

Progressive Visual Loss from Roth Spots in a Patient with Infective Endocarditis

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Abstract

Infective Endocarditis (IE) implies infection of the endocardial surface of the heart from the presence of microorganisms in the lesion. The variability in clinical presentation, heterogeneous patient population and microbiologic organisms makes the IE diagnosis challenging for clinicians.

Keywords: Endocarditis Roth spots; Multiple Emboli; Stroke

Case Presentation

A 60-year-old edentulous man with past medical history of hyperlipidemia, current smoker, and depression presented to the eye department with a 4-week history of progressive blurred vision. As per patient he saw a “white sheet” in front of his eyes worse in the right eye constantly. After his eye evaluation he was referred to the neurology clinic as he complained of having difficulty finding words to express him-self. He did report of having lost 10 lbs (4.5 kg) in weight during this period but no other complains such as fever, fatigue, sweating, chest pain, shortness of breath or any other neurological complains. There was no history of substance abuse including history of intravenous drug use.

Assessment

On general evaluation his Body Mass Index (BMI) was 26, had no clubbing or other skin manifestations such as splinter hemorrhages, petechiae (Osler nodes and Janeway lesions). He had no cardiac murmurs or extra heart sounds on auscultation. On abdominal examination there was no visceromegaly specially splenomegaly. His neurological examinations including his language were normal. His eye examination the fundus showed multiple, bilateral, white-centered retinal hemorrhages which were “Roth spots” (Figure 1).

Diagnosis

On initial work-up his hemoglobin was 9.5 g/dL, Mean Corpuscular

Volume 72.8 fl with white cell count 11.7x 10⁹/L. His sedimentation rate was 105 mm/h, and a C-reactive protein was 198 mg/L. His blood urea nitrogen was 9 mg/dL with a creatinine of 0.9 mg/dl, normal glomerular filtration rate greater than 60 mL/min/L, but urine analysis showed frank hematuria (4 Red Blood Cells/High Power Field). Head Magnetic Resonance Imaging (HMRI) demonstrated an acute infarct in the left frontal lobe on Diffusion Weighted Image (DWI) confirmed by Apparent Diffusion Coefficient (ADC) (Figures 2a and 2b). Abdominal Computerized Tomography (CT) scan showed presence of splenic and bilateral renal infarcts (Figure 2c). Blood cultures grew *Streptococcus oralis*. Trans-thoracic echocardiogram showed mitral regurgitation with

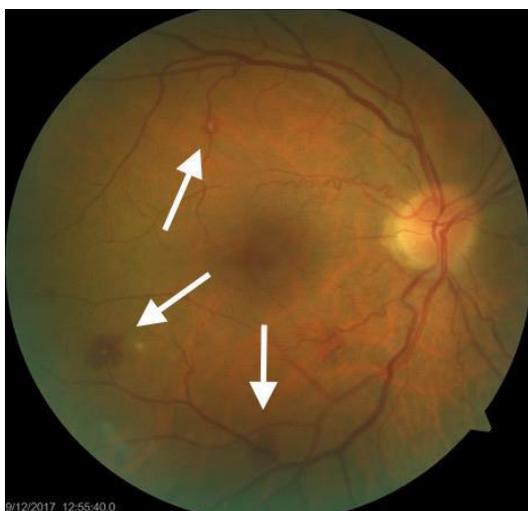


Figure 1: Roth spots.

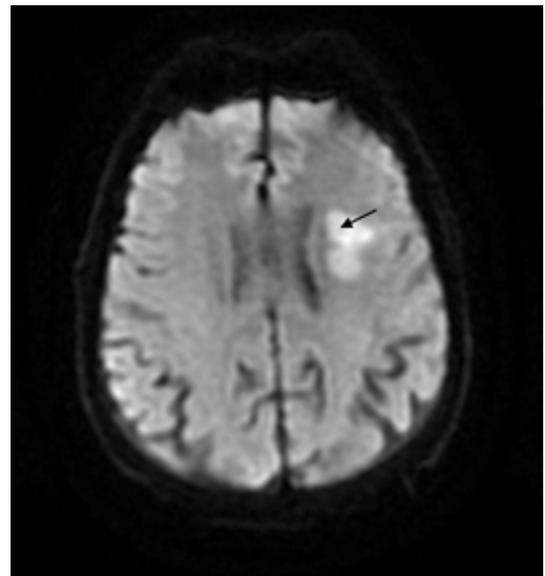


Figure 2a: Cerebral infarction (stroke) on Diffusion Weighted Image (DWI).

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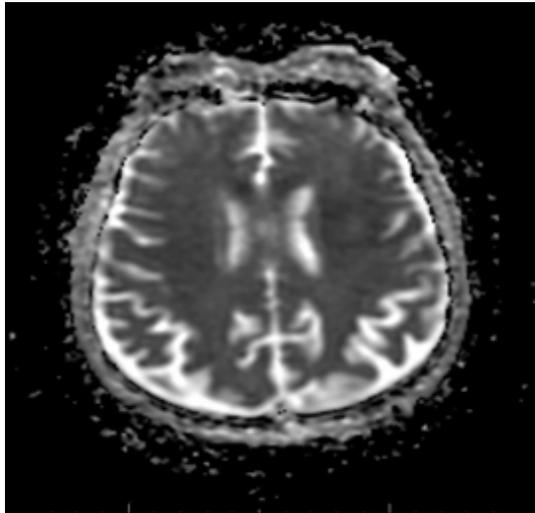


Figure 2b: Cerebral infarction (stroke) on Apparent Diffusion Coefficient (ADC).



Figure 2c: Renal and splenic infarcts.

medium sized mobile mitral valve vegetation on the left ventricular aspect of the anterior leaflet, the source of his generalized embolization (Figure 3).

Management

He was initially treated empirically with intravenous (IV) vancomycin and ceftriaxone. On receipt of culture and sensitivity report he was changed to IV ceftriaxone daily. He was discharged home a week later to continue his parenteral ceftriaxone daily for 9 weeks. When last seen in the clinic 3 months later he had no new neurological complaints, felt clinically improved with no growth on three separate blood cultures. He was also advised about the vital importance of dental hygiene and need for antibiotic prophylaxis when undergoing dental procedure/care. He is now awaiting mitral valve replacement surgery.

Discussion and Conclusion

This case of Infective Endocarditis (IE) according to the Duke criteria [1] is unique in several ways: First, he had no known history of valvular heart disease; second, was edentulous with no recent history of dental procedure [2]; third, his presenting symptom was mainly blurred



Figure 3: Vegetation on the mitral valve (mobile).

vision which affected his ability to drive and work; and finally, the organism responsible was *Streptococcus viridans*. IE is now a day more common in men than women [3], who are older (greater than 50 years of age) [4] due to rise in the frequency of degenerative valvular heart disease [5]. *Staphylococcus aureus* is the leading cause of bacteremia and IE. Mitral valve is the most commonly affected valve in 86% cases, followed by aortic 55%, tricuspid in 19.6%, and pulmonic 1.1% cases. This is due to degree of the mechanical stress exerted by the valve [6]. Splenic septic emboli are common during IE [7]. The risk of stroke is increased in patients with mitral than aortic valve involvement [8], especially if they are of a larger size (>1 cm) and mobile vegetation as in this patient [9]. Trans-esophageal echocardiogram (95% vs. 60-65%) [10]. Parenteral antibiotic therapy is preferred over oral antibiotic therapy, antibiotic treatment should be based on microbiologic etiological agent [11], and combination antibiotic therapy is advised in streptococcal infections [12]. Ceftriaxone was chosen as it has a high cure rate of 98%. Roth spots are named after Moritz Roth a pathologist at the University of Basel in 1872 [13]. They are characterized by retinal hemorrhages with a white center. The white center of the Roth spot corresponds to a platelet-fibrin thrombus at the site of the retinal capillaries rupture [14,15]. They are usually caused by immune complex mediated vasculitis. They can appear and disappear within hours. They occur in a wide variety of systemic conditions such as anemia, anoxia, leukemias, carbon monoxide poisoning, prolong intubation during anesthesia, human immunodeficiency virus retinopathies, diabetic and hypertensive retinopathies, pre-eclampsia and shaken baby syndrome; however, they are considered pathognomonic for Bacterial Endocarditis (BE). Thus, Roth spot is a morphological manifestation of retinal capillary rupture and a careful systemic evaluation is needed to elucidate the underlying cause. There is no specific treatment for Roth spot other than treat the underlying cause.

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