. The most common location of these aneurysms is in the anterior circulation and in stress points, up to 20% of cases of SAH have multiple aneurysms at the time of diagnosis ^{11,12}. Computed tomography (CT) without

Subarachnoid Hemorrhage in Patient with End-Stage Renal Disease Secondary to Thrombocytopenia

contrast medium is the diagnostic gold standard, since it has a sensitivity of 100% in the first 6 hours; if performed within 24 hours of the onset of symptoms, hyperdensity (blood in the subarachnoid space) can be observed in 90% of cases^{8,9,10}. Treatment of subarachnoid hemorrhage must be individualized in each case and taking into account the patient's condition, and requires a multidisciplinary team of neurosurgical specialists. It should be performed as quickly as possible, within the first 72 hours, to reduce the likelihood of rebleeding. Total occlusion of the aneurysm is recommended whenever possible surgical treatment that includes performing craniotomy and placement of clips at the neck of the aneurysm; it is indicated in patients with intraparenchymal hematoma of large volume (more than 50 ml), wide neck aneurysms and middle cerebral artery aneurysms¹³. However, medical management after surgical or endoscopic surgery is of vital importance: management of glycemia, blood pressure, sodium levels and temperature control among others, since this management has improved

morbidity after surgery and decreased complications and mortality. A better prognosis has been documented if patients are placed in services qualified for neurological events (stroke, cerebral hemorrhages, among others)¹⁴.

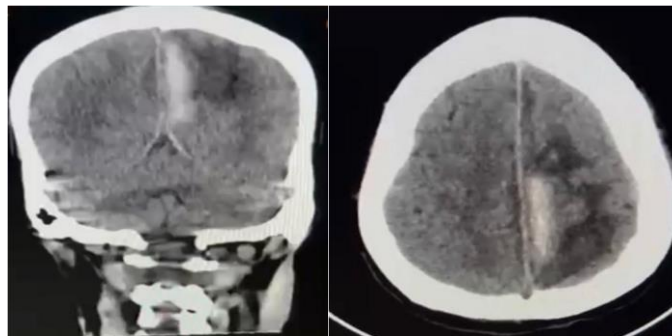
Case Presentation

60-year-old male patient with a chronic degenerative history of systemic arterial hypertension, 7 years of evolution, renal disease of 21 years of evolution, starting his current condition on April 21, 2023 with neurological deterioration, characterized by indifference to the environment and drowsiness, reason for which he is taken to hospital care, on admission laboratory studies are taken reporting thrombocytopenia, and in cabinet study with axial tomography of the skull report subarachnoid hemorrhage Fisher IV. For this reason he was hospitalized to follow the protocol of remission of subarachnoid hemorrhage and thrombocytopenia.



Simple tomography of the skull on admission 24.04.23 shows a Fisher IV subarachnoid hemorrhage. Labs on admission: Hb 7.30, Ht 21.20, MCV 91, Platelets 27.000, Leukocytes 7.88, Platelets with sodium citrate 20.0 Blood chemistry: Glucose 97, Urea 211.9, Cr 6.18, Serum electrolytes: Calcium 7.10, Phosphorus 6.80, Magnesium 2.93, Sodium 123, Potassium 3.80, Chlorine 91.

Labs at 3 days: Hb 13.90, Ht 37.90, MCV 85, Platelets 36,000, Leukocytes 6.29, Blood chemistry: Glucose 117, Urea 119.8, Cr 2.90, Serum electrolytes: Calcium 7.90, Phosphorus 7.50, Magnesium 2.40, Sodium 110, Potassium 4.14, Chloride 67.60, Liver function tests: AST 34, ALT/TGP 31, ALP 119.



Simple cranial tomography 8 days after admission still shows cerebral edema with data of intraparenchymal hemorrhage of the left parietal region.

Labs 8 days after application of 5 platelet packs and 1 globular pack: Hb 7.20, Ht 22, MCV 97.80, Platelets 146.000, Leukocytes 9.34, Clojng 5me TP 10.3, INR 0.9, APTT 27.5, Fibrinogen 472, Blood chemistry: Glucose 83, Urea 321, Cr

Subarachnoid Hemorrhage in Patient with End-Stage Renal Disease Secondary to Thrombocytopenia

4.55, Serum electrolytes: Sodium 136, Potassium 4.90, Chlorine 113.

DISCUSSION

Patients with chronic renal disease who do not receive any type of renal replacement therapy may develop thrombocytopenia due to consumption causing hemorrhage and platelet dysfunction, causing this type of massive hemorrhages at cerebral level, keeping the patient with glasgow of 8 and gradually improving his neurological status until leaving him in a vegetative state with no response to the environment with glasgow of 3, Therefore, it is of utmost importance to have this type of patient in a tertiary level hospital to receive multidisciplinary management where he will be evaluated by the neurosurgery service for subsequent platelet pack replacement by the hematology service. This patient was discarded by the neurosurgery service, he was only managed with an5hemorrhagics and subarachnoid hemorrhage, he did not improve during his stay in the hospital, laboratorially he improved by reposi5oning the platelet packs given, but without any recovery to the environment.

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