Hypotensive transfusion reaction – a case report

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ABSTRACT

Transfusions reactions can be caused by the toxicity of chemicals that leach in blood components from blood storage containers or by endogenous mediators generated in the blood during filtration, processing and storage, such as bradykinin mediated hypotensive reactions. Such transfusion reactions are characterized by early and abrupt onset of hypotension as the predominant clinical manifestation and subside once transfusion is stopped. We report a case who presented with hypotension on blood transfusion and its management.

Keywords: blood transfusion, hypotension, adverse reactions

INTRODUCTION

Each unit of blood or blood component transfused is associated with the possibility that patient may experience an adverse reaction to the product transfused. These reactions may range from mild febrile non hemolytic transfusion reaction or allergic reaction to hemolytic transfusion reaction. However, hypotensive transfusion reactions are less well recognised.[1,2] We hereby report a case of hypotensive transfusion reaction.

CASE REPORT

A 48 year old female was admitted in the emergency medicine ward with the chief complaint of increased shortness of breath since 2 weeks. She had hypothyroidism and was a patient of chronic kidney disease since four years. On physical examination, clubbing, pallor and bilateral wheeze were present. There was no pedal edema and heart sounds were normal on auscultation. On laboratory investigation, her hemoglobin was 7.5 g/dl, total leucocyte count (TLC) was 18,500/µl and differential leucocyte count (DLC) showed predominantly neutrophilia (84%). Her renal function tests were deranged with blood urea of 136 mg/dl and serum creatinine of 5.4 mg/dl. Her chest X-ray showed non homogenous opacity in left lower zone. Patient was shifted to intensive care unit (ICU) after 10 hours of admission and dialysis was planned. A request for 2 units of packed red blood cells (PRBCs) was received in the department.

On forward and reverse grouping, her blood group was found to be A Rh D positive. Two units of A Rh D positive packed red cells were cross-matched for the patient using tube technique and were found to be incompatible in anti-human globulin (AHG) phase. Antibody screen and identification was performed using microcolumn gel technique (BioRad, Switzerland) and anti E and anti c antibodies were identified. Antigen negative units were cross-matched and found compatible for the patient using microcolumn gel technique.

The treating physician demanded one unit of packed red cells which was issued to the patient. Before starting the transfusion, her vitals were within normal range. However, after about 80-90 ml of blood transfusion, patient experienced hypotension (90/60 mmHg) and tachycardia (160/min). The patient was afebrile and there were no rigors or chills. Blood transfusion was stopped and symptoms subsided. Blood unit along with post transfusion sample was sent to the department for transfusion reaction work up.

On reaction work up, blood group was confirmed to be A Rh D positive, cross-match was found to be compatible in AHG phase with both pre and post transfusion sample

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and direct anti-globulin test (DAT) was negative. Second unit of blood was issued the subsequent day and patient experienced similar symptoms. Transfusion reaction work up again revealed no discrepancy. Hence, a diagnosis of acute hypotensive transfusion reaction (AHTR) was made. Patient was provided with washed red blood cells on three different occasions and all the transfusions were uneventful.

Acute hypotension can occur as a part of constellation of symptoms resulting from bacterial contamination of blood products, acute hemolysis, and transfusion related acute lung injury and anaphylaxis.[3] Recently, acute hypotensive transfusion reactions have been characterized by early and abrupt onset of hypotension as the predominant and sole clinical manifestation which resolves quickly once the transfusion is stopped.[4] Similarly in our case, patient experienced only hypotension and tachycardia with absence of fever, chills, urticaria, laryngeal edema or flank pain. Symptoms resolved within half an hour once transfusion was stopped.

Hypotensive reactions occur due to disturbances in the production and metabolism of bradykinin. Bradykinin is generated as a result of Factor XII activation due to contact with negatively charged surfaces such as tubing systems, dialysis membranes and leukoreduction filters. Bradykinin is primarily metabolized by the angiotensin converting enzyme (ACE) and is a vasoactive peptide that binds to receptors on the endothelium causing hypotension. An increase in the recognition of such reactions has been due to the growing use of ACE inhibitors, the use of negatively charged leukoreduction filters and both genetic and induced alterations in bradykinin kinetics.[4] In our case, patient was a known case of chronic kidney disease and was undergoing dialysis for it.

In 1978, first report of hypotensive reactions resulting from the use of plasma derivatives was published. However, the authors failed to identify kallikrein or bradykinin in the samples.[5] Subsequently, correlation of the generation of bradykinin in plasma derivatives to the development of hypotensive transfusion reactions was recognized.[6] Usually, such reactions are self limiting and symptoms subside once the transfusion is stopped. However, if repeated transfusions are required, then use of washed cellular blood products, discontinuation of leuco-reduction filters or ACE inhibitors, use of kallikrein blockade drugs has been reported.[4,7] Similarly in our case, patient did not experience any transfusion reaction symptoms with washed packed red blood cells suggesting the likely role of plasma protein fraction in causing symptoms probably activated after contact with negatively charged dialysis membrane. Anaphylaxis was ruled out as patient had only hypotension and tachycardia without any urticaria, flushing or edema. However, bradykinin or kallikrein levels could not be performed due to financial constraints.

CONCLUSION

The exact pathophysiology of hypotensive transfusion reactions is probably multifactorial. Therefore, further research needs to be done in understanding and identifying the etiology and factors involved in causing these transfusion reactions for the optimum management of the patient.

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