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Acute Embolic Ischemic Stroke as the Sole Presentation of Subacute Enterococcal Endocarditis

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Abstract Neurological complications are the most frequent extra cardiac complications of infective endocarditis (IE) dominated by the stroke with a high mortality [1]. These manifestations might be the first sign of IE in rare cases. Atrial fibrillation and infective endocarditis (IE) are two of the most common causes of cardio embolic strokes. The patients with infective endocarditis (IE) can present with variable symptoms ranging from vague complaints of fever and chills to any embolic complication. Duke's criteria is the most common criteria used to diagnose infective endocarditis. We present a case of an acute ischemic stroke in a young patient, which was the sole presentation of subacute infective endocarditis associated with Enterococcus faecalis, thus leading to delayed diagnosis and treatment.

Keywords: *subacute infective endocarditis, embolic stroke, cryptogenic stroke*

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1. Case Presentation

A 30-year-old white male with the past medical history of hepatitis C and IV drug abuse came in complaining of acute onset of drooping of the right side of the mouth, slurred speech and right arm weakness. He denied any headache, fever, chills, chest pain, palpitations and shortness of breath. He accepted that he used illicit drugs a week ago but denied use of smoke and alcohol. On examination, he was oriented in time, place and space. No murmur, gallops or clicks were appreciated on auscultation of heart. Neurological examination revealed facial droop on right side and slurred speech. Sensations were intact bilaterally and power was 4/5 in right upper extremity and 5/5 in rest of extremities. Finger to nose and heel to shin test were normal. Babinski sign was negative and reflexes were absent in ankles bilaterally. Rest of the examination was unremarkable.

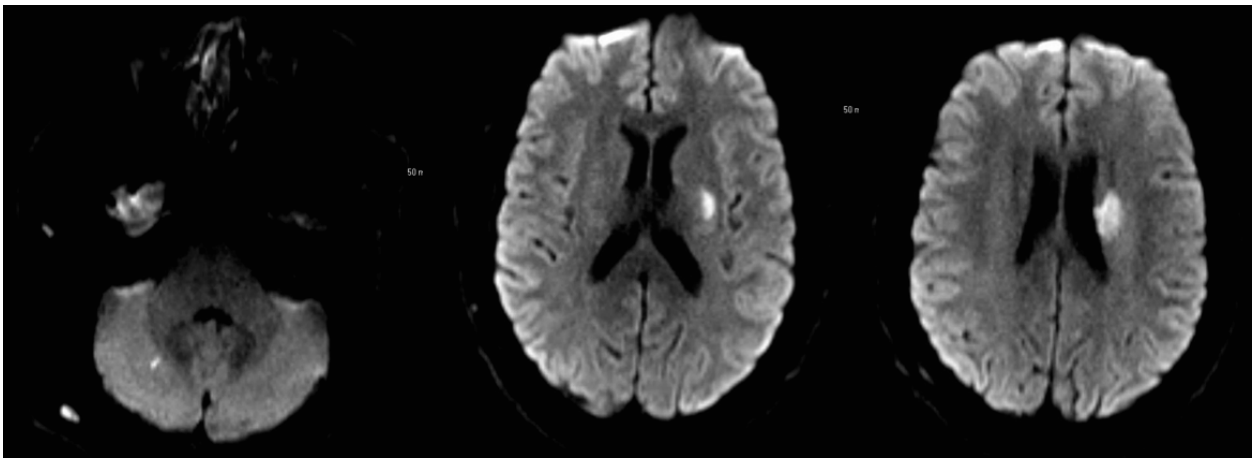

Figure 1. MRI brain with out contrast showed acute infarcts of the left cerebral deep white matter, basal ganglia and right cerebellum

All baseline investigations including urine drug screen and lipid profile were ordered which were unremarkable except WBC count that was 14000/cumm. Troponins were mildly elevated to 0.08, which did not go up on trending for next 24 hours. EKG done on admission showed normal sinus rhythm with no ST or T wave changes. CT

scan of head was ordered which did not show any intracranial pathology. He was admitted as an acute stroke and was given aspirin per rectally as he failed swallowing evaluation. TPA was not given as symptoms started more than 3.5 hours priors to presentation to emergency. MRI brain with out contrast showed acute infarcts of the left cerebral deep white matter, basal ganglia and right cerebellum (Figure 1) but MRA brain revealed patent cerebral circulation. Extensive work up was done to find the source of emboli. Carotid Doppler showed normal carotid arteries and venous dopplers of lower extremities did not show deep venous thrombosis. Homocysteine, ANA, anti cardiolipin antibodies were normal. Urine drug screen and HIV testing were normal too. Transthoracic echocardiogram was done which showed ejection fraction 60% and myxomatous changes of the mitral valve leaflets with mild prolapse of the anterior mitral leaflet. No vegetations were appreciated on TTE. He denied any fever or chills and his recurrent blood cultures were persistently negative.

His symptoms improved significantly in next few days with complete resolution of symptoms in 1 week. It was decided to discharge the patient with event recorder and perform Transesophageal echocardiogram (TEE) on outpatient basis. TEE was done in 2 weeks, which to our surprise revealed a vegetation (1cm x 0.4cm) attached to the mitral valve apparatus that was going in and out of the left atrium during systole (Figure 2). In addition, there was severe redundancy of the anterior mitral valve leaflet, which appeared to prolapse substantially. No shunting was appreciated on bubble study. Patient again denied any fever, chills, chest pain or shortness of breath. No signs of IE such as purpura, osler nodes, roth spots, janeway lesions or splinter hemorrhages were found on examination. He did not have any weakness in any of the extremities. He was admitted again and blood cultures were taken which came positive for Enterococcus faecalis group D endocarditis. He was started on ampicillin and gentamycin for 6 weeks. Repeat Echo after 2 months showed normal ejection fraction and moderate mitral regurgitation with no vegetations. Through out the course of his disease, he never developed any sign or symptoms of endocarditis.

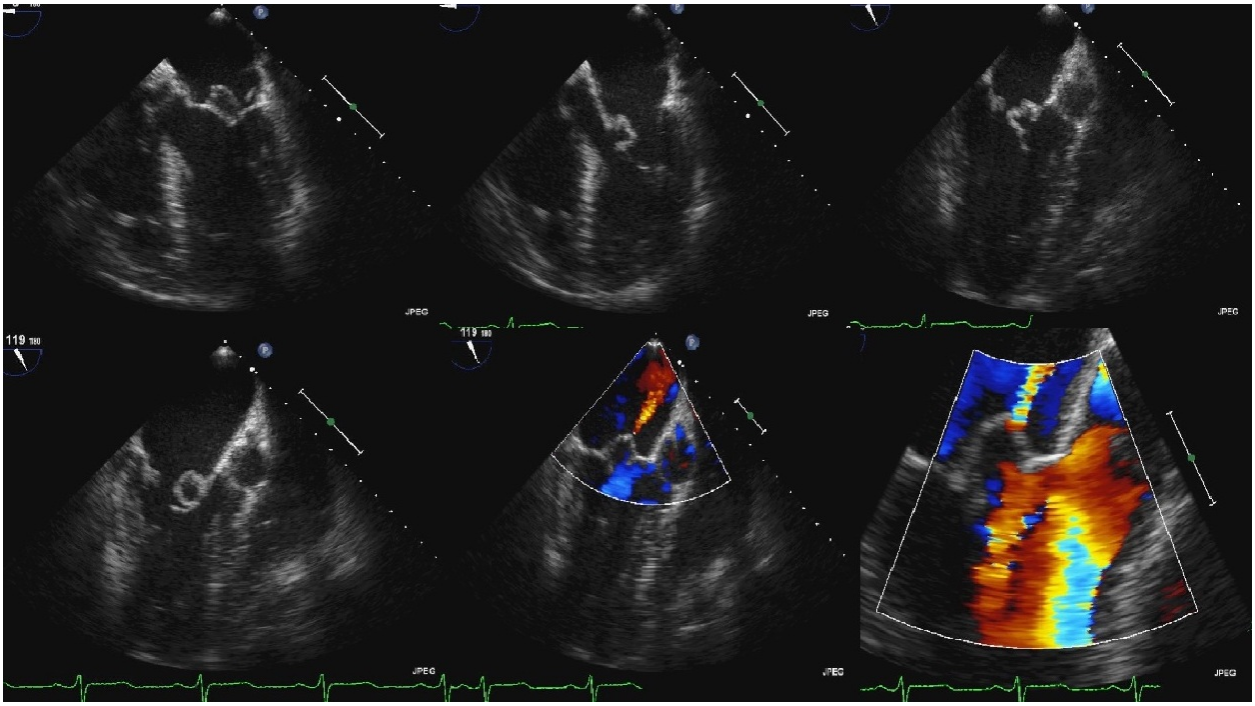

Figure 2. TEE showed a vegetation (1cm x 0.4cm) attached to the mitral valve apparatus that was going in and out of the left atrium during systole

2. Discussion

Enterococci, most often Enterococcus faecalis, cause 5%–20% of cases of infective endocarditis (IE). Enterococcal IE is usually a disease of older men, and the most frequent source of infection is the genitourinary tract. In cases of enterococcal IE, both normal and previously damaged valves can be involved. The disease most commonly presents in a subacute fashion over days or weeks. Variables such as advanced age, cardiac failure, and brain emboli have been recognized as risk factors for mortality.

In adults with infective endocarditis, neurological complications develop in 20 – 40% of patients [2]. The most frequent neurological complications include cerebral infarction, intracerebral hemorrhage, bacterial meningitis, and mycotic aneurysms [3,4]. Such neurologic sequel mainly affects the middle cerebral artery (>40%) [5]. Strokes in these patients can be subtle or even subclinical. Stroke is common in young patients with IE, often involving multiple vessels and is prone to hemorrhagic transformation. Stroke associated with endocarditis has significant morbidity and mortality rates, especially due to delay in diagnosis and multiple sites of brain embolization. Higher risk of embolism has also been found with Staphylococcal and fungal infections [6], vegetation size of >10 mm (measured by transoesophageal echocardiography), high mobility, elevated C-reactive protein, and associated embolic events; furthermore, mitral valve (or multivalvular)

involvement has been linked to an increased risk of embolism from IE [7,8].

The most popular diagnostic criteria used is the Dukes criteria. Major criteria is characterized by 2 or more blood cultures positive for typical organisms and echocardiographic finding suggestive of IE. The presence of two major criteria or one major criterion and three minor criteria or five minor criteria suggest IE. Research has shown that initiation of antibiotic treatment can reduce the rate of neurological complications [9] and the overall embolic risk, which further highlights the need for rapid diagnosis and treatment. As a result, the recommended practice at some institutions is to obtain the blood cultures and transthoracic echocardiogram (TTE) in all cases of stroke [10]. The mainstay of diagnosis of infective endocarditis is echocardiography and blood culture. The most popular diagnostic criteria used is the Dukes criteria. In general, TTE is the first test of choice in patients with suspicion of IE, though a negative TTE does nor rule out IE. It is reasonable to perform transesophageal echocardiogram (TEE) in patients with negative TTE and high suscpicion of IE such as persistent bacteremia and multiple minor criteria [11].

The recommended treatment for IE is ampicillin in combination with either gentamicin or ceftriaxone. E. faecalis endocarditis continues to be a very serious disease with considerable percentages of high-level gentamicin-resistant strains and high in-hospital mortality. The definitive antibiotic treatment should be based on results of culture and sensitivity. In general, antibiotics need to be continued for 4 to 6 weeks. Some indications for surgical treatment are fungal IE, severe valvular dysfunction, acute congestive heart failure, new conduction defects or arrhythmias, or persistent bacteremia after appropriate antibiotic therapy. IE is a contraindication for thrombolysis in acute ischemic stroke as it increases the hemorrhagic risk. However, the suspicion of IE is only rarely raised in the acute stroke and thrombolysis is often initiated, leading either to multifocal hemorrhage or thrombus resolution [12,13].

Our patient presented with acute stroke for which extensive work up was done which came back normal. He denied any fever or chills, which led to delayed diagnosis and treatment of IE. TEE was not done during hospital stay as he met only 1 minor Dukes criterion, thus reducing the likelihood of IE. Instead, it was decided that patient should get TEE and event recorder on outpatient basis. His outpatient TEE revealed vegetation on the mitral valve. Again patient was completely asymptomatic with no complaints of fever, chills, chest pain or shortness of breath. His blood cultures were positive for enterococcus for which he was treated with antibiotics for 6 weeks

3. Conclusion

1. Subacute infective endocarditis can present even without constitutional symptoms of fever and chills thus delaying diagnosis and management, which can lead to increase mortatlity and morbidity.

2. IE is an important cause of ischemic stroke especially in young adults with risk factors. A high degree of suspicion is required for its early diagnosis and treatment.

3. Modified Dukes criteria is a good tool for diagnosing IE but patients not meeting the dukes criteria can still present with IE. After normal TTE, TEE for the evaluation of cryptogenic stroke might be performed in such patients who have one or two risk factors of infective endocarditis.

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