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Case Report

Subacute infective endocarditis presenting as upper abdominal pain

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ABSTRACT

Splenic abscess develops in 3-5% of patients with infective endocarditis. In more protracted subacute cases of infective endocarditis, symptoms and signs such as anorexia, weight loss, weakness, arthralgia and abdominal pain may occur in 5-30% of patients and thereby misleading the clinician to pursue incorrect diagnosis such as malignancy, connective tissue disease, or other chronic infection or systemic inflammatory disorders. Left upper quadrant pain can be a presenting symptom in a patient with IE, if it is complicated by septic embolization to spleen. Here reported a case of subacute infective endocarditis complicated with splenic embolization in a 34-year-old male with diabetic nephropathy and ischemic dilated cardiomyopathy, presented as acute abdominal pain.

Keywords: Infective endocarditis, Splenic abscess, Abdominal pain

INTRODUCTION

Embolization is a dreaded complication of infective endocarditis. Stroke comprises up to 65% of embolic events.[1] Emboli may also involve liver, spleen, kidneys and lungs. The spleen is a common site of septic embolization. Splenic abscess develops in 3-5% of patients with infective endocarditis.[2] Splenic embolization most often is not identified by localized symptoms or findings but is discovered incidentally on CT or other imaging techniques.[3] The spleen may be affected either by bacteraemia seeding an infarcted splenic zone, or directly through seeding of the spleen by infected embolized vegetations of the heart valves.[4]

CASE REPORT

A 38-year-old male with history of Type 2 diabetes for last 5 years on OAD (Teneligliptin 5mg OD), CKD-stage 5, not on MHD, hypertensive on triple anti hypertensives including a diuretic and ischemic DCM with moderate TR, mild MR, severe LV systolic dysfunction (EF-28%), presented in ER with complaints of upper abdominal pain for 1 day, palpitations and difficulty in breathing for 7 days, and generalised weakness, anorexia for 1 month.

On examination: patient was conscious, oriented, afebrile, pulse-110/minute, regular, BP-140/90 mmHg, SPo2-96% in RA, pallor (+), bilateral pitting pedal oedema (+) and JVP was raised. A pan systolic murmur heard in mitral area with radiation to axilla, air entry was absent in left basal area of lung field with dull note on percussion, P/A-ascites with diffuse abdominal tenderness and bowel sounds were present.

Chest and abdominal X-ray showed cardiomegaly and left sided pleural effusion with no gas under diaphragm or multiple air fluid levels, ECG-sinus tachycardia, RBS-180 mg/dl, Hb 7.6 g/dl, TC 20000/mm^3, DC-P92L8, ESR-50 mm/hour, PS-normocytic normochromic anaemia, s.albumin 2.5 mg/dl, serum creatinine 3.3 mg/dl, sodium 115 mEq/l, potassium 4.9 mEq/l, urine RE-4+ proteinuria, 5-7 pus cells/hpf, 2-3 RBC/hpf. Serum amylase and lipase levels were normal, USG abdomen-B/L renal parenchymal disease and ascites.

Ascitic fluid was haemorrhagic in appearance with Protein-4, albumin-1.6, sugar-241, TC-5500, DC-P96L4, gram negative bacilli+, ADA-5. Pleural fluid analysis showed a transudate effusion. Hence a provisional diagnosis of spontaneous bacterial peritonitis was made and the patient was admitted in ward for further management.

After admission, empirical antibiotics for SBP, injectable diuretics, ivabradine and other supportive treatment for heart failure was started for the patient. OAD was replaced with insulin in view of uncontrolled blood sugars and 2 units of PRBC transfusion was given for the correction of anaemia. Patient was symptomatically better with the initial treatment, his heart failure symptoms disappeared, but on day 3 of hospital admission, patient developed severe left hypochondrial pain with tenderness on palpation, TC increased to 24000, urea-334, creatinine-8, K-6. X-ray abdomen was repeated and was normal. Ascitic fluid, pleural fluid and urine culture showed no bacterial growth.

Non-contrast computed tomography (NCCT) abdomen (Figure1) was done to rule out any secondary causes of peritonitis. It showed splenomegaly (13cm) with several well demarcated hypodense wedge-shaped lesions reaching the capsule from hilum predominantly involving upper and mid pole region suggestive of multiple splenic infarcts. Repeat ultrasound abdomen showed a heterogeneous lesion with floating debris without vascularity noted in spleen (10×7 cm) suggestive of liver abscess. Surgery consultation given for further management of splenic abscess and image guided transcutaneous aspiration of abscess was planned for the patient, but it was not done because patient didn't give consent for the same.

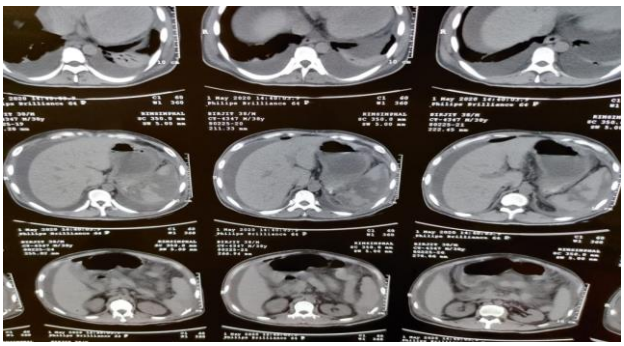

Figure 1: NCCT abdomen of multiple infarcts in spleen.

The patient was further evaluated for the etiology of splenic infarct. 24-hour urine protein was 1.2 g and coagulation profile were normal. Therefore, hypercoagulable state secondary to nephrotic syndrome was ruled out. Echocardiography was advised to see for any intracardiac source of systemic emboli. Echo (Figure 2) showed a vegetation 13.3 mm attached to AML chordae-papillary junction with severe LV systolic dysfunction (EF-25%). Two consecutive blood cultures showed the growth of staphylococcus aureus. Thus, a diagnosis of staphylococcal left sided native valve subacute endocarditis involving anterior mitral leaflet complicated with heart failure with reduced ejection fraction and systemic embolization to spleen was made. Patient was started on injectable Ceftriaxone and renal adjusted dose of Gentamycin. Patient improved symptomatically, repeat blood cultures were sterile and discharged and kept under follow up.

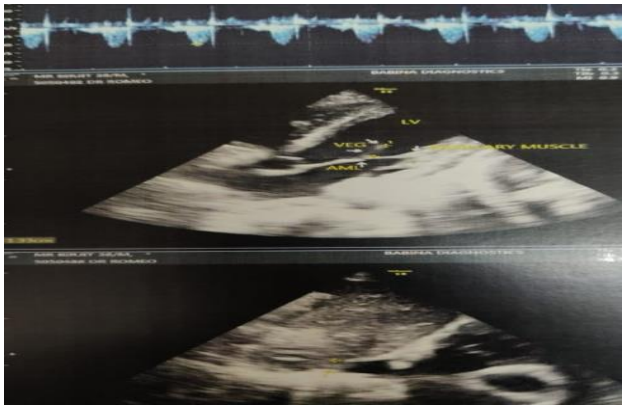

Figure 2: 2D of vegetation attached to anterior mitral leaflet.

DISCUSSION

Infective endocarditis is a disease caused by microbial infection of the endothelial lining of intracardiac structures and is invariably fatal if untreated. With the aging of the population, an increase in the prevalence of degenerative heart valve disease, the use of implanted heart valve substitutes and intracardiac devices are now common.[1] The number of patients with chronic, predisposing medical comorbidities such as diabetes, HIV infection and end-stage renal disease have also increased.[1] Splenic abscess is described as a rare complication of left-sided infective endocarditis. Splenic infarctions are more common conditions and are often asymptomatic. Approximately 5% of patients with splenic infarction will eventually develop splenic abscess.[5]

The patient with IE may report fever, fatigue, anorexia, weight loss, night sweats, joint pain and back pain[1]. Fever (38^0 C) is the most common presenting symptom in up to 95% of patients. Fever may be blunted in patients who are elderly, are severely debilitated, or have renal failure.[2] In this case, there was no history of fever and patient was afebrile throughout the hospital stay. The absence of fever can be due to co-existing T2DM and ESRD. In more protracted subacute cases of IE, symptoms and signs such as anorexia, weight loss, weakness, arthralgias and abdominal pain may occur in 5-30% of patients and thereby misleading the clinician to pursue incorrect diagnosis such as malignancy, connective tissue disease, or other chronic infection or systemic inflammatory

disorders.[3] In this case also presence of abdominal pain misleads us to make a diagnosis of SBP, missing the primary diagnosis of IE.

Features on clinical examination that raise suspicion for IE includes fever, a new heart murmur, signs of heart failure and vascular and immunological phenomenon.[1] In this case, classical peripheral signs of IE like splinter haemorrhage, Janeway lesion, Osler nodes, petechiae and Roth spots were absent. Abdominal examination may elicit nonspecific findings of tenderness and discomfort, particularly in the left upper quadrant, suggestive of splenic embolization and infarct, particularly if complicated by splenic abscess. Splenomegaly usually is associated with a more protracted course of subacute IE.[3]

The diagnosis of IE rests on the ability to demonstrate endocardial involvement of infection and persistent bacteraemia. Clinical, microbiological, echocardiographic features are the foundation for the modified duke criteria, which has become the standard for diagnosis of IE.[1] In this case 2 major criteria and 2 minors (predisposing heart condition, major arterial emboli) were there. Diagnosis of splenic infarcts was an incidental finding on NCCT abdomen in this case. The transformation of splenic infarct to an abscess was documented with an abdominal ultrasound.

Effective therapy requires either image guided percutaneous drainage or splenectomy. Antithrombotic therapy does not prevent systemic emboli in NVE.[2] The frequency of embolization decreases rapidly with effective antibiotic therapy.[2] The 2-week penicillin/gentamycin and ceftriaxone/gentamycin regimens should not be used to treat PVE or NVE complicated by cardiac or extra cardiac abscess.[2] Therefore, in this case, have given the appropriate intravenous antibiotics for a period of 4 weeks and splenic abscess and heart failure with reduced ejection fraction was managed conservatively.

CONCLUSION

Left upper quadrant pain can be a presenting symptom in a patient with IE, if it is complicated by septic embolization to spleen. Possibility of IE should be considered in patients with end-stage renal disease with a predisposing heart valve lesion, presenting with symptoms of heart failure or systemic embolization even in the absence of fever and peripheral signs of IE. Thus, a high index of suspicion is needed in appropriate clinical settings.

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