Reversible Cortical Blindness Following Successful Surgical Repair Of Two Stab Wounds In The Heart

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Abstract
This report describes a case of cortical blindness that followed successful surgical repair of two stab wounds in the heart in a 29-year old Libyan man. The patient presented in a state of pre cardiac arrest (shock and low cardiac output status), following multiple chest stab wounds. Chest tube was immediately inserted. Surgery was urgently performed suturing the two wounds; in the root of the aorta and in the left ventricle, and haemostasis was secured. Cardiac arrest was successfully prevented. The patient recovered smoothly, but 24 hours later he declared total blindness. Ophtalmic and neurological examinations and investigations that included fundoscopy, Electroencephalograms (EEGs) and Computed Tomography Scans revealed no abnormalities, apart from absence of alpha waves in the EEGs. We diagnosed the case as cortical blindness and continued caring for the patient conservatively. Three days later, the patient regained his vision gradually and was discharged on the 7th postoperative day without any remarks.

Key words: Cortical blindness, Cardiac Arrest, Resuscitation, cerebral anoxia.

Introduction

Cortical blindness is a total or partial loss of vision in normal-appearing eyes caused by damage of the geniculocalcarine pathways [1]. The incidence and etiology of cortical blindness seems to be changing with the development of effective means of cardiopulmonary resuscitation and better understanding of neurovascular physiopathology [1,2,3]. The precise nature of the pathogenesis of this condition has not yet been understood. However, cerebrovascular disease is the most common cause, followed by cardiac surgery, cardiac arrest, head injury, pre-eclampsia and cerebral angiography [1,4,5,6]. Temporary blindness after coronary angiography has also been reported by many authors [7,8,9]. Other reported causes of cortical blindness are Guillina Barr syndrome [10], Reversible posterior leucoencephalopathy syndrome [11]. Blue rubber bleb naevus syndrome [12], and injection of bleeding gastric varices with cyanoacrylate glue [13]. It seems that the key to understanding cortical blindness following cardiac arrest is understanding the associated cerebral iscahemia [1,2,3]. Cardiac arrest usually leads to generalised brain anoxia or to focal neurological abnormalities, resulting in a variable spectrum of diffuse or multifocal cerebral atrophy due to neuronal loss and cortical and subcortical gliosis or even severe cortical damage [2-3]. The global ischemic anoxia results in diffuse hypometabolism with preferential localisation of the metabolic alterations in the parieto-occipital cortex [4]. Although most patients recover from cortical blindness, the mechanisms of this recovery are poorly understood1. The posterior cerebral artery supplies the occipital lobe, and also seems to play an important role. All conditions that cause vascular impairment to the occipital cortexes may lead to cortical blindness [1]. Cortical blindness has not been described in the medical literature to follow surgical repair of stab wounds in the heart. In this report, we describe a 29-year old male patient who experienced compete loss of vision following successful surgical repair of stab wounds in the heart. To the best of our knowledge, this is the first case describing the association between cortical blindness and surgical repair of stab wound in the heart.

Case report

Cortical blindness followed successful surgical repair of two stab wounds in the heart in a 29-year old Libyan man, who was brought in a state of shock to the accident and emergency unit of our hospital following a fight. The patient was semi comatosed, with cold extremities, and with capillary filling more than 4 seconds. He was in sinus bradycardia with weakening pulse of 45 per minute, and his blood pressure was 70/50 mmHg. The pupil was dilated and sluggishly-reacting to light. Monitor ECG showed sinus bradycardia. Chest X Ray showed massive left haemothorax with left lung collapse. The patient didn't arrest but was in a pre arrest condition with low cardiac output status and progressively collapsing pulse and dropping blood pressure. We estimated a blood loss from the clothes of at least 2 litres. Cross matching for 5 units of whole blood was ordered, while left chest tube was immediately inserted with 850 ml of fresh arterial blood gushed out, and continued to drain at an estimated rate of more than 200 ml per hour into the water-seal reservoir. Urgent left anterior thoracotomy was performed under general anaesthesia. The heart was reached via pericariotomy. During this time, IV fluid infusion therapy was instituted with 1000 ml 5% dextrose saline and 1000 ml Ringers solution, and adrenaline 3 mg was given in the IV. Blood samples were sent urgently to the laboratory. Two profusely bleeding wounds were determined; one was at the ascending aortic root just above the aortic annulus, and the second was on the anterior surface of the left ventricle; a few millimetres to the left side of the proximal left anterior descending artery. The bleeding was completely stopped after repairing the 2 wounds with 4/0 pledgeted polypropylene sutures. By then, the cross matched blood arrived to theatre and was immediately given. Five units of whole blood were given over 24 hours, and were controlled by multiple Complete blood counts. The patient didn't arrest during surgery, but had an episode to sinus tachycardia of 180 minutes per minute that didn't turn into ventricular fibrillation, and thus no DC shock was given. The patient recovered smoothly in the intensive care unit, and was discharged to the surgical

ward after 24 hours with stable haemodynamics. He realised in the ward that he lost his vision completely. Specialised Ophtalmic and neurological examinations revealed no abnormalities. An electroencephalogram (EEG) was marked with the absence of alpha waves. Computed Tomography Scans showed no abnormalities. Visual evoked potential (VEP) was not performed as it was unavailable. Accordingly, the diagnosis of cortical blindness was established. The patient was reassured and treated conservatively. Three days later, he started to see again and eventually regained his vision completely. Repeat EEGs revealed regaining of alpha waves at this point. He was discharged with normal vision and in good condition on the 7th day of admission. One week, one month, and 3 month follow-up revealed no abnormalities.

Discussion

Cortical blindness is not an uncommon clinical condition, but has not received due attention by the medical community. Cardiac arrest usually leads to global brain anoxia and to lack of cortical functioning or to more focal neurological abnormalities, which may lead to cortical blindness, resulting in a variable spectrum of diffuse or multi-focal cerebral atrophy due to neuronal loss and cortical and subcortical gliosis or even severe cortical damage [2-3]. It has been shown that the prognosis of cortical blindness was best in patients under the age of 40 years, in those without a history of hypertension or diabetes mellitus, and in those without associated cognitive, language, or memory impairments [1]. It has been shown that EEGs, which demonstrate alpha waves absence during the episodes of cortical blindness, are more specific than VEP and CT scans for the diagnosis. Some reports have shown that when bi-occipital abnormalities are demonstrated in the CT scans, a poor prognosis follows [1,2].

This report describes a typical reversible cortical blindness with sudden loss of vision, absence of alpha waves in the EEGs and complete regaining of vision. What is unique in our case is the association with the surgical repair of stab wound in the heart and the unreported cardiac arrest during the course of resuscitation; before or during the surgical intervention. It seems that there were episodes of cerebral anoxia during the state of shock following the profuse bleeding and hypovolaemia that was caused by the multiple stab wounds in the chest. Lack of cortical functioning would have caused this cortical blindness despite the un occurrence of cardiac arrest. The quick chest tube insertion improved the compromised respiratory function, and prevented cardiac tamponde, which provided an immediate improvement of circulatory functions and haemodynamics despite the continuing bleeding. The immediately given IV fluids and adrenaline have helped to support the failing circulation and correct the hypovolaemic shock. This prevented the cardiac arrest cascade at a proper pre cardiac arrest end point. Our quick and proper surgical intervention helped prevent the cardiac arrest and prevented undergoing progressive cortical and brain damage, and thus saved his vision and life. Swift and proper patient transportation to advanced centres may prevent cardiac arrest in pre cardiac arrest cases. Applying cardiac resuscitation guidelines is mandatory to stop pre cardiac arrest cases from developing cardiac arrest and to increase the survival rates of cardiac arrest resuscitation. Sudden loss of vision may follow pre cardiac arrest cases and surgical intervention, even if cardiac arrest has not occurred, should lead to the suspicion of cortical blindness particularly when no gross ophthalmic and neurological abnormalities could be demonstrated. Increased awareness of healthcare providers about this phenomenon will ensure avoiding confusing diagnosis, un-necessary investigations and improper treatment for cortical blindness.

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