CASE REPORT

REVERSIBLE VISION LOSS IN A PATIENT WITH INFECTIVE ENDOCARDITIS

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ABSTRACT

Visual complications in patients with infective endocarditis include central retinal artery or vein occlusion, corneal ulcers, endophthalmitis, sub-retinal abscess, papillitis, optic neuritis and cortical blindness due to septic micro-embolization. Although there is no epidemiological data available on the visual complications of infective endocarditis, however, many case reports reveal that these are common and could be the first presentation of infective endocarditis.[1]

We presented a case of a young gentleman, an intravenous drug abuser who presented with fever and bilateral, painless, vision loss. Fundoscopic examination revealed Roth spots. Echocardiography showed vegetation on anterior mitral leaflet. Blood cultures showed growth of Staphylococcus Aureus. His MRI brain showed small areas of diffusion restriction in bilateral parieto-occipital regions.The patient receivedbroad spectrum antibiotics for native valve endocarditis. His vision improved after 18 hours of hospital stay.

We emphasize the importance of recognizingthe visual complications in patients with infective endocarditis. Early diagnosis leads to early treatment and reversibility of this focal deficit.

KEYWORDS: Vision, Endocarditis, Early diagnosis

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INTRODUCTION

The most common organisms causing Infective endocarditisis Staphylococcus Aureus. Incidence of IE in intravenous drug abusers is 2 to 4 cases per 1000 years of intravenous drug abuse.It is more common in males (3:1)[3,4] Another review of 105 patients of infective endocarditis in intravenous drug abuser at a tertiary care hospital in Vancouver, revealed that 86% involve right side, while 14% had left sided involvement.[5] Septic embolization is a common complication of IE.[1]Visual complications due to micro-embolization and staphylococcal bacteremia are common in patients with infective endocarditis.[2]We present a case of young, intravenous drug abuser with vision loss. In our case, the likely cause of vision impairment is microembolization of bacteria in the basilar artery, leading to vasospasm of posterior circulation.Early reversibility is due to appropriate administration of antibiotics.

CASE REPORT

A case of 24 year old gentleman, known intravenous drug abuser, presented with the sudden onset of bilateral visual loss for few hours. No associated ocular symptoms of redness, lacrimation, peri-orbital pain, headache, diplopia or proptosis were found. There was no history of visual impairment in the past. He was having high grade, intermittent fever for 3 weeks. Fever was associated with joints pain and there was also significant pain at the tips of his fingers. He denied any history of rash or oral ulcers.

On examination, his temperature was 100 degree Fahrenheit, heart rate 110 beats/minute & regular, Respiratory rate 18 breaths/minute, Blood pressure: 110/70 mm Hg. His general physical examination revealed Osler's nodes(tender lesions found on

finger pulps). Ophthalmological examination showed visual acuity of light perception only. Pupils were 3 mm in size, bilaterally equal and reactive to light.There were few pale-centered hemorrhagic areas seen on the retina (Roth spots). **(Figure 1)** Fundi and maculae were normal. Neurological assessment was unremarkable. On auscultation of heart, there was early systolic murmur of grade 2/6 on tricuspid area.

His lab workup showed hemoglobin of 12.8 mg/dl, Total leukocyte count of 26,000 /cu mm(4-11), Platelets were 150,000/ cu mm(150-400,000), Urea: 53mmol/L(<40), Creatinine: 1.59 mg/dl, SGPT: 115units/liter (up to40 units/liter),Gamma GT:226 units/liter (11-50), Alkaline phosphatase:167 IU/liter (39-117), ESR:79 mm/hour (1-12), CRP:183 mg/liter (5), Urine detailed report revealed Red cells:10 mg/dl, no casts.Ultrasound abdomen was normal. Chest x ray showed multiple heterogenous opacities propagating alikely possibility of septic emboli. MRI brain showed focal areas of hyperintensities seen in the left occipital lobe and right centrum semiovale location on diffusion weighted sequences, few hyperintense foci appreciated on flair images seen in the right parieto-occipital lobe. Likely possibility is of multiple ischemic areas due to cardio-embolization **(figure 2)**. Echocardiography showed intracardiac vegetation seen attached to anterior mitral leaflet (10mm), mild Mitral regurgitation and Tricuspid regurgitation was also noted. Three sets of blood cultures were sent and treatment started empirically for native valve endocarditis. His blood culture showed growth of Staphylococcus Aureus.HIV serology's, hepatitis profile and fungal cultures were negative. Patient was managed on broad spectrum antibiotics by the team of general physician, cardiologist, Infectious disease expert, pulmonologist and neurologist. Cardiothoracic surgery input was taken and they advise conservative management. His vision improved after 18 hours of hospital stay, although his fever remained for another week. Antibiotics were continued for total of 4 weeks. Follow up echocardiography revealed reduction in size of vegetation from 10 mm to 6 mm.

DISCUSSION

Sudden simultaneous, bilateral visual loss is rare and has a restricted differential diagnosis which includes bilateral occipital lobe infarction or hemorrhage, pituitary apoplexy, bilateral sequential ischemic optic neuropathy, bilateral acute glaucoma, bilateral optic neuritis, bilateral retinal detachments, bilateral central retinal artery and vein occlusions. In our case, examination findings of normal bilaterally equal and reactive pupils, normal optic discs, with the normal angiography of retinal vessels, exclude all other possibilities of bilateral visual loss and localized the lesion at visual cortex.

Septic embolization is a common complication of infective endocarditis, embolic events occur in as many as 50% of all patients.Embolic stroke can result from embolization of an artery in the central circulation from a variety of sources. In infective endocarditis, besides clot, fibrin, and pieces of atheromatous plaque, emboli include bacterial clumps. Superficial branches of cerebral and cerebellar arteries are the most frequent targets of emboli. Most emboli lodge in the middle cerebral artery distribution because 80% of the blood carried by the large neck arteries flow through the middle cerebral arteries, this is contrary to our case.

The neurological outcome from strokecaused by septic embolization depends not only on the occluded vascular territory but also on the ability of the embolus to cause vasospasm by acting as a vascular irritant. Septic emboli in infective endocarditis are very irritant to the vessel and at times can cause necrotic arteritis. The vasospasm can occur in the vascular segment where the embolus lodges or can involve the entire arterial tree. Vasospasm tends to occur in younger patients, probably because the vessels are more pliable and less atherosclerotic.Degree of damage by embolic occlusion depends on the time emboli remained in cerebral circulation and richness of collateral circulation. Although MRI brain was done after 24 hours in our case, it showed multiple areas of diffusion restriction in parieto-occipital areas and centrum semiovale, with the impression of cerebral septic emboli. In our case, likely possibility of vision impairment is micro embolization of bacteria to basilar artery leading to vasospasm of posterior circulation. As mentioned earlier, in our case there was a delay in neuro imaging so MRA brain failed to show any vascular stenosis or occlusion. Our case clearly delineates the thought of cerebral embolic phenomenon and its reversibility due to appropriate and timely administration of antibiotics leading to reversal of vasospasm as proven in international collaboration on endocarditis. A prospective cohort study showed that the crude incidence of stroke in patients receiving appropriate antimicrobial therapy was 4.82/1,000 patient days in the first week of therapy and decreased to 1.71/1,000 patient days in the second week. This rate continued to decline with additional therapy[6].So our case emphasizes the importance of early identification and management of infective endocarditis patients with septic embolization to brain.

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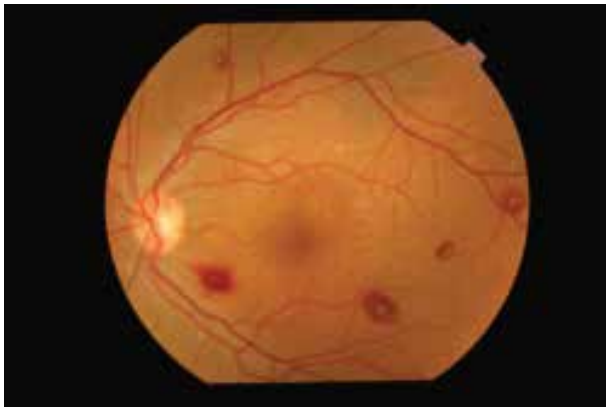
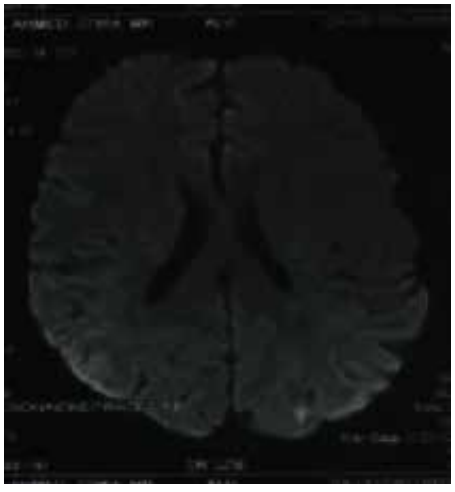
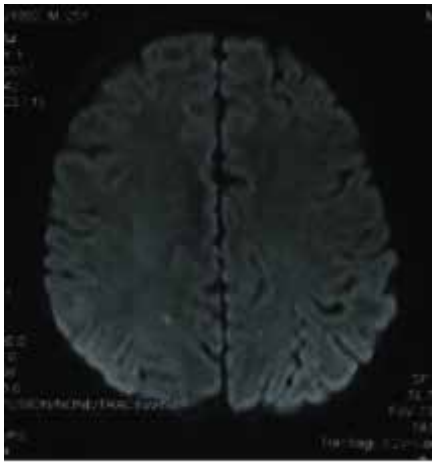


Figure 1: EYE FUNDUS SHOWING ROTH SPOTS



a. Left parieto-occipital area of diffusion restriction



b) Right centrum semiovale area of diffusion restriction

Figure 2(a&b): MRI BRIAN (DWI SEQUENCES)