

Case Report

Infective Endocarditis in the Elderly: Severe Mitral Regurgitation in Acute Coronary Syndrome. A Case Report Highlighting the Changing Patterns of Endocarditis

Hasan Ali Khan, Rashed Al-Hamdan, Adnan Al-Asousi
Department of Medicine, Cardiology Unit, Al-Jahra Hospital, Kuwait

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ABSTRACT

Endocarditis is increasingly becoming a disease of the elderly. We report a case of a 70 year old man with coronary artery disease, who presented with chest pain and fever. His cardiac markers were elevated and an echocardiogram revealed large vegetation on the anterior leaflet of the mitral valve. He also had severe mitral regurgitation and hypokinesia of the posterior and

inferior wall. The association of a mitral regurgitant valve secondary to myocardial infarction and bacterial endocarditis has not been reported in the literature making this case unique and interesting. A discussion on the changing patterns of infective endocarditis reveals interesting facts which are useful in the management of such cases.

KEY WORDS: acute coronary syndrome, elderly, endocarditis, mitral regurgitation

INTRODUCTION

Infective endocarditis (IE) is a highly variable disease. Its clinical course is dependent on a number of factors, including the causative pathogen, age, the underlying health of the patient and the nature and extent of the underlying valvular disease^[1]. There is a general agreement that new trends in the epidemiology of endocarditis have occurred during the past thirty years. Most of these changes relate to the number and types of susceptible hosts rather than to the shifts in the virulence of the infecting microorganisms. In addition, the age distribution of endocarditis has clearly changed. Endocarditis has increasingly become a disease of the elderly^[2]. We report an interesting case of an elderly male patient with infective endocarditis whose mitral valve was damaged as a result of myocardial infarction (MI) and discuss the changing pattern of cases presenting with infective endocarditis in the elderly.

CASE REPORT

A 70-year old male patient was admitted with epigastric pain, lower chest pain for a few hours and fever for one week. The epigastric pain was associated with sweating and vomiting. The fever was intermittent and associated with chills and rigors. He also complained of burning micturition. Patient was treated with oral antibiotics before coming to the hospital. He was a known case of

coronary artery disease (CAD) with a history of inferior myocardial infarction in 1996. With this history of established CAD, the patient was kept in the CCU as unstable angina with an associated urinary tract infection (UTI).

Clinical examination revealed that the patient was conscious, oriented but looked toxic. His heart rate was 110/min, blood pressure 160/90 mmHg and temperature 38° Celsius. There was no pedal edema or cyanosis and Jugular Venous Pressure (JVP) was not elevated. There was no clubbing but the peripheral pulses were weak in the left lower limb and the extremity was cold to feel.

A pansystolic murmur was heard in the mitral area, radiating to the left axilla. Lungs were clear and there was no organomegaly on abdominal examination. The central nervous system was grossly intact.

There was a past history of inferior myocardial infarction in 1996, diabetes mellitus with retinopathy since 1970, peripheral vascular disease with aorto-pulmonary bypass surgery done in 1997. The patient was on regular outpatient follow up and maintaining a fair general health.

Investigations: Hemoglobin 119 g/L, WBC count $25 \times 10^9/L$, biochemistry revealed blood urea of 18.6 mmol/L, creatinine of 189 $\mu\text{mol/L}$, a blood glucose of 22 mmol/L and serum cholesterol of 6.7 mmol/L. Cardiac markers, which included CKMB mass and Cardiac troponin I were elevated and serially increased indicating a myocardial



Fig. 1: 2-D echocardiography of the heart in the long axis view showing large, highly mobile vegetation attached to the atrial side of the anterior mitral leaflet. There was an associated severe mitral regurgitation observed in the Doppler mode.

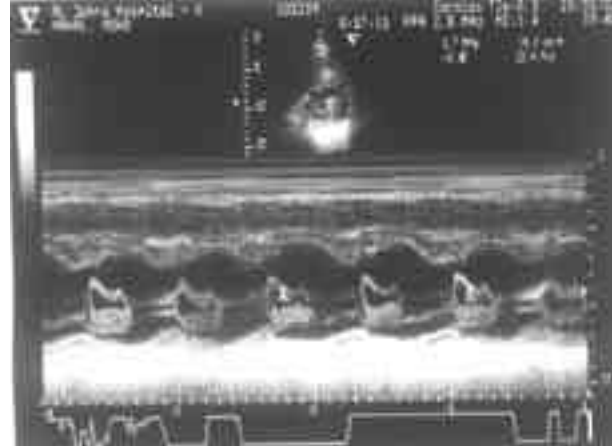


Fig. 2: M-mode echocardiography of the heart showing the vegetation (arrow) between the two leaflets of the mitral valve

infarction. X-ray chest showed cardiomegaly and a small right-sided pleural effusion. Urine routine showed many pus cells and proteins were positive. Urine and blood cultures were sent.

Electrocardiogram showed evidence of old inferior MI and new changes were seen in the form of hyperacute T waves in leads V_2 , V_3 and V_4 , with no Q waves, ST elevation or depression.

A diagnosis of urinary tract infection with suspected septicemia and a non-ST elevation MI (NSTEMI) was made. The patient was appropriately treated for NSTEMI and for UTI with possible septicemia using third generation cephalosporins.

On the third day post admission, as a routine, and because the patient's condition was not improving as expected, an echocardiogram was done which revealed one large vegetation on the anterior mitral leaflet (AML) on 2D mode (Fig. 1) and on M mode (Fig. 2). Additionally, there was a left ventricular dilatation with hypokinesia of the posterior and inferior wall. Left ventricular ejection fraction was 50% and Doppler analysis showed severe mitral regurgitation.

In the meantime, blood culture report was received and it was positive for *Staphylococcus aureus*. Patient was started on an intensive multiple antibiotic combination of vancomycin, cefuroxime and rifampicin. Patient's fever started to settle down but keeping in mind the possibility of a valve replacement surgery, the patient was transferred to the tertiary cardiac center for further management.

DISCUSSION

Various terms have been used to describe the many forms of endocarditis encountered by clinicians. The term infective endocarditis (IE) has now replaced the older terms subacute and acute bacterial endocarditis^[1]. The epidemiology of IE has

evolved over the past fifty years. Mitral valve prolapse and degenerative valvular diseases have replaced rheumatic heart disease as the most common predisposing conditions. The average age of patients with IE has increased, and nosocomially acquired cases are becoming more common^[3]. IE in the elderly patients presents a unique diagnostic and therapeutic challenge. Atypical presentations frequently lead to delayed diagnosis and poor outcome. IE in elderly persons is somewhat more common among men^[4]. Bacteremia is a common disorder in the elderly, and its prevalence and incidence increase with age. It carries a mortality of 20 to 40%. The signs and symptoms of bacteremia are often blunted or nonspecific in the elderly^[5]. Nosocomial endocarditis is usually a complication of bacteremia induced by an invasive procedure or a vascular device and now accounts for 10% of endocarditis in some areas^[6,7]. IE is a multisystem disease and patients may present with diverse clinical features. Elderly patients with bacteremia form a special group due to the presence of associated conditions like cerebrovascular accidents, dementia, and decubitus ulcers, which increase sharply with age, and therefore need a high index of suspicion for IE^[5]. In a recent paper from the Tokyo Medical University, a comparison was made between elderly (20 cases > 60 years) and middle aged (30 cases < 60 years) patients of IE^[8]. The results suggest that although predisposing heart disease and causative microorganism in IE are different between the elderly and the middle-aged patients, the incidence of major complications is similar^[8].

Taking the microbiological pattern changes into consideration, it must be noted that streptococci are currently responsible for a smaller proportion of IE cases than previously, but this group of bacteria remains the most common cause of prosthetic and native valve endocarditis. In some community

hospitals, staphylococci are the most common cause of nosocomial IE, particularly in intravenous drug abusers^[3]. All series of IE had a variable proportion of cases without an etiologic agent because all cultures were negative. New microbiological techniques have permitted the discovery of the role of many other microorganisms in IE. *C. burettii* is an increasingly common causative agent of IE and in its diagnosis the detection of antiphase-I-antibodies, immunochemical, molecular techniques and cellular cultures have been added^[9]. *C.albicans* and *Aspergillus spp.* are the most frequent fungal agents causing IE, and peripheral emboli and large vegetations are frequent. *Legionella*, *Mycoplasma*, *Chlamydia*, *Mycobacteria* and viruses are other potential agents of IE^[9]. Elderly patients have a predilection for anaerobic bloodstream infections, and for multiresistant bacteria, age is not an independent risk factor for resistance^[5].

Antibiotics form the mainstay of treatment and in choosing the appropriate antibiotic, the physician should consider the patient's salient features and the overall susceptibility of the microorganisms in the local ecosystem. The decision to undertake surgery in patients with IE is mainly based on clinical data.

However, echocardiography in particular using a transesophageal approach, will offer important guidelines for diagnosis and management. Surgery must be considered in the following cases: ring abscesses or fistulae, severe mitral regurgitation, large and mobile mitral valve vegetation. The physiological status and the microorganism also are important parameters for the decision to refer cases for surgical intervention^[10]. Also, early surgical therapy is advisable for patients who develop heart failure as a result of severe acute aortic valvular regurgitation. Valve replacement surgery can be performed with acceptable mortality and morbidity even in the very elderly patients^[11].

The prognosis of IE is not uniform. Mortality is high during the initial phase, but after one year the risk of dying is low, although still above that of the general population. Part of the risk is probably the direct consequence of IE, but part is due to the course of the underlying heart disease and factors

like: age, gender, congestive heart failure at the onset and the microorganisms^[12].

Our patient had the unusual association of an IE occurring in a valve damaged due to myocardial infarction, causing the severe mitral regurgitation and to the best of our knowledge, there are no such reports in the literature.

CONCLUSIONS

Our case report reviews the current questions and the new aspects related to IE in elderly patients. In particular, four points are emphasized: a) epidemiological changes during the last decades, b) clinical features and diagnostic challenges of IE in the elderly, c) mortality and prognosis and d) preventive and therapeutic measures that must be taken into account^[13].

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