Restoration of sight through ischemic treatment.

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Abstract

Posterior ischemic optic neuropathy (PION) is characterised by severe acute painless unilateral or bilateral loss of vision caused by ischemic damage to the retro bulbar optic nerve. It differs from anterior ischemic optic neuropathy (AION) in that it lacks the oedema of the optic nerve that characterises the more common condition. The blood supply to the retro bulbar optic nerve is distinct from the blood supply to the optic nerve head and is derived primarily from the capillary plexus.

Keywords: Ischemic treatment, Capillary plexus.

Introduction

The optic nerve's posterior segment is divided into three sections: intraorbital, intracanalicular, and intracranial. Because the blood supply to these various sections comes from a variety of branches and arterial sources, the pathology of PION does not have to be limited to a single artery or location. The pial plexus provides a peripheral centripetal blood supply, and branches of the central retinal artery provide an axial centrifugal blood supply to the intraorbital segment. The intracanalicular portion is only supported by the peripheral centripetal system via ophthalmic artery branches, while the intracranial portion is only supported by a pial vascular plexus via variable arterial branches PION causes can be divided into two categories: arteritis (associated with giant cell or other vasculatures) and non-arteritis. Non-arteritis PION is further subdivided into spontaneous cases associated with typical vasculopathic risk factors (hypertension, hyperlipidaemia, diabetes, and smoking) and cases clearly associated with peri-procedural hypo perfusion. PION is most commonly associated with spinal surgery. Any procedure that causes adequate optic nerve hypoperfusion, such as intentional hypotension, intraoperative blood loss, or prone positioning, can result in retrobulbar optic nerve damage. Although PION caused by hemodialysis is uncommon, it has previously been reported in a patient with hypertension, chronic kidney failure, and gout. This patient had anaemia and periprocedural hypotension. The results of the computer tomography and magnetic resonance imaging magnetic resonance angiography of the head were normal.

While there have been reports of successful cases treated with hyperbaric oxygen therapy and steroids, there is no established treatment protocol, and the prognosis for vision recovery in patients with peri-procedural PION is poor.

Anemia in chronic kidney disease, both on and off dialysis, [1] the effects of concomitant myelo suppressive chemotherapy, and the reduction of allogeneic RBC transfusions in patients undergoing elective, non-cardiac, non-vascular surgery are all indications for its use. The primary goal of treating anaemia with erythropoiesis-stimulating agent therapy in the dialysis population is to prevent anaemia symptoms such as fatigue, dyspnea, and decreased exercise tolerance, as well as to reduce the need for blood transfusions. When used according to established protocols, erythropoietin is generally well tolerated. The risks of use include worsening hypertension and thromboembolic events, especially when high haemoglobin targets are set [2].

Ischemic events of the posterior optic nerve are caused by decreased perfusion to the pial vessels that supply the optic nerve's posterior portion. These vessels are more sensitive to low oxygen levels. Because pial vessels are less affected by anatomical constriction, ischemic events take longer to cause permanent damage, and prompt treatment in restoring perfusion by controlling blood pressure and increasing red blood cells is thought to affect outcome. EPO's neuroprotective effects should be considered. EPO has anti-inflammatory properties due to the release of nitric oxide, which opposes the action of tumour necrosis factor-alpha, and the release of myeloperoxidase and glutathione peroxidase, which counteract the effects of oxygen free radicals. Because PION is uncommon [3], other causes of transient vision loss, such as migraine or bilateral occipital lobe ischemia, should be considered intense optic neuropathy in patients beyond 50 years old and the second-most normal reason for extreme vision misfortune in grown-ups with glaucoma.

References

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