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A CASE OF INFECTIVE ENDOCARDITIS COMPLICATED BY ACUTE ISCHEMIC STROKE

Medicine

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ABSTRACT

Infective endocarditis is an infectious and inflammatory process involving endothelial lining of heart structures and valves. Cerebrovascular complications (CVCs) frequently occur in patients who are in the active stage of infective endocarditis (IE), and result from cerebral septic embolization of an endocardial vegetation. Acute stroke due to septic emboli is a particularly dreaded complication , with a frequency of 25-35%. Here we present a case of 32 year old male patient, who comes to the ER with high grade fever and palpitations since 9 days. On examination we found hyperdynamic impulse with decrescendo type systolic murmur at mitral area and we decided to do a blood workup and also requested a 2D ECHO. Blood culture and 2D ECHO showed different species of streptococci and mitral regurgitation respectively. Based on the investigations we started the patient on antibiotics, However, on the day 7 of treatment, patient developed slurring of speech and hemiparesis followed by motor aphasia. We sent the patient for brain MRI that showed acute infarct in left central semioval, left corona radiata and left perisylvian region. Acute ischemic stroke is the complication of the infective endocarditis and we started tpA along with intravenous antibiotics after which he experienced significant clinical improvement in few days.

KEYWORDS

Infective Endocarditis, Acute Ischemic Stroke, Endocardial Vegetation, Septic Embolism

INTRODUCTION

Infective endocarditis is an infectious and inflammatory process involving endothelial lining of heart structures and valves. It is characterized by high morbidity and mortality [1].This infection is usually caused by bacteria entering the blood stream and infecting the heart. The bacteria originates from the mouth, skin, intestines, the respiratory system and urinary tract. When such a infection is caused by bacteria, it is called bacterial endocarditis. In rare cases, it is also caused by fungi or other microorganisms. Most frequently encountered organisms include streptococci, enterococci, staphylococci, HACEK organisms (Hemophilus parainfluenza, Hemophilus aphrophilus, Actinobacillus actinomycetemcomitans, Cardiobacterium hominis, Eikenella species, and Kingella species)[1]. The risk of IE is high when certain medical procedures are performed that allow bacterial access to the bloodstream like invasive dental procedure or insertion of catheters or needles. However, these procedures do not put healthy people at risk. At risk here are the patients who have artificial heart valves, congenital heart disease, damaged heart valves, history of endocarditis, history of illegal drug use, hypertrophic cardiomyopathy or mitral valve prolapse. Mitral valve is the most frequently involved. Tricuspid valve endocarditis is associated with IV drug abuse-organisms include S. aureus, Pseudomonas and Candida. Acute infections occur on previously normal valves and are rapid in onset with large vegetations. Subacute infections occur on congenitally abnormal or diseased valves and are gradual in onset with smaller vegetations. Infective endocarditis is a serious condition that requires prompt medical treatment. Complications include Congestive Heart Failure, peri annular abscess, systemic embolization, splenic abscess, neurological manifestations , valve dysfunction, mycotic aneurysms[2-3]. Stroke is a frequently encountered complication in patients with infective endocarditis secondary to septic embolization.

Case Report

A 32 year old male patient presented with the chief complain of high grade, intermittent fever(104F) with chills and rigors since 9 days, associated with palpitation and significant weight loss around 10kg and anorexia. There is no diurnal variation. On arrival, his mental status was intact, was febrile and the temperature measured was 105 F, hypotensive with the blood pressure of 102/70mmHg. Heart rate is tachycardic at 100 beats per minute and tachypnoeic at 28 breaths per minute. On examination, palpation revealed a hyperdynamic impulse at the apex. On auscultation, systolic murmur was heard over the mitral area which was decrescendo in pattern. He was admitted and aggressively fluid resuscitated and started on anti-pyretics and intravenous 2gmceftriaxone OD and 60mg gentamycin TDS empirically. Her blood pressure stabilized and multiple sets of blood cultures from different sites grew Streptococcus mitis group of Streptococcus viridans. Leucocyte counts were 18000, ESR was increased, and CRP was positive.2D ECHO showed mitral regurgitation secondary to AML prolapse with rupture of chorda tendinea consistent with EI. After 5 days of antibiotic therapy patient become afebrile. But after 7 days of admission, the patient developed slurring of speech with right sided hemiparesis which later progressed to motor aphasia. On examination, the power of muscle was 2/5 and DTR +2/+2 and plantar extensor response on right side. Pupils were normal bilaterally and reactive to light. Sensory and cerebellar signs were intact. MRI of Brain was ordered, and it showed acute infarct in left central semioval, left corona radiate and left perisylvian region. tpA was started immediately along with intravenous antibiotics and he experienced significant clinical improvement after few days. He was discharged on a single antiplatelet therapy after the completion of a 4 week course of IV antibiotics. On discharge, the patient was afebrile.

TABLE 1	
Hb	9.4
WBC	18000
Platelet	3,30,000
ESR	108
CRP	6.9
Serum Creatinine	0.92
Urea	22
K+	4.3
Na+	132
SGPT	30
P-ANCA,C-ANCA	Neg.
24 hour urinary examination	328mg
USG ABD/KUB	Neg
ECG	NSR

DISCUSSION

Acute ischemic stroke is a dreaded complication of Infective Endocarditis. Stroke is the initial presenting sign in 5-15% of the patients with IE. It is caused by cardioembolic cause. Arterial embolism, one half of which precede the diagnosis of endocarditis are clinically apparent in up to 50% of patients. Endocarditis caused by mobile vegetation of >10mm in diameter and infection involving anterior leaflet, mitral valve and antiphospholipid antibodies are independently associated with increased risk of embolization. According to studies, cerebrovascular emboli presenting as stroke or encephalopathy complicate 25-35% of cases of IE.

The frequency of stroke is 10 per 1000 patients-days during the week prior to diagnosis; subsequent fall in frequency to 4.8 and 1.7 per 1000 patients-days during the first and second weeks of effective anti-microbial therapy, respectively. Only 3% stroke occur after one week of effective therapy. Immediate treatment with tpA within 4.5 hours of onset of symptoms of ischemic stroke is proven to be beneficial in the patient. However sometimes it causes haemorrhagic conversion in patients with stroke in such patients. Here, mechanical thrombectomy is the option. Other neurological complication includes aseptic or purulent meningitis. ICH due to haemorrhagic infarct or rupture

mycotic aneurysms and rarely seizures. Prevention of systemic embolization is an important aspect in the treatment of IE. Immediate initiation of appropriate antibiotics decreases the risk of septic embolization to the brain. Prior daily antiplatelet therapy and statin therapy before diagnosis of IE may prevent septic embolization.

Table 2

FEATURES	FREQUENCY, %
fever	65-95
Chills and sweat	40-72
anorexia, weight loss, malaise	30-55
myalgias, arthralgias	15-30
back pain	7-14
heart murmur	80-85
new/worsened regurgitant murmur	20-50
arterial emboli	20-50
splenomegaly	20-50
clubbing	10-30
neurologic manifestations	20-40
peripheral manifestations (osler's nodes, subungual hemorrhages, janeway lesions, roth's spots)	5-15
petechiae	10-45
Laboratory manifestations	
ANEMIA	70-95
leukocytosis	20-30
microscopic hematuria	35-55
elevated erythrocyte sedimentation rate	60-90
elevated c-reactive protein	>90
rheumatoid factor	60
circulating immune complexes	65-100
Decreased serum complement	5-55

CONCLUSION

Infective endocarditis is a life-threatening cause of acute ischemic stroke. Prevention of systemic embolization is an important aspect in the treatment of IE. Immediate initiation of appropriate antibiotics decreases the risk of septic embolization to the brain. Immediate treatment with tpA within 4.5 hours of onset of symptoms of ischemic stroke is proven to be beneficial in the patient. Prior daily antiplatelet therapy and statin therapy before diagnosis of IE may prevent septic embolization.

Some points need to keep in mind while treating infective endocarditis patient :-

1. IV antibiotics given according to growth of microbial in blood culture.
2. Antithrombotic therapy
3. Surgical management reserved for:-

i. Perivalvular abscess
ii. Persistent bacteraemia
iii. Partially dehisced unstable prosthetic valve
iv. Severe congestive heart failure due to valve dysfunction

Competing Interest

No conflicts of interest or sources of funding.

REFERENCES

1. Wilson, W. R. (1995). Antibiotic treatment of adults with infective endocarditis due to streptococci, enterococci, staphylococci, and HACEK microorganisms. American Heart Association. *JAMA: The Journal of the American Medical Association*, *274*(21), 1706–1713. https://doi.org/10.1001/jama.274.21.1706
2. Mocchegiani, R., & Nataloni, M. (2009). Complications of Infective Endocarditis. *Cardiovascular & Hematological Disorders-Drug Targets*, *9*(4), 240–248. https://doi.org/10.2174/1871529x10909040240
3. Mocchegiani, R., & Nataloni, M. (2009b). Complications of Infective Endocarditis. *Cardiovascular & Hematological Disorders-Drug Targets*, *9*(4), 240–248. https://doi.org/10.2174/1871529x10909040240
4. Anavekar, N. S., Schultz, J. C., De Sa, D. D. C., Thomas, J. M., Lahr, B. D., Tleyjeh, I. M., Steckelberg, J. M., Wilson, W. R., & Baddour, L. M. (2011). Modifiers of Symptomatic Embolic Risk in Infective Endocarditis. *Mayo Clinic Proceedings*, *86*(11), 1068–1074. https://doi.org/10.4065/mcp.2011.0111
5. Cabell, C. H., Pond, K. K., Peterson, G. E., Durack, D. T., Corey, G., Anderson, D. J., Ryan, T., Lukes, A. S., & Sexton, D. J. (2001). The risk of stroke and death in patients with aortic and mitral valve endocarditis. *American Heart Journal*, *142*(1), 75–80. https://doi.org/10.1067/mhj.2001.115790
6. Chan, K. L., Dumesnil, J. G., Cujec, B., Sanfilippo, A. J., Jue, J., Turek, M. A., Robinson, T. I., & Moher, D. (2003). A randomized trial of aspirin on the risk of embolic events in patients with infective endocarditis. *Journal of the American College of Cardiology*, *42*(5), 775–780. https://doi.org/10.1016/s0735-1097(03)00829-5
7. Dababneh, H., Hedna, V. S., Ford, J., Taimeh, Z., Peters, K., Mocco, J., & Waters, M. F. (2012). Endovascular intervention for acute stroke due to infective endocarditis. *Neurosurgical Focus*, *32*(2), E1. https://doi.org/10.3171/2011.11.focus11263
8. Dickerman, S. A., Abrutyn, E., Barsic, B., Bouza, E., Cecchi, E., Moreno, A., Doco-Lecompte, T., Eisen, D. P., Fortes, C. Q., Fowler, V. G., Lerakis, S., Miro, J. M., Pappas, P., Peterson, G. E., Rubinstein, E., Sexton, D. J., Suter, F., Tornos, P., Verhagen, D. W., & Cabell, C. H. (2007). The relationship between the initiation of antimicrobial therapy and the incidence of stroke in infective endocarditis: An analysis from the ICE Prospective Cohort Study (ICE-PCS). *American Heart Journal*, *154*(6), 1086–1094. https://doi.org/10.1016/j.ahj.2007.07.023