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Hemodialysis induced severe thrombocytipenia: A case report

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Abstract

Thrombocytopenia is commonly seen in patients undergoing hemodialysis, and effectively addressing and investigating this condition poses challenges. Research indicates that platelet counts may decrease by as much as 15% during hemodialysis sessions, although they typically recover post-treatment. This decline in platelet levels is attributed to mechanisms such as platelet adhesion and complement activation, which occur irrespective in type of dialysis membrane used. Various studies examining platelet surface markers have shown that exposure to cardiopulmonary bypass can lead to heightened platelet activation and aggregation.

This case report focuses on a hemodialysis patient who experienced severe thrombocytopenia during hospitalization. To identify potential causes, an investigation ruled out heparin-induced thrombocytopenia, medication side effects, hypersplenism, and hematological disorders. It was then proposed that the thrombocytopenia might be related to the hemodialysis process itself, especially since the platelet count significantly dropped during sessions but showed partial recovery following changes to the dialyzer. Consideration of the sterilization techniques and the practice of dialyzer reuse is crucial for managing this issue. In this particular instance, the reuse of the dialyzer was effective in reducing the extent of platelet count decline associated with cyclical dialysis.

Introduction

The decrease of total platelet count is often observed in patients receiving hemodialysis, presenting significant challenges for accurate diagnosis and management. It has been noted that during hemodialysis sessions, platelet counts may drop by as much as 15%, although they usually rebound after treatment concludes. This thrombocytopenia is related to factors like the adhesion of platelets and the activation of the complement system which occur in dialysis.

Several studies examining the surface markers of platelets reveal that there is increased activation and aggregation of platelets, particularly following procedures such as cardiopulmonary bypass.

In our clinical report, we discuss a hospitalized patient undergoing regular hemodialysis and due to dialysis developed significant thrombocytopenia. A comprehensive evaluation was conducted to exclude common causes, including heparin-induced thrombocytopenia, adverse drug reactions, hypersplenism, along with other blood disorder conditions. Eventually, hemodialysis-related thrombocytopenia was considered, as the platelet drop was more significant during dialysis cycles in which some recovery was observed on changing the dialysing unit. Also paying attention to the sterilization process and repeated usage of the dialyzer proved beneficial in reducing the decline in platelet counts linked to hemodialysis.

Case report

A 56 year-old male, who has been on regular dialysis treatment for seven months, had been admitted due to complaints of severe shortening of breath, dyspnoea and bilateral pedal edema due to missing two cycle of hemodialysis in he past week had ben admitted foe emergency hemodialysis. His clinical history includes type two diabetes mellitus, hypertension , obstructive sleep apnoea, ischemic heat disease for which he got a ptca done 5 yeas ago and grade two fatty liver with hepatomegaly for which he is on regular medications. He has stage five chronic kidney disease caused to due hypertensive etiology. He history of chronic smoking. The patient undergoes three cycles of hemodialysis each week with the Fresenius hemoflow 80 Capilar, following the standard regulations regarding reuse.

His current medication regimen includes human actrapid insulin, metoprolol, telmisartan, metformin, atorvastatin statin, clopilet, minipress xt, ecosprin gold, pantoprazole, erythropoietin, and clexane. On the time of patient admission, he had a platelet count of 120,000/mm³ (with a reference range of 150,000 - 450,000/mm³) and hemodialysis sessions were initiated.

Following the regular and next sessions of hemodialysis using a Fresenius Helixone Fx 800 dialysing unit, the patient's platelet count decreased significantly to 54,000/mm³. Remarkably, he had no symptoms during the cycles of hemodialysis and showed no bleeding tendencies or symptoms. The diagnosis of thrombocytopenia was corroborated through manual counting, during which we ruled out any blood clots present along with any and indications of idiopathic thrombocytic etiologies.

Assessment using the 4T score indicated a moderate likelihood of Heparin-Induced Thrombocytopenia (HIT), prompting the discontinuation of all heparin products, including the one sealing the catheter. Pantoprazole was substituted with ranitidine. Despite these changes, there were no sings of improvement in the patient showed, leading to consideration of thrombocytopenia linked to the capillary membrane. Consequently, the Fresenius helison fx 60 preferred for use for the subsequent cycles of hemodialysis. However, the patient continued to experience significantly lowered platelet counts, dropping to 38,000/mm³. A thorough hematological evaluation was conducted, ruling out a medullary cause after an extensive assessment. Tests including protein electrophoresis, immunoelectrophoresis, ana screening, serum cortisol, coagulation profiling and HIV and Hbsag serologies all returned as negative results. A bone marrow biopsy further revealed no abnormal immunophenotypic results. The liver function tests were also normal, and ultrasound showed mild grade one fatty liver disease with no evidence of significant and relative hepatomegaly.

Therefore, the patient was observed to have oscillatory thrombocytopenia, which did not improve following the suspension of heparin. Platelet production remained normal, there was no indication of hypersplenism, and a significant reduction in platelet levels was noted during the hemodialysis session. At this point, coagulogram results were normal, and the patient did not exhibit leukopenia; however, elevated d-dimer levels were detected alongside moderately elevated fibrinogen (results: TP INR 1.0 - 10.2 sec - RV: 0.6-10.9; TTPA R 1.18 - 33.2 sec - RV: <1.19; DHL 158 IU / L - RV 80-238 IU/L; fibrinogen 451 mg/dL - RV: 250-380 mg/dL; D-Dimer: 1.08 µg/mL - RV: <0.3 µg/mL).

With the aim to further analyze the impact of hemodialysis, the total platelet counts were recorded immediately before the procedure, 45 minutes into it, and 45 minutes post-treatment. Our findings indicated a decrease in platelet levels that exceeded the anticipated 15%. The initial platelet count before dialysis was measured at 56,000/µL, which fell to 25,000/mm³ 45 minutes later, and then dropped further to 18,000/mm³ 45 minutes after the session concluded. There was no abnormalities in the bone marrow findings of the patient, during the dialysis session, there was notable platelet consumption. Consequently, the dialysis unit was changed from the Fresenius Helixone Fx 60 to the Fresenius hemoflow, which is identical to that currently being utilized on OPD basis. The trend of drop in the platelet count remained the same. The platelet count checked prior to initiating dialysis was 67,000/mm³ which after 45 minutes of dialysis, it dropped to 31,000/mm³, and a further 45 minutes post-dialysis, declined further to 16,000/mm³.

Fortunately, there were no bleeding indications present, and the platelet count rebound to 84,000/mm³, allowing for the discharge of the patient for continued to be discharged for ongoing outpatient management and treatment. Following three hemodialysis sessions in the outpatient clinic with the Fresenius hemoflow dialysing unit and on the setting of nil heparin, the patients platelet count prior to hemodialysis was 98,000/mm³, which dropped to 78,000/mm³ during the session, maintaining a predictable decrease of about 15%.

One and two months after discharge, routine clinic tests showed platelet counts of 168,000/mm³ and 147,000/mm³ respectively. The use of heparin was eventually reintroduced for hemodialysis, and three months later, the patient's platelet count was recorded at 154,000/mm cube during their last appointment with a hematologist.

Discussion

Thrombocytopenia is a common issue for patients undergoing renal replacement therapy, and addressing it can be quite complex. After ruling out conditions like bone marrow disorders, both immune and non-immune causes of increased platelet destruction, heparin-related issues, adverse drug reactions, and hypersplenism, the primary focus tends to be on platelet consumption during hemodialysis. In individuals on hemodialysis, platelet levels typically drop to a value of 160-175.000/mm cube, compared to the normal level of 250,000/mm³ found in healthy individuals. Research indicates that there is a 5-15% reduction in platelet counts during the initial thirty minutes of hemodialysis, with normalization by the time of completion of the hemodialysis cycle session, with nil significant bleeding complications reported. Various factors, including the type of dialyzer, its cleaning process, and whether it is reused, can influence these platelet changes.

Dialyzers can be cleaned and reused after rinsing them to remove blood and sterilizing them, a practice that is generally regarded as safe and effective. In the 1990s, a majority of about 80-90% of dialysis patients in U.S.A were on usage of reused dialyzers, though this system of usage has declined in recent years. While there are benefits, such as reduced exposure to chemicals from new dialyzer production and improved biocompatibility due to less immune activation, there are also risks, including potential bacterial contamination and chemical exposure during reprocessing, as well as a possible decline in b2-microglobulin clearance.

Currently, dialyzers made from materials that trigger less immune activation, such as cellulose acetate, polysulfone, and polymethacrylate, are preferred over older cellulose-based models. Research is ongoing into alternative pathways of platelet activation that do not involve the complement system, which could play a role in thrombocytopenia.

Factors such as blood flow rates, turbulence, and an increase in hematocrit caused by ultrafiltration can lead to hypercoagulability in external circuits due to direct blood contact with the dialyzer. This activation can initiate the coagulation cascade, leading to platelets being consumed in the clot formation pro-

cess. Evidence of elevated D-dimer levels in the patient suggests that this pathway may be more relevant in the context of dialysis-related platelet consumption rather than the classical complement pathway.

Clinical literature supports the notion of thrombocytopenia and its direct clinical corelation to hemodialysis, inclusive of the usage of biocompatible dialysis membranes, and it's recognized that platelet consumption can occur through mechanisms beyond the complement pathway. Various treatment strategies have been proposed, including changing the dialysis materials, altering the sterilization method, or rinsing the system with saline before treatment. Unfortunately, there was no data available on antibody levels to conclusively rule out Heparin-Induced Thrombocytopenia (HIT), although the evidence suggested a connection to the hemodialysis procedure itself without improvement after discontinuing heparin.

Different sterilization methods employed by manufacturers directly affect biocompatibility. Studies indicate that dialyzers sterilized with electron beams are associated with significant thrombocytopenia after dialysis, as this method may alter the membrane properties and research into these effects is still ongoing.

Platelet activation and aggregation often increase on exposure to materials in extracorporeal setups, furthermore, on the usage of biocompatible membranes, and a decrease in the total count of platelets is common at the time of initiation of dialysis. However, continuous low platelet levels warrant closer scrutiny. Adjusting dialyzer materials, sterilization techniques, and considering reuse practices may help address this complication. In our patient's case, merely the change of the dialysis unit and membrane type was insufficient, necessitating reutilisation of dialysis unit.

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