Acute Myocardial Infarction in a Young Patient of Rheumatic Heart Disease

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Introduction

Primary valvular heart disease ranks well below coronary heart disease, stroke, hypertension, obesity and diabetes as major threats to public health. Rheumatic fever is the dominant cause of valvular heart disease in developing countries. Rheumatic heart disease accounts for 12-65% of hospital admissions related to cardiovascular disease [1]. Nevertheless, it is a source of significant morbidity and mortality.

We report a case of a young patient with rheumatic heart disease who presented with an acute myocardial infarction with features of cardiogenic shock. Early recognition and aggressive management led to satisfactory recovery in this case.

Case Report

A 23 year old lady a known case of operated rheumatic heart disease with double valve replacement done one year ago, on anticoagulation, presented with history of acute onset precordial chest pain and difficulty in breathing for duration of two hours. On examination the pulse was 92/min and the blood pressure was 94/62 mmHg. The respiratory rate was 22/min and she was in apparent discomfort. Examination of the cardiovascular system revealed a mid diastolic murmur and the valvular clicks were well heard. The chest was clear and the rest of the systemic examination was normal. An urgent electrocardiograph (ECG) was done which revealed sinus rhythm with ST elevation of 2mm in lead I, aVL, V3 -6 (Fig. 1, 2) and cardiac enzymes were raised. She was diagnosed to have an acute myocardial infarction and thrombolysed with intravenous streptokinase. Thereafter patient developed hypotension and features of cardiogenic shock and was managed with inotropic support in the form of dopamine and noradrenaline. Investigations revealed an INR of 2.29 and the rest of the hematological and metabolic investigations were essentially within normal limits. 2D Echocardiography and color doppler showed that both the prosthetic valves were functioning well with no vegetations. The anterior wall

was akinetic with a left ventricular ejection fraction of 37%. Echocardiography done two months prior to current event showed an ejection fraction of 55%. A coronary angiography showed normal epicardial coronaries (Fig. 2).

The patient was subsequently discharged with advice to follow up regularly and to continue anticoagulants, diuretics, ACE inhibitors and antiplatelet agents.

Discussion

Coronary artery embolism has been reported in cases of bacterial endocarditis, mitral valve disease, syphilitic heart disease, prosthetic valves, intracardiac thrombus, Teflon patches and paradoxically from systemic veins in patients with right to left intracardiac shunts. Since the advent of prosthetic valve surgery, another source of coronary embolism has been introduced, that is, fragments of prosthetic material or more commonly thrombus formed at the surface of the prosthesis [2]. It is often difficult to prove conclusively that an episode of myocardial ischemia is due to coronary embolism and in those patients who survive the diagnosis must be inferential. The occurrence of an acute myocardial infarction in this case with a predisposing illness of rheumatic heart disease with prosthetic valves with normal epicardial coronaries is strongly suggestive of a coronary embolism.

Coronary artery embolization is a rare cause of myocardial infarction. Though coronary emboli are a relatively frequent finding, it was rarely responsible for myocardial infarction whilst many other studies have described an important incidence of myocardial infarction, subsequent angina pectoris, heart failure and other sequelae. In an autopsy series of 1,050 patients with myocardial infarction, Prizel et al [3] found only 55 patients who had coronary embolisms. The conflict of evidence is at least partly due to making a clinical diagnosis of coronary embolism. A study of coronary

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Fig. 1 : ECG showing STEMI.

embolism in valvular heart disease has suggested that in some patients a history of myocardial infarction may be atypical or absent while in others ECG changes could be transient.

Most coronary embolisms occur in the LAD at the take off and downward courses of the LAD are more favorable for embolization than those of the right and left circumflex coronaries which run at 90 degrees from the parent trunk [4]. The incidence of coronary embolism is reported to be about 12% in cases of infective endocarditis [5]. Thromboembolic complications including myocardial infarctions are common in patients with bacteremia with or without endocarditis, but acute myocardial infarctions are rarely diagnosed during life [6]. Final proof that acute myocardial infarction has been due to coronary embolism can only be obtained by pathological examination unless it is clearly demonstrated that embolism occurred during cardiac catheterization. The rapidity with which the embolus resolve means that coronary angiography may fail to demonstrate embolus unless it is performed early.

The consequences of coronary embolism depend on two major factors: the size of the embolus and the size of the lumen of the artery in which it becomes impacted. The smaller the embolus, the greater the chance that it will travel distally to a smaller coronary arterial segment

Fig. 2 : Normal lefty coronary system.

and less is the likelihood of a myocardial infarction or fatal arrhythmia [7].

Currently there is no clear guideline for effective treatment of coronary embolism. Kamishirado et al [8] reported a case of coronary embolism managed with 960,000 units of urokinase and other cases of coronary embolism have been managed with streptokinase and antiplatelet agents. Taniike et al [9] have reported a case of myocardial infarction caused by bacterial embolism and was managed with RESCUE thrombectomy catheter. Quinn et al [10] have reported a case of a 63 year old lady with a St Jude aortic and mitral valve replacement who presented with an acute myocardial infarction. Urgent catheterization revealed a saddle embolus in the circumflex and ramus arteries and another embolus in the diagonal branch. Angioplasty was employed for the totally occluding diagonal embolus and a combined therapy of intracoronary urokinase and intravenous abciximab was successful in achieving complete resolution of the emboli.

To conclude coronary embolism is an uncommon but distinct clinical entity. It can be diagnosed clinically and should be suspected in all cases when myocardial infarction occurs in association with an underlying disease which predisposes to coronary embolism. The diagnosis may be obscured by atypical symptoms and transient ECG changes. The diagnosis is supported by normal coronary arteries by selective coronary arteriography. Treatment with long term anticoagulation may prevent further emboli.

Conflicts of Interest

None identified

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Book Review

Acute Endocrinology. Greet Van der Berghe. Publisher: Humana Press C/o Springer Link, Science +Business Media LLC, 233, Spring Street. New York N.Y.100013 USA. Year of Publication: 2008. Illustration: 27. Hard Cover. Price: 99.95 Euro.

Endocrinology and critical care medicine are considered distant cousins in medicine in that they have been practiced independent of each other. However research in basic sciences and results of landmark studies (Greet Van der Berghe et al) have demonstrated interdependence of the two specialities with important clinical implications. 'Acute Endocrinology' aims at compiling the new findings. The first part deals with the classical life threatening illness caused by primary endocrine diseases such as thyrotoxicosis, hypothyroidism, acute adrenal crisis, acute calcium disorders, pheochromocytoma, severe hyper-and hypoglycaemia.

The second part looks at endocrinology from the ICU side, starting with a general overview of the dynamic neuroendocrine and metabolic stress responses in the condition of intensive care dependent, non endocrine critical illness. Alterations within several of the endocrine axes briefly touched upon in the overview chapter are then further discussed in detail in the following chapters: critical illness induced alterations within the growth hormone axis, the thyroid axis and pituitary adrenal axis, changes in catecholamines, glucose control, salt and water metabolism. This last chapter on salt and water disturbance bridges both the endocrine and non-endocrine causes and their specific approaches. The book will be useful to physicians managing patients in critical care settings.

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