To the Editors:

Disseminated intravascular coagulation (DIC) is a life-threatening clinical state seen in sepsis, burns, prolonged hypoxia and haemolytic transfusion reactions (1). Characteristically, a diagnosis of DIC is made in patients who show a bleeding tendency, a thrombocytopenia, fragmented red cells in the peripheral blood, a prolonged activated partial thromboplastin time (APTT), prothrombin time (PT) and thrombin time (TT), and elevated D-dimers and reduced fibrinogen in the blood. D-dimers are commonly elevated in liver disease, acute leukaemias and malignancy (2). They are also elevated in the aged (3), in prolonged immobilisation (3), deep vein thrombosis (4), pulmonary embolism (4), in atherothrombotic disease (5), cerebral thrombosis (6) and myocardial infarction (7).

I report two patients with a bleeding tendency, fragmented red cells, thrombocytopenia and elevated D-dimers mistaken to have DIC.

Case reports

Patient 1

An 84-year-old man admitted for a massive haematoma in the left thigh following minor trauma had a long-standing abdominal aortic aneurysm and was being investigated for anaemia. He had fragmented red cells, thrombocytopenia, and raised D-dimers (4000 ng/ml, reference value less than 500 ng/ml).

Patient 2

A 64-year-old man in the intensive care unit with an inferior myocardial infarct and cardiogenic shock had a coronary angiogram followed by insertion of stents. He developed haematuria, and his blood showed fragmented red cells, a thrombocytopenia and raised D-dimers (6000 ng/ml).

Since the presence of a bleeding tendency, fragmented red cells, elevated D-dimers and thrombocytopenia suggested a diagnosis of DIC both men were treated with fresh frozen plasma. Had the PT, APTT and TT been done this error could have been avoided.

Both patients had vascular endothelial damage. One had a long-standing abdominal aortic aneurysm and the other an atherothrombotic event with extensive handling of the endothelium and insertion of stents. Both patients were diagnosed to have DIC initially and treated with fresh frozen plasma. However, subsequently, normal PT, APTT, TT and blood fibrinogen levels excluded this diagnosis. Patient 1 was diagnosed to have myelodysplasia which causes thrombocytopenia as well as platelet function defects. The haematuria in the second patient was likely to be due to the thrombocytopenia as it ceased with a platelet infusion.

It is important to remember that the triad of red cell fragmentation, thrombocytopenia and elevated D-dimers, even in the presence of a bleeding tendency, occurs in various clinical conditions other than DIC. Coagulation screening tests must be done to confirm DIC. This would prevent unnecessary use of blood products that are used in the management of DIC, and the exposure of patients to the risks of and unnecessary transfusions.

References


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