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Stroke Associated with Thrombocytopenia

Dr.Sanjith Saseedharan

Head-Dept of Critical Care
S.L.Raheja Hospital- A Fortis Associate
Mumbai

Intravenous thrombrolysis for stroke has become a fairly common procedure as a result of heightened awareness of the condition among the lay people and the medical community. However it is fairly rare to encounter a low platelet count during stroke thrombolysis.

We came across a 44 year lady who was brought to the hospital by coworkers within 15 minutes of right sided weakness of power grade 4/5 in upper and lower limb, left side angle deviation of the mouth and right sided hemi sensory loss .There was no aphasia on arrival to the hospital.History was negative for convulsions or recent fever or malaise. Bloods were drawn for basic lab investigations which included complete blood count,serum electrolytes,serum creatinine and prothrombin time. An ecg was done which revealed normal sinus rhythm.She was rushed for an magnetic resonance imaging as per the stroke protocol of the hospital and the intensive care unit was kept in a state of thrombolysis preparedness. She was found to have an acute left middle cerebral artery territory stroke. She presented to the intensive care unit around 45 minutes post beginning of stroke.By now she had also turned aphasic and power of right upper and lower limb had fallen to grade 3/5 .As per protocol the thrombolytic checklist was verified (except platelet count-as it was not yet available from the laboratory) and thrombolysis was initiated after consent which was given by a cousin of the patient who had by then arrived on the scene. Meanwhile other close relatives were summoned.10 minutes into thrombolysis the laboratory called back saying that the platelet count was found to be 80000 /cmm. with normal serum creatinine levels.Thrombolysis was withheld and immediately further history was sought from the brother of the patient who had arrived on the scene. On history the brother conveyed that her platelets remained on the lower side and she had also undergone a plasma exchange last year.

No other details were available. There was no history of medication use, loss of weight or appetite or diarrhea.

A hematologist was immediately involved in the care of the patient. A reticulocyte count and serum lactate dehydrogenase was sought.A pathologist was requested to review the peripheral smear. Meanwhile perinuclear anti-neutrophile cytoplasmic antibody,anti-nuclear antibody and cytoplasmic anti-neutrophile cytoplasmic antibody was also sent.The lactate dehydrogenase levels and reticulocyte counte was high.A diagnosis of " active" thrombotic thrombocytopenic micorangiopathy was entertained,with probability of the entity being thrombotic thrombocytopenic purpura due to the preponderance of neurological symptoms and absence of features of renal failure. The relatives were counselled and plasmapheresis was instituted in an emergency in order to halt further progression of the stroke as a result of increased thrombotic microangiopathic activity. Patient underwent 6 cycles of plasmapheresis (daily for first three and then alternate day) along with pulse doses of methylprednisolone. Azathioprine was initiated after third cycle of plasmapheresis.Progress of the devastating stroke was noted to have halted and no further worsening of condition was noted. Platelet counts rose after third cycle of plasmapheresis .Lactate dehydrogenase levels returned o baseline within 4 days.Aphasia recovered over 2 weeks. However residual right sided weakness of lower and

upper limbs of power 4/5 persisted. Subsequently patient was discharged from the inyensive care unit on a combination of azathioprine and a steroid tapering regime.

TTP is a rare disorder characterized by extensive microscopic clots that are formed in the small blood vessels throughout the body which cause damage to many organs like the brain ,kidney and heart.[1] TTP is characterised by the classical clinical pentad of thrombocytopenia, microangiopathic haemolytic anaemia (MAHA), fever, renal dysfunction and fluctuating neurological deficit. However usually many of the features of the pentad are absent.[1] Circulating antibodies to ADAMTS13 enzyme[2] or in rarer cases a genetically dysfunctional ADAMTS 13 enzyme is responsible for TTP. inhibition by antibodyIt is not necessary to have the classical pentad of TTP .Heightened level of suspicion should be entertained with neurological signs and low platelet counts.TTP can be primary (idiopathic) or secondary to drugs, autoimmune disease, cancer and infections linked with the human immunodeficiency virus. Whatever the cause, early plamapheresis is the cornerstone of the treatment of thrombotic microangiopathies like TTP[3]Acute phase survival has improved upto 79% after the advent of plasmapheresis5[5].The use of steroids in ttp has been anecdotal in the absence of well conducted rcts.However long term remission and thus reduction of relapses can be achieved by using cytotoxic drugs like azathioprine,rituximab,steroids,cyclophosphamide.[5]

This case reminds physicians involved in stroke care to accelerate investigations to rule in thrombotic microangiopathy in similar cases with reduced platelet counts presenting with strokes.Prompt accelerated plasmapharesis in such cases prevents the disease activity from progressing and thus reducing the possibility of a devastating neurological outcome.This case also lays emphasis on following up basic lab investigations like platelet count which generally takes a backseat in the hurry to achieve a quick door to needle time for thrombolysis.

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