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Mitral valve prolapse – do all patients need an echocardiogram?

Sir,

Mitral valve prolapse (MVP) is one of the commonest cardiac abnormalities and yet confusion still exists as to its diagnostic criteria.¹ The diagnosis can be made by several methods. Clinical criteria include auscultation of midsystolic clicks and late systolic murmurs. Echocardiography has played an increasingly important role in diagnosis and is currently the most commonly used method of diagnosis because it is non-invasive and, if strict criteria are followed,^{2,3} accurate. However, in developing countries, echocardiographic facilities are not easily available and are relatively expensive when compared with clinical methods.

The purpose of this study was to assess the usefulness of performing echocardiographic examinations on patients with a clinical diagnosis of MVP.

Thirty six consecutive outpatients (male: 10) had been referred for an echocardiogram from the outpatient clinics (non-cardiological) of the University Hospital, Kuala Lumpur, with a clinical diagnosis of probable or definite mitral valve prolapse. By the nature of this study, referring doctors were unaware that such a study was taking place and hence, no uniform diagnostic criteria were used. Their ages ranged from 13–65 years (mean 29.3 years).

All patients underwent a Doppler echocardiogram examination (Toshiba colour Doppler ultrasonograph SH 65 A). Interpretation of the examination was based on established criteria.^{3,4} Mitral valve prolapse was deemed present if it was detected on at least 2 views, one of which included the parasternal long axis view.² Statistical analysis was performed using the chi-square test.

Of the 36 patients, 24 had a clinical diagnosis of definite MVP while 12 had a clinical diagnosis of probable MVP. In the former group, 12 patients (50%) had MVP on echocardiogram and in the latter group, 7 patients (58.3%) had MVP. There was no significant difference in prevalence of MVP between patients with a clinical diagnosis of definite or probable MVP.

It is well known that prevalence rates of MVP based on auscultatory criteria vary depending on the examiner's skill.¹ However, it cannot be expected that all patients in the community will be screened by a skilled cardiologist. The question therefore arises among doctors concerned with health economics, as to whether or not all patients with a possibility of MVP should be submitted to an echocardiographic examination.

The author would submit that, based on this study, echocardiography is justified. Specificity of auscultatory criteria was low. The pool of referring doctors was not preselected and these doctors referred patients in the course of their usual clinical practice. If an echocardiogram was not available, nearly half the patients would have been misdiagnosed as having MVP with its many medical, social and financial implications.

Echocardiography is currently the most widely accepted diagnostic test for MVP and is used to predict prognosis and plan further management.^{2,5} It would not be unreasonable to suggest that this non-invasive test be made freely available to all patients when a diagnosis of MVP is suspected clinically.

Acknowledgement

The author wishes to acknowledge Miss M.N. Lim for her technical assistance.

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Myasthenia gravis and reversible pyramidal tract signs in a thyrotoxic patient

Sir,

A 33 year old Indian male was admitted with a 3 year history of easy fatigability, tremulousness, weight loss and heat intolerance. Six months previously he had developed protrusion of the eyes, ptosis and diplopia. Later he noticed weakness in both lower limbs, predominantly distal and especially after exertion, which spread to involve the upper limbs, muscles of mastication, swallowing and also the bladder for he started having hesitancy of micturition.

Physical examination revealed bilateral ptosis with restricted ocular movements in all quadrants, with normal vision and fundus, weakness of facial and limb muscles (distal more than proximal), normal tone, generalized hyperreflexia and absent abdominals, cremasterics and bilateral extensor plantar responses.

His neostigmine test and electromyography confirmed the diagnosis of myasthenia gravis. His chest X-ray was normal but computed tomographic scan showed thymic

hyperplasia. Thyroid function tests confirmed thyrotoxicosis. He was treated with carbimazole and neostigmine and within 4 days his upper motor neurone signs reverted to normal.

Of interest is the hyperthyroidism which has been reported in 5–10% of cases of myasthenia gravis.¹ Thymic hyperplasia is seen in 75% of patients with myasthenia gravis² and has been noted with myasthenia gravis associated with thyrotoxicosis.³ Distal limb weakness was more marked in our patient though the literature scanned mentions predominant ocular and proximal involvement. Finally, the most uncommon and interesting association was the transient corticospinal involvement, signs of which reversed in a few days of hospitalization, possibly on the basis of reversible corticospinal tract changes with thyrotoxicosis and their improvement with antithyroid drugs.⁴ The pathophysiological basis of pyramidal tract dysfunction in hyperthyroidism which is reversible⁵ is not known and no histopathological studies have been reported.

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Pericardial tamponade resulting from changing a central venous catheter over a guide wire

Sir,
Pericardial tamponade resulting as a complication of central venous cannulation is well recognized. In renal dialysis units it is routine practice to replace central venous catheters using guide wires. I report a case of haemopericardium in a dialysis patient which followed replacement of a subclavian vein catheter over a guide wire which was inserted through a catheter already in the vein.

A 42 year old female with renal failure secondary to systemic lupus erythematosus was being temporarily haemodialysed via a single lumen 20 cm 8F central venous catheter which had been inserted into the left subclavian vein infraclavicularly using the Seldinger

technique.¹ A chest radiograph after insertion of the catheter showed a normal cardiac outline.

On the fifth dialysis session, 9 days after insertion of the catheter, it was decided to change the catheter due to poor blood flows. A straight uncoated 70 cm × 0.038 in. guide wire (Vas-Cath Inc) was easily inserted through the catheter lumen and the catheter was removed. A new 20 cm 8F catheter with a single lumen was inserted over the guide wire which was then removed.

A chest radiograph following catheter replacement confirmed satisfactory positioning of the catheter in the superior vena cava and showed a normal cardiac outline. The patient was commenced on haemodialysis with a continuous infusion of heparin at a rate of 1500 units/h. Ninety minutes later she became unwell with a sinus tachycardia and hypotension. An echocardiogram confirmed a large collection in the pericardial space and showed right ventricular diastolic collapse confirming tamponade. After unsuccessful attempts at subxiphisternal pericardial aspiration she underwent emergency thoracotomy.

At operation 300 ml of blood with a haematocrit of 0.27 was removed from the pericardial sac. The peripheral blood haematocrit was 0.24. A drain was left *in situ* and a subsequent chest radiograph did not show any change in the position of the catheter. This was later removed and the patient made an uneventful recovery.

We believe that the haemopericardium resulted from a combination of anticoagulation and breaching of the wall of the superior vena cava or right atrium.

The use of a J shaped instead of a straight guide wire may have prevented this complication. This case illustrates that patients requiring anticoagulation are at particular risk of complications related to central venous catheterization.^{2,3} It also shows that a satisfactory chest radiograph following cannulation does not exclude the possibility of serious late complications.

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Leukopenia and thrombocytopenia due to fusidic acid

Sir,
Fusidic acid is increasingly used especially for methicillin-resistant staphylococci. Side effects are limited to gastrointestinal upset,¹ skin rashes,¹ impaired liver function¹ and, rarely, granulocytopenia.^{2,3} We report here leukopenia and thrombocytopenia in association with fusidic acid.