Neurologic Manifestations of Infective Endocarditis.

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Abstract

Infective endocarditis (IE) is a life-threatening condition often manifesting as a multisystem disease. The clinical presentations are heterogeneous making the diagnosis sometimes difficult. We report the case of a 59-year -old presented to the emergency department with acute abdominal pain. In this context, endocarditis was suspected. Transesophageal echocardiography (TOE) was realized confirming the diagnosis and the abdominal pain was explained by to mesenteric ischemia.

Neurologic manifestations of IE mainly occur before antibiotic treatment is began, thus reinforcing our belief that rapid diagnosis and initiation of antibiotic therapy may still be the most effective way to prevent neurologic complications.

Keywords: Infective endocarditis; Diagnosis; Neurologic Manifestations; Emergency

INTRODUCTION

Infective endocarditis (IE) is an infection of the endocardial surface of the heart which may include one or more heart valves, the mural endocardium, or a septal defect. Its intracardiac effects include severe valvular insufficiency, which may lead to intractable. congestive heart failure and myocardial abscesses. It is a life-threatening condition often manifesting as a multisystem disease. Diagnosis is often challenging due to its heterogeneous features and is often delayed due to the non-specific nature of its presentations.

CASE PRESENTATION

A 59-year-old man from a nursing home presented to the emergency department with acute abdominal pain over the past few hours. The patient described his pain as severe, without exacerbating or relieving factors, and associated with one episode of non-bloody, non-bilious vomiting. He has no past medical history. He was a lifelong nonsmoker, and non-alcoholic. On physical examination; slight dyspnea, pulsed oxygen saturation was 99%, the hemodynamic state was correct, conscious patient, pinpoint pupils, and the temperature was 39, and diffuse abdominal pain, especially in the epigastric area. However, these physical findings did not correlate with his abdominal pain which he described as severe. Thus, he received a morphine injection. Initial laboratory tests showed a white blood cell count of 12700, no liver or pancreatic dysfunction, hypokalemia at 3,4, and no further metabolic dysfunction. Urine analysis showed 230 leukocytes per µL with negative culture. Peritonitis was suspected. An abdominal computed tomography (CT) with contrast ruled out this diagnosis. Meanwhile, the patient's clinical condition deteriorated; his state of consciousness was altered with a Glasgow Coma scale at 12 without focal neurologic deficits or clinical meningeal syndrome. We performed a non-contrast cerebral CT scan that showed no stroke or recent trauma. Blood testing for drugs was negative. A lumber puncture was realized; leukocytes <1, no hyperproteinorrachy, and a normal glucose ration. A cerebral MRI was performed, it was also negative, excluding maxillary sinusitis (Figure 1).

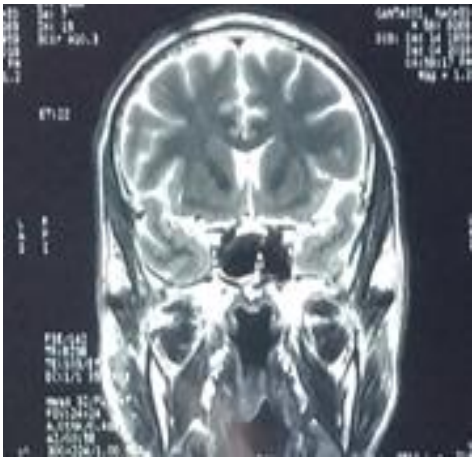

Figure 1: First cerebral MRI, with no obvious abnormalities

Three days after; the state of consciousness has deteriorated; a cerebral computed tomography (CT) with contrast showed no specific lesions. The second cerebral MRI confirmed the presence of rhombencephalitis with bi-thalamic involvement. We suspected infective encephalitis and realized a Second lumber punction which was negative. PCR herpes, Serology for HIV, hepatitis B virus, hepatitis C virus, enterovirus, West Nile virus, and listeria were negative. Drug-induced encephalitis was suspected; the toxicological blood test was negative. A third cerebral MRI showed multiple vascular ischemic strokes (Figure 2).

In this context, echo-doppler of the supra-aortic vessels was realized for suspicion of thrombosis and was normal. An endocarditis was suspected transthoracic echocardiography TTE performed the same day didn't objectify vegetations or valvulopathy neither left auricular/ventricular thrombus and no dilatation of left auricular. That's why transoesophageal echocardiography (TOE)was realized showing interauricules septum aneurysm of 14 mm and mitral valve leak

that could explain the origin of the stroke, abdominal pain due probably to mesenteric ischemia.

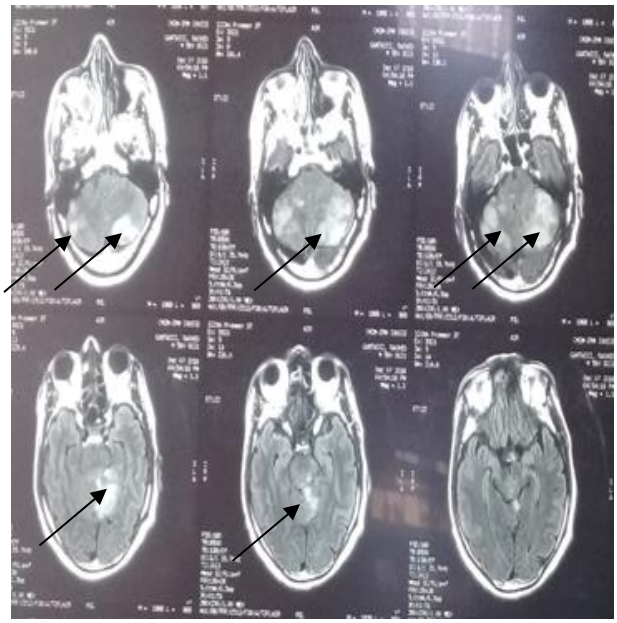

Figure 2: Cerebral MRI showing multiple vascular ischemic stroke (↓)

The clinical diagnostic threshold for possible infective endocarditis was reached using the modified Duke criteria with three minor criteria; fever at 39; metastatic embolization (cerebral and abdominal infarcts) and positive echocardiography. Treatment was initiated with empiric antibiotic therapy for septic urines, aciclovir for suspicion of encephalitis; and secondarily curative treatment.

DISCUSSION

Infective endocarditis (IE) is a rare disease, but its impact is significant [1]. It affects 3 to 10 per 100,000 per year in the population at large, and epidemiological studies suggest that the incidence is rising [2–3]. Reaching a rapid and accurate diagnosis in cases of suspected IE is the challenge. Delayed diagnosis and initiation of therapy lead to complications and worse clinical outcomes [4–5].IE can present acutely or insidiously with non-specific systemic symptoms and/or variable organ pathologies due to complications of septic embolism, hematogenous seeding, immunological phenomena, and, rarely, vasculitis, in addition to its cardiac manifestations. Clinical diagnosis is based on the Modified Duke's criteria: two positive blood cultures for a microorganism typical of IE (or two positive cultures drawn 12 h apart for other microorganisms consistent with IE or a single blood culture or serology consistent with Coxiella burnetti infection), together with evidence of endocardial involvement (2 major criteria). Alternatively, various minor criteria, of which 5, or 3 with 1 of the 2 major criteria, are considered diagnostic [6]. There are a few concerns about the reliability of Duke's criteria. Failing to realize a blood culture due to initial lack of suspicion of Infectious Endocarditis, while giving empiric antibiotic therapy, may result in subsequent negative cultures after an organism has been isolated. In the case that we report here, the initial presentation of acute abdominal pain in the context of fever with good hemodynamic respiratory and neurological state did not raise the suspicion of endocarditis in the first instance. Echocardiography remains the cornerstone of imaging and is rapid, straightforward, and, in many cases, diagnostic (9). Transthoracic echocardiography (TTE) is the recommended

initial modality of choice for both native valve infective endocarditis (NVE) and PVE [10]. For suspected NVE, TTE has a sensitivity of 50% to 90% and a specificity of 90%. For suspected PVE, the sensitivity of TTE is lower, at 40% to 70%, and it provides value in the assessment of ventricular size and function, hemodynamic severity of valve lesions, and in the diagnosis of anterior prosthetic aortic valve abscesses, which may be difficult to visualize on transesophageal echocardiography (TOE). TEE is indicated when TTE is positive or nondiagnostic, when complications are suspected, or when intracardiac devices are present. For suspected NVE, TEE has a sensitivity of 90% to 100% and a specificity of 90% for the detection of vegetations, and it is superior to TTE for the detection of complications, such as perforations, abscesses, and fistulae (11, 12, 13). In PVE, a recent meta-analysis reported a sensitivity of only 86% (95% confidence interval [CI]: 77% to 92%) for TEE in making the diagnosis, and other imaging modalities are emerging to help make or exclude the diagnosis in cases TEE is non-diagnostic. Even when abnormalities are detected, it can be difficult to differentiate nodules from small vegetations or distinguish signs of infection from post-operative change (1). The neurologic complications were classified into the following categories: (1) embolic brain infarction, (2) TIA, (3) cerebral hemorrhage, (4) meningitis, (5) brain abscess, (6) toxic encephalopathy, and (7) headache. Neurological complications occur in 20–40% of cases and are the presenting features in approximately half of these patients with an embolic event as the most frequent manifestation 42% [2,6,14]. The risk of stroke is highest at diagnosis and decreases rapidly after the initiation of antibiotic therapy. Although not explicitly recommended in the European Society of Cardiology's previous guidelines which only stated that "systematic abdominal and cerebral CT scan may be helpful",[7] CT scans or MRI are common and now recommended diagnostic procedures in patients with IE, even in the absence of neurological symptoms. In the 2008 French survey on IE, [8] 70% of the patients with left-sided IE had neuroimaging procedures, of whom 65% had t in the absence of any neurological symptom. These systematic imaging procedures aim to assess the existence of asymptomatic complications of IE, which may both support the diagnosis and modify the therapeutic strategy. The visualization of large vegetation often leads practitioners to perform such examinations. Morphine sulfate intoxication may be responsible for leuko encephalopathy which results in hyper-intense T2 signals in the white matter of the semiovascular center, corpus callosum, and cerebellum. Otherwise, in our patient, the lesions are bithalamic and in the cerebellar peduncles.

CONCLUSION

In conclusion, neurologic manifestations of IE mainly occur before antibiotic treatment is begun, thus reinforcing our belief that rapid diagnosis and initiation of antibiotic therapy may still be the

most effective way to prevent neurologic complications. These data relate the importance of diagnostic alertness to the prognosis of patients with IE.

REFERENCES

1. Cahill TJ, Prendergast BD. Infective endocarditis. Lancet 2016;387:882–93.

2. Pant S, Patel NJ, Deshmukh A, et al. Trends in infective endocarditis incidence, microbiology, and valve replacement in the United States from 2000 to 2011. J Am CollCardiol 2015;65:2070–6.

3. Dayer MJ, Jones S, Prendergast B, et al. Incidence of infective endocarditis in England, 2000-13: a secular trend, interrupted time-series analysis. Lancet 2015;385:1219–28.

4. Dickerman SA, Abrutyn E, Barsic B, et al., ICE Investigators. 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). Am Heart J 2007;154:1086–94.

5. Lodise TP, McKinnon PS, Swiderski L, et al. Outcomes analysis of delayed antibiotic treatment for hospital-acquired Staphylococcus aureus bacteremia. Clin Infect Dis 2003;36:1418–23.

6-Li JS, Sexton DJ, Mick N, et al. Proposed modifications to the Duke criteria for the diagnosis of infective endocarditis. Clin Infect Dis 2000;30:633–8

7 - Habib G, Hoen B, Tornos P, Thuny F, Prendergast B, Vilacosta I, et al. (2009) Guidelines on the prevention, diagnosis, and treatment of infective endocarditis (new version 2009): the Task Force on the Prevention, Diagnosis, and Treatment of infective endocarditis of the European Society of Cardiology (ESC). Endorsed by the European Society of Clinical Microbiology and Infectious Diseases (ESCMID) and the International Society of Chemotherapy (ISC) for Infection and Cancer. EurHeart J 30: 2369–2413. pmid:19713420

8- Selton-Suty C, Celard M, Le Moing V, Doco-Lecompte T, Chirouze C, Iung B, et al. (2012) Preeminence of Staphylococcus aureus in infective endocarditis: a 1-year population-based survey. Clin Infect Dis 54: 1230–1239. pmid:22492317

9- Randolph P. Martin MD. Clinical utility of two-dimensional echocardiography in infective endocarditis. The American Journal of Cardiology.1980;46: 379-385.

10- Dillon JC, Feigenbaum H, Konecke LL, et al. Echocardiographic manifestations of valvular vegetations. Am Heart J1973;6:698–704.

11- Reynolds HR, Jagen MA, Tunick PA, et al. Sensitivity of transthoracic versus transesophageal echocardiography for the detection of native valve vegetations in the modern era. J Am Soc Echocardiogr2003;16:67–70.

12- Shapiro SM, Young E, De Guzman S, et al. Transesophageal echocardiography in the diagnosis of infective endocarditis. Chest1994;105:377–82.

13- Erbel R, Rohmann S, Drexler M, et al. Improved diagnostic value of echocardiography in patients with infective endocarditis by transoesophageal approach. A prospective study. Eur Heart J1988;9:43–53.

14- Neurologic Manifestations of Infective Endocarditis: A 17-Year Experience in a Teaching Hospital in Finland.Maija Heiro MD. Arch Intern Med. 2000;160(18):2781-2787.