Cardiac Abscess with Ventricular Aneurysm Secondary to Old Myocardial Infarction

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Abstract
Myocardial Abscess (MA) is a rare life threatening condition mostly occurring secondary to underlying infective endocarditis. But it can also be found secondary to septicemia with some unknown focus of sepsis in the body. Development of MA as a consequence of left ventricular aneurysm (LVA) secondary to myocardial infarction is very rare. We report the case of 56 year old male, who presented with high grade fever and leukocytosis. On detailed evaluation, he was found to have a myocardial abscess with underlying LVA.

Introduction
In ischemic heart disease patients, LVA develops in up to 30 to 35 percent of patients with Q wave MI. However, the incidence of this complication is decreasing, and currently is about 8 to 15 percent in such patients. However, cardiac abscess is very unusual in such circumstances. A few such cases have been reported till now, so we report a case of myocardial abscess in left ventricular aneurysm.

Case Report
A 56 year old male presented with complaints of intermittent, high grade fever for 3 weeks associated with chills and rigors. Patient took intermittent course of antibiotics from local practitioner with partial relief. There was a history of exertional dyspnoea, but not associated with any postural changes. Patient had a past history of acute anterior wall myocardial infarction 2 years back for which he was thrombolysed, (of which no records were available) and was discharged without any complications but his compliance to treatment was poor. He was a chronic smoker consuming approximately one bundle of bidi per day for last 30-35 years.

On examination, patient was febrile with 102°F temperature, had tachycardia with a pulse rate of 112/min, low volume pulse and tachypnoea with a respiratory rate of 24/min, thoraco-abdominal type. His blood pressure was 102/64mmHg. His jugular venous pressure was normal with no clubbing, cyanosis, pallor, icterus or lymphadenopathy. There were no cardiac murmurs. No peripheral signs of infective endocarditis were present. Fine inspiratory crackles were present in left infrascapular area. Abdomen was soft with no organomegaly or tenderness. Patient was conscious on the day of admission but became drowsy on the third day without any focal neurological deficit.

On investigation, patient had a markedly raised total leukocyte count of 35,000/cumm with 90% polymorphonuclear leukocytes. Urine examination revealed 25-30 pus cells per high power field with culture showing Escherichia Coli >10⁵ CFU. Blood culture was sterile. Blood urea nitrogen was 59 mg/dl with a serum creatinine of 3.1 mg/dl suggestive of pre-renal type of renal dysfunction. His random blood sugar was 249 mg/dl and fasting glucose was 169 mg/dl. Cardiac enzymes were within normal limits. Liver function tests were within normal limits. Chest X ray was within normal limits. Ultrasonography showed grade 1 hydrenephrosis with dilated pelvis and upper ureter in left kidney. ECG revealed features of old anterior wall myocardial infarction with Q waves and deep negative T waves in V1-V6; with T wave inversion in lead I, II, III, aVL and aVF. 2D echocardiography showed thin dyskinetic distal interventricular septum, apex and anterolateral wall; a large mass with refractile rim in the left ventricular apex suggestive of LV clot with surrounding abscesses; mild mitral regurgitation; left ventricular ejection fraction of 30% and grade 3 diastolic dysfunction (Figure 1). CECT thorax showed mediastinal and bilateral axillary lymphadenopathy with largest 13×8 mm with a well-defined 38×38×48 mm sized lesion with multiple air fluid levels and hyper intensity within; peripheral enhancement was noted in myocardium of left ventricle with loss of fat plane with pericardium and fat stranding of paracardiac mediastinal fat and was s/o a myocardial abscess (Figure 2). Patient was managed on intravenous antibiotics and fluids, and was referred for surgical intervention.

Discussion
Myocardial abscess has been described in various anatomic locations including the atrial auricle, free wall of the ventricles, perivalvular region, septal wall, and in ventricular septal defect. Myocardial abscess usually occurs in the setting of infective endocarditis, essentially aortic valve and prosthetic valve endocarditis,
but it can also be found in septicemia without infective endocarditis. It can also occur as a complication of various clinical conditions, such as acute myocardial infarction, blunt trauma and penetrating injuries, following invasive cardiac procedures, left ventricular aneurysm infection, and infection of an atrial myxoma.

Primary bacterial infection of myocardial tissue without associated endocarditis occurs only rarely. This report concerns a case of an abscess that developed in a pre-existing ventricular aneurysm. Our patient presented with hectic fever with dyspnoea of which he got partial relief with antibiotics and had significant past history of acute anterior wall myocardial infarction 2 years back. Patient was investigated for fever as PUO and was found to have polymorphonuclear leucocytosis with urine showing pus cell and culture of the same revealed E. coli. Unexplained dyspnoea warranted a trans-thoracic echocardiography which revealed apical aneurysm with LV clot surrounded by myocardial abscess. The diagnosis of which was confirmed by further CECT thorax.


Staphylococcus aureus is the most common recognized cause of myocardial abscess followed by Streptococci and Neisseria meningitides. Escherichia coli was a common cause of myocardial abscess in the series of Sanson et al. In their review of the pathogenesis of myocardial abscess, Tennant and Parks, described three groups of patients. First, and most common, were myocardial abscesses that occurred in the setting of overwhelming sepsis, in which there were generally other organs with abscesses, but no evidence of endocarditis. The second group were patients with endocarditis who developed myocardial abscess by direct extension. The third group were patients who seeded their myocardium, but not any other organ, from a suppurative focus elsewhere. A subgroup of the latter category included rare patients who seeded an injured area of the heart, e.g. an infarct or a ventricular aneurysm.

Clinical picture of myocardial abscess varies widely with mild symptoms to frank heart failure, arrhythmias, wall rupture etc. Routine laboratory testing may reveal leucocytosis with band forms. Electrocardiograms demonstrate a range of abnormalities. Tachycardia is common as are non-specific ST-T wave changes which may be a consequence of direct cardiac injury, or may results from alterations in the host physiologic and metabolic state, i.e. hyperpyrexia, electrolyte abnormalities and respiratory and central nervous system disturbances which may produce hypoxia.

TEE is the imaging modality of choice for establishing a correct diagnosis. But presence of unexplained dyspnoea in our patient with a background of myocardial infarction warranted an echocardiography which revealed presence of myocardial abscess which was confirmed by CECT thorax which had been found to be equally efficacious in detecting vegetation, abscesses and pseudo-aneurysms by Feuchtner et al. Although a non-cardiac gated CT was performed in our patient, it was sufficiently abnormal to prove the diagnosis of myocardial abscess.

**Conclusion**

Because of the variable clinical manifestations, which overlap with other disease states, the diagnosis of myocardial abscess can be challenging. Hence a high index of suspicion is required when PUO occurs in the setting of pre-existing heart disease.

**References**