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Question: While completing the transfusion of one unit of packed red blood cells, my patient's temperature rose from 37.2°C to 38.4°C, without any other associated symptoms. What are the next steps I should take and what could be causing this reaction?

Answer: As with all suspected transfusion reactions, the first step is always to stop the transfusion and report the suspected reaction to the blood bank. Fever during the transfusion of a blood product may occur with febrile non-hemolytic transfusion reactions (FNHTRs), acute hemolytic transfusion reactions, septic transfusion reactions, transfusion-related acute lung injury (TRALI), or could be the result of an underlying medical condition. All of these possibilities must be assessed using clerical, clinical and laboratory evaluations before the patient is transfused any additional product.

To work up a suspected transfusion reaction, the blood product bag should be returned to the laboratory along with a post-transfusion sample of the patient's blood. The laboratory and the blood bank will perform tests to rule out hemolysis and clerical error, as well as a gram stain and culture if bacterial contamination of the blood product is suspected. This testing will help to rule out more serious transfusion reactions such as hemolytic and septic transfusion reactions.

The most common type of transfusion reaction associated with fever is the FNHTR, which occurs in approximately 1% of all blood product transfusions. These reactions are associated with a rise in body temperature of greater than 1°C either during a transfusion or within four hours following a transfusion; symptoms are generally self-limited. FNHTRs are a diagnosis of exclusion which can only be made in the absence of hemolysis, evidence of sepsis, or any underlying medical condition which could result in a fever.

FNHTRs are caused by several different mechanisms. The recipient may have pre-formed alloantibodies which stimulate donor leukocytes to release fever-inducing cytokines. Alternatively, the patient's own leukocytes may be stimulated to release cytokines by immune complexes formed between donor cell antigens and the patient's antibodies. FNHTRs may also be caused by the passive transfer of cytokines that have accumulated within the blood product during storage. Pre-storage leukoreduction of cellular blood products, which is now commonly performed at many blood donor centers, has significantly reduced the incidence of FNHTRs.

Treatment of FNHTRs is symptomatic with antipyretics, and once more serious reactions have been excluded there is no contraindication to additional blood product transfusion. Premedication prior to future transfusions is generally not effective in preventing transfusion reactions for most patients, but it may be considered in patients who have frequent FNHTRs.

References

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