"Duplication" of aortic cusp
New M-mode echocardiographic sign of intimal tear in aortic dissection

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SUMMARY M-mode and two-dimensional echocardiograms were obtained in a patient with acute dissecting aneurysm of the ascending aorta. The M-mode echocardiogram disclosed apparent "duplication" of the non-coronary aortic cusp. Two-dimensional echocardiograms showed this finding to be the result of the motion of a flap of torn aortic intima. This new M-mode finding appears to be a highly specific echocardiographic sign for aortic dissection.

M-mode echocardiographic techniques have proved useful in the detection of dissecting aneurysm of the aorta in patients in whom the disorder is clinically suspected.1 5 Their use is limited by an undetermined incidence of false negatives and false positives. We present a new M-mode finding of dissection—apparent "duplication" of an aortic valve cusp. A two dimensional echocardiographic study in this case shows that this finding was caused by the motion of a flap of torn aortic intima and suggests that this M-mode finding may be highly specific for aortic dissection.

Methods

M-mode and two dimensional echocardiograms were recorded using a Toshiba SSH-10A sonolayergraph. This is a phased array scanner which uses a 32 element transducer with a frequency of 2-4 MHz and a scanning angle of 78°. A single element can be selected and aimed during two dimensional viewing to record an M-mode recording of the area of interest.

M-mode recordings were made on a Honeywell 1896 strip chart recorder on dry silver paper. Two dimensional videotape recordings were made of the same area within minutes of the M-mode recording and stop frame photographed from a monitoring television screen.

Case report

A 35-year-old black man presented with a three week history of increasing dyspnoea on exertion, orthopnoea, paroxysmal nocturnal dyspnoea, ankle oedema, and palpitation. He had been known to have a heart murmur for 15 years. There was no history of chest pain. He had been seen as an outpatient one year and again two months before admission and had been noted to have a blood pressure of 270/150 mmHg in both arms, but he had refused admission or treatment. On physical examination he was in moderate respiratory distress. The blood pressure was 180/52 mmHg in both arms, the heart rate was 140, and the respiratory rate 40. The jugular venous pressure was normal and the carotid pulses were hyperdynamic. There were râles half way up the chest bilaterally. The apex beat was diffuse and laterally displaced. The first sound was loud, the second sound single, and there was a loud summation gallop. There was a grade 3/6 systolic ejection murmur heard all over the precordium. There was a grade 3/6 blowing decrescendo diastolic murmur, loudest at the left sternal border and radiating to the apex. There was moderate bilateral ankle oedema. Certain features suggested Marfan's syndrome: a height of 198 cm (6 ft, 6 in), pectus excavatum, a high arched palate, arachnodactyly, and hyperextensible joints. The chest x-ray film showed cardiomegaly, dilatation of the ascending aorta, and pulmonary oedema. The electrocardiogram showed sinus tachycardia, left atrial "enlargement", borderline voltages for left ventricular hypertrophy, and nonspecific lateral ST-T changes. The patient was stabilised on nasal O2, morphine, intravenous frusemide, and a nitroprusside infusion. Both
M-mode and two dimensional echocardiograms were obtained (see Results). An aortogram, performed during nitroprusside infusion, showed a type I dissecting aneurysm starting near the origin of the right coronary artery, and involving the right lateral wall of a massively dilated aortic root. There was free communication between the true and false lumens and gross aortic regurgitation. The central aortic pressure was 150/60 (mean 88 mmHg). Right heart pressures were normal. The mean pulmonary capillary wedge pressure was 18 mmHg. The mixed venous oxygen saturation was 45 per cent (haematocrit 45%). These findings were consistent with left ventricular failure and a low output state. At operation incision into the aorta disclosed a wide intimal tear of the ascending aorta beginning slightly superior to the right coronary ostium, and a large type I dissecting aneurysm extending up the right lateral wall of the arch. The proximal portion of the dissected intima lay on the aortic valve, and the distal portion formed a large false lumen. The aneurysm was repaired, the tricuspid aortic valve replaced, and a right coronary artery bypass graft performed. The patient recovered uneventfully after control of his hypertension. Pathological examination of the resected aortic leaflets showed myxomatous changes.

Results

The M-mode echocardiographic study disclosed a hyperdynamic, concentrically hypertrophied ventricle with a shortening fraction of 25 per cent and an ejection fraction of 49 per cent. At the level of the aortic root the aortic diameter measured 4.3 cm (1.9 cm/m²) and the left atrium measured 2.3 cm (1.0 cm/m²). There was thickening of the right coronary valve cusp. Slight superior sweep of the transducer from this position showed further dilatation of the aorta to a dimension of 5.3 cm (2.3 cm/m²) with compression of the left atrium to 1.6 cm (0.7 cm/m²) (Fig. 1).

Inspection of the aortic valve cusps at this level showed the presence of a structure (curved arrows) which lay posterior to the non-coronary cusp (straight arrows) and generally paralleled its motion. The onset of its end-systolic anterior motion was slightly delayed relative to the true cusp and it appeared to join both aortic cusps in diastole.

The genesis of this apparent "duplication" of the non-coronary cusp can be understood by inspection of the two dimensional long axis echocardiograms recorded within minutes of the M-mode tracings. Fig. 2 shows a systolic frame from a videotape of the dilated aortic root, left ventricle, and compressed left atrium. A line is superimposed on the sector scan to show the angle of incidence of a single M-mode beam which would produce the systolic M-mode image.

The origin of this line does not pass through the apex of the sector scan because of slight alteration in transducer placement between M-mode and two dimensional recordings. The flap of torn intima (arrows) can be seen behind the non-coronary cusp extending cephalad in the dilated ascending aorta. Fig. 3 shows the same view in diastole with the flap of torn intima (arrows) now having prolapsed on to the closed aortic leaflets (thicker arrow), which would produce the M-mode appearance of conjunction of the "duplicated" leaflet with the true aortic leaflets.

Discussion

M-mode echocardiographic criteria for dissecting aneurysm of the ascending aorta as proposed by Nanda et al. include: (1) dilatation of the aortic root (> 42 mm); (2) separation and widening of the anterior aortic wall (16 to 21 mm) and/or posterior
“Duplication” of aortic cusp

Fig. 2 Long axis view two dimensional echocardiogram in systole. The arrows indicate the torn intimal flap. The long white line indicates a single M-mode beam which would produce the systolic M-mode image. LV, left ventricle; LA, left atrium; A, aortic lumen.

aortic wall (10 to 13 mm); (3) parallel motion of the separated walls. All three criteria are required for the diagnosis. Fluttering of the anterior mitral leaflet (caused by aortic regurgitation) and pericardial effusion are supportive as signs of complications of dissection. Brown et al. have emphasised that these criteria can lead to false positive diagnoses unless anterior aortic wall thickening is present and the patient is clinically suspected of having dissection. Conversely, false negative echo studies may result from asymmetric dissections or dissections in the adventitial space surrounding the aorta. Among commoner causes of a false positive diagnosis are dilatation of the ascending aorta and non-specific thickening or calcification of the aortic root.

De Maria et al. have recently reported on the superiority of two-dimensional echocardiography to M-mode for the detection of aneurysm of the ascending aorta. Nevertheless, M-mode evidence of aortic enlargement was found in 11 of their 12 cases. Only one of their three patients with aortic dissection had M-mode findings consistent with this diagnosis. To our knowledge there is no information based on a large number of cases on the sensitivity of M-mode echocardiography in diagnosing this disorder. The case under discussion did not meet the second criterion of Nanda et al.

M-mode echocardiography remains a useful, relatively inexpensive, and readily available technique in clinical cardiology. The apparent “duplication” of the non-coronary cusp detected by M-mode study, shown to be caused by the motion of a flap of torn intima by two-dimensional echocardiography, is a new finding in dissecting aneurysm of the ascending aorta. Though this finding appears to be highly specific for this diagnosis it is conceivable that a mobile vegetation or clot at the base of the aorta in proximity to an aortic leaflet might produce similar appearances. Such a finding, however, has never been reported to our knowledge. Diagnostic confusion is also unlikely because of clinical and other echocardiographic characteristics of such lesions. It is likely that similar “duplication” of the right coronary cusp may be produced by anterior dissection of the aorta. Thus we have identified a new M-mode echocardiographic finding in aortic dissection which is probably highly specific. Its sensitivity awaits further study.

References

Fig. 3 Long axis view in diastole. The arrows point to the intimal flap with the thicker arrow indicating its contact with the conjoined true leaflets. Abbreviations as in Fig 2.


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