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Case Report

# OCULAR MANIFESTATIONS OF APLASTIC ANEMIA FOLLOWING PLATELET TRASFUSION : A CASE REPORT

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Abstract:
Aplastic anaemia is a rare haemopoietic stem-cell disorder that results in pancytopenia and hypocellular bone marrow. Ocular findings are manifestations of preexisting anemia. Here we are reporting a case of aplastic anemia which presented with the ocular findings following platelet transfusion which has not been reported in literature to the best of our knowledge.

Keywords : Aplastic anemia, platelet transfusion, ocular manifestations

## Introduction :

Aplastic anaemia is a rare haemopoietic stem-cell disorder that results in pancytopenia and hypocellular bone marrow. Although most cases are acquired, there are unusual inherited forms. The pathophysiology of aplastic anemia is believed to be immune-mediated, with active destruction of blood-forming cells by lymphocytes. Environmental exposures, such as to drugs, viruses, and toxins, are thought to trigger the aberrant immune response in some patients, but most cases are classified as idiopathic.[1]

Aplastic anaemia is a life threatening condition. It usually presents with anaemia, bleeding and infection. Ocular findings are manifestations of preexisting anemia. The ocular findings include cotton wool spots, nerve fibre layer or preretinal haemorrhages, vitreous haemorrhages and optic disc oedema.[2]

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Aplastic anemia can be effectively treated by stem-cell transplantation or immunosuppressive therapy. Antithymocyte globulin and cyclosporine restore hematopoiesis in approximately two thirds of patients. Supportive care includes transfusion of RBCs and platelets.[3]

We are reporting a case of aplastic anemia which presented with the ocular findings following platelet transfusion.

## Case report:

A 60 yr old elderly male diagnosed with acquired aplastic anemia, presented with history of sudden diminution of vision in both eyes of 1 week duration, worse in left eye. He had a prior history of fatigability, breathlessness on exertion with fever 2 months back. There was no preceding history of overt bleeding manifestations, trauma, or exposure to noxious chemicals or irradiation. He was a diabetic on treatment. A relevant family history was absent. He had received platelet transfusion a week before he noticed the drop in vision.

On clinical examination, he was moderately built and appeared pale. His vital parameters were normal except that peripheral pulses were weak. Visual acuity was 6/18 in right eye and 6/60 in left eye. Anterior segment findings were unremarkable and pupillary reflex was normal. Intraocular pressure by applanation tonometry was normal. Both eyes fundoscopy showed blurred disc margins with slightly tortuous vessels, extensive

superficial, deep blotchy and preretinal hemorrhages in the posterior pole with cotton wool spots and macular edema worse in left eye. Optical coherence tomography showed an incomplete PVD in both eyes with subretinal fluid worse in left eye.

Blood investigations revealed low hemoglobin levels at 6.5g%, low white cell count of 2300cells/$mm^3$ and platelets 17000cells/mm3. Serum glucose, electrolytes and urea, folate and vitamin $B_{12}$ were normal. Screening for blood borne viruses was negative. Trephine bone marrow biopsy had revealed an aplastic marrow. He was subsequently started on cyclosporine. He had received platelets and packed cell transfusion twice. Following the third platelet transfusion patient had noticed a sudden drop in vision.

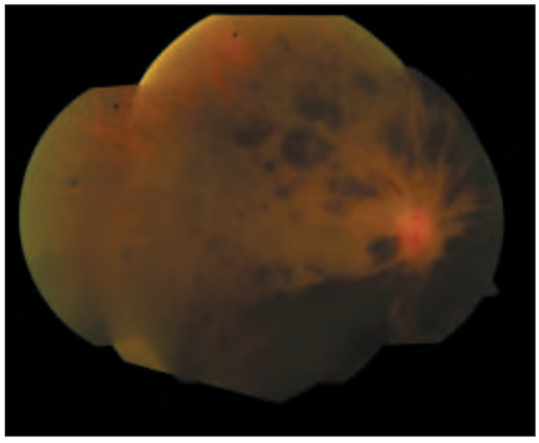

Fig 1 (a): RE showing hemorrhages, cotton wool spots and macular edema

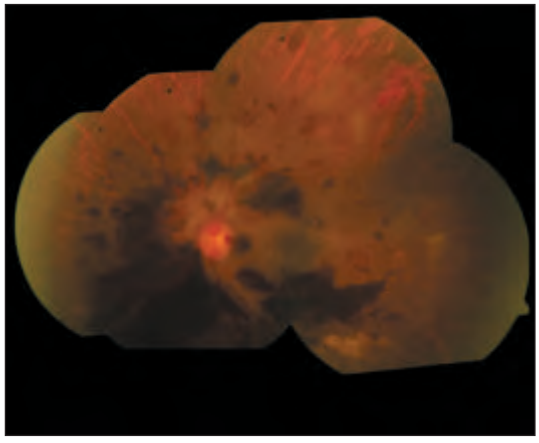

Fig 1 (b): LE showing hemorrhages, cotton wool spots, macular edema with exudates

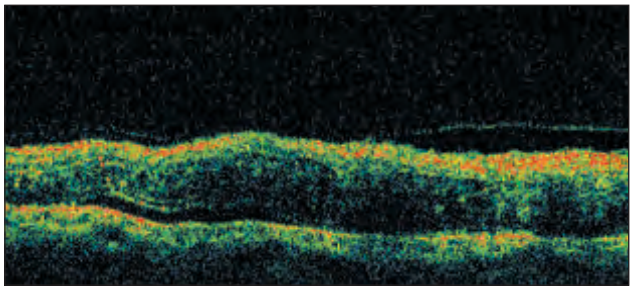

Fig 2(a): RE OCT showing incomplete PVD and retinal thickening

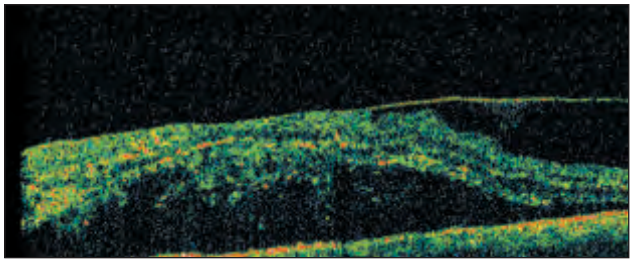

Fig 2(b): LE OCT showing incomplete PVD and subretinal fluid

Discussion :

Aplastic anaemia is a life threatening condition. Ocular findings are a manifestation of anemic retinopathy. It is believed that anaemia causes diminished capillary oxygenation, which increases the vessel wall permeability resulting in extravasation of blood products[4]. A direct correlation between the degree of anaemia and the severity of the retinopathy has been reported.

Mansour et al have shown that 78% of cases of aplastic anemia exhibit ophthalmic manifestations. Typical ophthalmic manifestations in aplastic anemia include eyelid hematoma, subconjunctival hemorrhage, cotton wool spots, retinal nerve fiber layer hemorrhage, Roth's spots, pre-retinal hemorrhage, vitreous hemorrhage, and disc edema[5].

A case of aplastic anemia simulating central retinal vein occlusion has also been reported. In our case disc edema and vessel tortousity was not much as in central vein occlusion. Some patients with aplastic anaemia have been reported to have pseudotumour cerebi. Papilloedema associated with aplastic anaemia has been proposed to be due to increased intracranial pressure from anaemia-induced cerebral hypoxia[7]. Our case did not have any signs of raised intracranial pressure[6].

Our patient developed the visual loss and findings of retinopathy following an episode of platelet transfusion. Visual loss in our case was due to macular edema. To the best of our knowledge, similar presentation following platelet transfusion in a case of aplastic anemia has not been reported. Similar ocular manifestations of blurred vision, retinal edema and hemorrhages were reported 10 days after hypotensive and anticoagulant treatment and blood transfusion in a young female patient diagnosed as HELLP syndrome[7]. Possibility of hemorrhagic retinopathy due to vascular hyperpermeability cannot be ruled out in our case. Thus the treating clinician should anticipate hemorrhagic retinopathy anytime during the course of treatment of aplastic anemia.

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