Rheumatic mitral stenosis with epistaxis as an initial symptom

Kumar S, Jain A P, Jain S

ABSTRACT

Epistaxis is a frequent presentation in the emergency department and often causes significant anxiety among patients and their relatives. We report a 30-year-old man with mitral stenosis presenting with epistaxis as the initial symptom. He had no other symptoms related to mitral stenosis prior to this episode. Epistaxis is a very unusual initial presentation of rheumatic mitral stenosis.

Keywords: mitral stenosis, epistaxis, rheumatic heart disease

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INTRODUCTION

Epistaxis is one of the most common presentations with more than two-thirds of the population suffering from it at some stage in their lives. It is often influenced by environmental conditions. The general practitioner may be required to deal with this condition and can do so in the majority of cases in an appropriate and safe manner. With training and knowledge, they may also be able to take appropriate measures to deal with other causes of bleeding. We report a case of epistaxis as the only presentation of mitral stenosis (MS), with the aim of making general practitioners aware of the possibility of this underlying cause.

CASE REPORT

A 30-year-old man presented to the emergency department of our hospital with profuse bleeding from the nose. He also complained of fever, occasional sweating (no night sweats), fatigue and intermittent shortness of breath on exertion. The patient had no history of chest pain, palpitations, nausea, vomiting, diarrhoea, rash, congestion, haematuria, arthralgia or myalgia, headache, trauma, recent travelling, or any known sick contacts. He had been in his usual state of health until recently and denied any significant past medical history. There was no history of past surgical procedures. He was not on any medication (including any herbal preparations). He had no history of any drug or environmental allergies.

On examination, the patient was alert, fully oriented and sitting up comfortably in bed. He was afebrile, his pulse was regular at 110/min, blood pressure was 110/80 mmHg, respiratory rate was 22/min and his oxygen saturation was 93% on room air. Jugular venous pressure was not raised. There was no lymphadenopathy. The chest examination was normal. There was tachycardia with a loud S1 without any appreciable murmurs, rubs or gallops. The abdomen was soft with normal bowel sounds and no hepatosplenomegaly. A nose and throat examination showed that the bleeding was from the posterior part of both nasal cavities. It was profuse and the exact site could not be localised. It was brought under control by bilateral anterior nasal packing. Nasal endoscopy was done after removal of the nasal pack. There was no septal deviation or nasal mass or any raw area over septum.

His haemoglobin level was 12 g/dL, total leucocyte count was 10×10^9/L with a differential of 68% neutrophils, and platelet count was 255×10^9/L. Serum electrolytes,

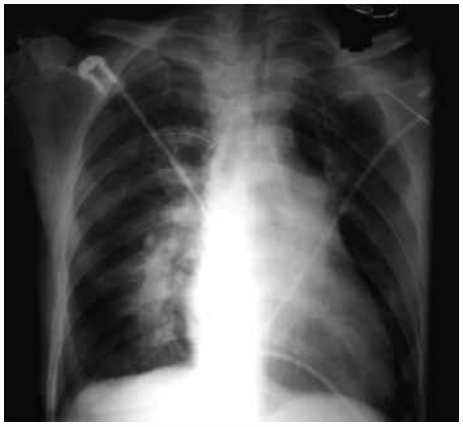

Fig. 1 Radiograph of the chest shows a normal lung field with mitralisation of the left heart border and evidence of pulmonary hypertension.

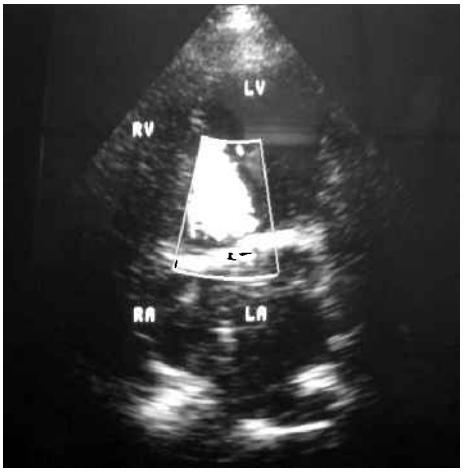

Fig. 2 2D echo with colour Doppler imaging shows a thickened and pliable mitral valve without evidence of calcification. Mitral valvular area (by planimetry) is 3.48 cm^2 and the mitral valve stenosis is mild to moderate.

Department of Medicine, Mahatma Gandhi Institute of Medical Sciences. Sewagram, Wardha 442102, Maharastra, India

Kumar S, MBBS, MD
Senior Lecturer

Jain AP, MD, FICP, PhD
Professor and Head

Department of Otolaryngology

Jain S, MBBS, MS
Senior Lecturer

Correspondence to:
Dr Sunil Kumar
Tel: (91) 9850 393 787
Fax: (91) 7152 284 967
Email:drsunilkr_med@rediffmail.com

hepatic and renal function tests were unremarkable. His chest radiograph showed normal lung fields with mitralisation of the left heart border and evidence of pulmonary hypertension (Fig. 1). Mitralisation of the heart, or straightening of the left border of the heart, is a result of convexity produced due to prominent left atrial (LA) appendages. His electrocardiogram was normal except for sinus tachycardia. Echocardiography showed mitral valve stenosis and prominent LA appendages (Fig. 2). He was given conventional diuretic therapy, and referred for mitral valvotomy. He was doing well on follow-up.

DISCUSSION

MS is the most frequent valvular lesion of established rheumatic heart disease. MS, combined MS, mitral regurgitation are generally rheumatic in origin; very rarely is MS congenital. Pure or predominant MS occurs in approximately 40% of all patients with rheumatic heart disease.[1] This patient also had only MS. Sometimes the symptoms and severity of the disease are poorly correlated because the disease often progresses insidiously and patients reduce their activity without being aware of it. Many patients are asymptomatic, until symptoms are exacerbated by the onset of arrhythmia like atrial fibrillation, intercurrent infection (e.g. pneumonitis), pregnancy or anaemia. This happens due to fusion of the mitral valve apparatus, ultimately resulting in a small orifice; as the valve area decreases, the transvalvular pressure gradient increases. This leads to increasing LA pressure, with resultant LA dilatation and hypertrophy, and pulmonary hypertension. The most common clinical presentation is dyspnoea, orthopnoea, paroxysmal nocturnal dyspnoea and fatigability due to reduced mitral valve area. Palpitation due to atrial fibrillation, haemoptysis, hoarseness and exercise-induced angina may be the presenting symptoms of MS.[2] These symptoms typically do not appear until 15–40 years after an episode of rheumatic fever, but in developing countries, much younger children may become symptomatic because streptococcal infections may not be treated with antibiotics and recurrent infections are common.[3]

Epistaxis is classified on the basis of the primary bleeding site as anterior or posterior. Bleeding is most commonly anterior, originating from the nasal septum. In this patient, bleeding occurred from the posterior part of the nasal cavity. A common source of anterior epistaxis is the Kiesselbach plexus, an anastomotic network of vessels on the anterior portion of the nasal septum. Posterior bleeding occurs mainly from the branches of the sphenopalatine artery in the posterior nasal cavity or nasopharynx, but it may also originate from Woodruff's plexus, a collection of large blood vessels found in many people in the lateral wall of the inferior meatus posteriorly. These vessels appear to originate from the posterior pharyngeal wall and are venous in origin.[4] Some common causes of epistaxis are enumerated:

- Idiopathic, where an identifiable cause is not found.
- Trauma. Local trauma (e.g. nose picking) is the most common cause. Others may be facial trauma, foreign bodies and prolonged inhalation of dry air in dry climates and cold weather.
- Nasogastric and nasotracheal intubation.
- Upper respiratory infection, such as nasal or sinus infection, especially in children.
- Oral anticoagulants and coagulopathy, thrombocytopenia or AIDS-related conditions.
- Hypertension.
- Vascular abnormalities, such as sclerotic vessels, hereditary haemorrhagic telangiectasia, arteriovenous malformation, neoplasm, septal perforation or deviation, and endometriosis may present with epistaxis.

Epistaxis was considered a minor manifestation when the Jones criteria were introduced in 1944 as a set of clinical guidelines for the diagnosis of rheumatic fever.[5] Subsequently, it has been omitted from the Jones criteria owing to a lack of specificity of the symptoms. However, it may be of considerable clinical importance because they may often appear hours or days before major manifestation of the disease, as in this case. A probable mechanism of epistaxis from MS may be increased systemic vascular resistance which is translated back to the nasal mucosa, ultimately leading to bleeding. Although epistaxis as sequelae of MS has been mentioned in the literature,[6] we have made an extensive search on PubMed and other sources, and found no other report of the incidence of epistaxis in MS. In conclusion, MS may be considerd as a possible aetiology in the list of systemic causes of a very common condition, epistaxis. Further studies need to be undertaken to support this hypothesis.

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