Echocardiographic Mimics of Aortic Root Dissection*

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Echocardiograms were recorded in two patients with suspected aortic root dissection. In the first patient with a massive pulmonary embolus, a simultaneous recording of the mitral ring and posterior aortic wall created a pattern similar to true posterior aortic wall dissection; in the second patient with severe generalized atherosclerosis, thickened aortic walls recorded echocardiographically were found at autopsy to be atherosclerotic plaques. These cases emphasize the potential for the false positive ultrasonic diagnosis of aortic root dissection.

We recently had the opportunity to examine echocardiographically two patients with suspected aortic root dissection. In the first patient the clinical diagnosis of dissection was rejected only after analysis of the complete echocardiographic scan, and the absence of dissection was confirmed at necropsy. In the second, aortic root dissection was strongly suspected clinically and radiographically, suggested echocardiographically, but absent at necropsy. It is the purpose of this report to emphasize the potential for the false positive echocardiographic diagnosis of aortic root dissection.

In both of these acutely and severely ill patients, the echocardiogram was accomplished in the Intensive Care Unit using a 2.25 mHz unfocused transducer and an Ekoline 20 echocardiograph coupled to a Cambridge strip-chart recorder. The technique for continuous M-Mode scan was employed.¹

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In the first case, the heart assumed a horizontal position in the chest. In order to obtain a technically complete scan, the transducer was placed higher and more lateral than usual (in the third left interspace approximately 4 cm to the left of the sternal border).

The echocardiogram revealed a small amount of pericardial fluid and right ventricular chamber enlargement (2.9 cm). The aortic root diameter was well within normal limits (2.8 cm). However, in the area of the posterior aortic wall, there were two parallel pulsating structures. These superficially simulated the walls of a false lumen of an aortic root dissection. However, on careful inspection of the scan from the aorta to the mitral valve, it became apparent that the more anterior of these two parallel echoes was recorded from the mitral annulus or mitral valve near the annulus. As the scan was continued up into the ascending aorta, this anterior echo disappeared abruptly and the true posterior aortic wall was recorded alone (Fig 1).

The apparent discontinuity between the posterior aortic wall and the mitral valve resulted from damping the low intensity echoes from the free edge of the mitral valve. At a higher gain setting or with the transducer placed more medially on the chest wall, direct continuity between the mitral valve and posterior aortic wall was established.

Approximately 30 minutes following the completion of the echocardiogram, the patient expired. At autopsy a massive pulmonary embolus occluded the right pulmonary artery. No aortic root dissection was present.

In the second patient, the aorta was echocardiographically dilated, measuring 4.8 cm with a central lumen of 2.4 cm. The anterior and posterior aortic walls were thickened measuring 16 mm and 8 mm respectively (Fig 2). These findings were thought to confirm the clinical diagnosis of aortic root dissection. Other possible explanations were aortic wall thickening secondary to aortitis (such as in syphilis) or atherosclerosis, but they did not fit the clinical situation as well. Drug treatment was implemented and hypertension brought under control. Five months later severe hypertension (300/190 mm Hg) brought the patient back to the hospital, renal failure dominated the picture, and the patient died. At autopsy both renal arteries were severely narrowed by atherosclerosis. There was marked diffuse atherosclerotic thickening of the aortic wall throughout the length of the aorta. At the level of the aortic valve, a large focal through and through plaque measuring 13 mm in thickness was present (it extended into the aortic lumen and through the aortic wall to form a localized convexity on the external surface). No aortic root dissection was present.

DISCUSSION

Recently several reports have shown the value of echocardiography in demonstrating dissection of the aortic root.²⁻⁵ Nanda et al⁶ correctly diagnosed six patients with aortic root dissection. All showed marked parallel widening of the anterior (16 to 21 mm) and/or posterior (10 to 13 mm) aortic walls together with enlargement of the aortic root image.
While five of these patients had dissection involving the posterior wall, in only one case was the dissection localized to the posterior wall.

The normal embryonic fibrous continuum between the anterior leaflet of the mitral valve and the posterior wall of the aortic root forms the anatomic basis for the echocardiographic differentiation of an intact aortic root from dissection of the posterior aortic wall. A scan showing echocardiographic continuity of these structures would imply anatomic fibrous continuity and absence of aortic root dissection. However, demonstration of discontinuity and separation of the posterior aortic root echo into two parallel echoes would suggest posterior dissection.

In our first patient, if one examines only that portion of the scan taken at the level of the aortic

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valve, the two echoes in the area of the posterior aortic wall simulate true dissection of this wall. However, by using the scan technique, it was possible to demonstrate that the more posterior of the two parallel echoes represented the true posterior aortic wall and the more anterior echo represented the mitral ring or the mitral valve near the ring. Clues to the correct identification of the mitral ring echo are direct continuity with anterior mitral valve leaflet and abrupt loss of the echo as the scan is carried into the ascending aorta.

In true dissection, as the scan is carried into the aortic root, the single echo of the mitral annulus appears to bifurcate into the sides of the dissected aortic wall. The aortic root image has been characteristically enlarged in all previous echocardiographic reports of aortic dissection. The lack of this characteristic feature, as is seen in our first patient, may be helpful in excluding a dissection.

The ability to record the mitral ring and posterior aortic wall simultaneously is explicable on the basis of the width of the sound beam. Structures which are anatomically continuous yet angulated so that echoes can be reflected from different depths may appear in an anterior posterior relationship on the echocardiogram (Fig 3). The picture termed “false dissection” can frequently be created even in a normal aorta simply by technical factors such as transducer position and angulation or by changing damping controls. This resultant echocardiogram may mistakenly be interpreted as aortic root dissection unless an appropriate scan is carried out.

The thickened aortic walls in our second patient resulted from diffuse atherosclerosis. In addition, the sound beam traversed a large focal anterior wall plaque. By using the strip chart recording technique and constant transducer position, only a small area of the aorta was recorded. If one extrapolates this picture (Fig 2) to the entire aorta, an overestimation of aortic narrowing is created. Anatomically, no significant luminal obstruction was present. Detection of atheromatous plaques by ultrasound has not been previously reported. Echocardiographic thickening of the aortic wall previously reported as diagnostic for dissection, now appears consistent with atherosclerotic thickening as well.

In summary, presented are two patients with the false positive echocardiographic diagnosis of aortic root dissection. It is concluded that the echocardiographic pattern formerly believed to be diagnostic for aortic root dissection is not absolutely diagnostic; it may be simulated by technical artifact or anatomic thickening of the aortic wall by causes other than dissection.
Uncommon Conditions of the Diaphragm

Of the vertebrates only mammals have diaphragms completely separating the body cavities. The diaphragm is an unusual muscle structurally, functionally as well as topographically. With its tendinous center it serves not only as a partition between the thoracic and abdominal cavities but also its utilitarian function is of importance in respiration, vocalization (screaming, singing), cough, hiccup, sneezing, yawning, urination, defecation and in the treatment of “cafe coronary” by the recently described Heimlich maneuver (mechanical expulsion of aspirated food from the trachea by forcing the diaphragm upward while the patient is slumped forward) (JAMA 239:746, 1974). Each of the hemidiaphragms is innervated by the phrenic nerve and occupies an asymmetrical position. Several ingenious but unproved theories have been offered to explain this positional discrepancy. Mandelstamm, M et al (Ergeb Int Med Kinderheilk 34:154, 1928), Roessler J (Wien Arch Inn Med 19:505, 1930), Lichtman, S S (Arch Int Med 48:866, 1931), and Carlson, H C et al (Proc Staff Conf Mayo Clin 37:25, 1962) attributed the lower position of the left hemidiaphragm to the weight of the heart. The authoritative investigation of Wittenborg, M H et al (Brit J Radiol 36:280, 1963) led to the conclusion that there was no evidence in favor of the assertion that the mass and the activity of the heart depress the respective hemidiaphragm. It might be worthy to consider the theory that the heart reduces the volume of the lung ipsilaterally and decreases the negativity of the intrapleural pressure in relation to the opposite side; this results in decreased upward traction upon the respective hemidiaphragm. Diaphragmatic flutter is a most unusual functional disturbance. It is likely to be associated with chest pain of 1 to 3 hours duration and recur at irregular intervals. The rhythmic diaphragmatic contractions are readily visualized by fluoroscopy and audible throughout the chest. Their frequency varies from 240 to 250 per minute, with 2 to 5 second intervals. Despite the flutter, the function of the diaphragm remains normal. In some instances, the flutter was attributed to rheumatic fever or epidemic encephalitis, in others its cause remained undetermined. Scheffley, C W et al (Ann Int Med 26:129, 1947) observed a case of tonic spasm of the diaphragm, associated with knife-like pain in the lower part of the left hemithorax, which radiated to the left shoulder. In instances of incomplete embryonic descent of a hemi-diaphragm which consists mostly of fibrous tissue and insufficient muscular elements, its respiratory movements are reduced or absent. Such circumstances permit a cephalad shift of some of the abdominal structures. Several authors reported instances of accessory hemidiaphragm, in which one entire lobe or a portion of it was entrapped between the true and the accessory hemidiaphragms. Dystrophia myotonica, a hereditary disease transmitted as an autosomal dominant, may be associated with atrophy, inadequate movements and high position of the diaphragm, with consequent hypventilation and basal atelectasis. Shafer, J O (JAMA 188:1000, 1964) reported a case of congenital absence of the left hemidiaphragm, with multiple abdominal viscera filling the respective hemithorax completely. Follow-up for 27 months attested to the successful use of a knitted Dacron prosthesis hemidiaphragm. Large series of routine chest x-rays reveal an incidence of entration of the diaphragm from 1 to 3 per 10,000. More appropriately this term should be changed to ectopia of the diaphragm. It is regarded as congenital malposition of this structure. In rare instances, it may be brought about by phrenic nerve injury during birth or caused by neoplasm or infection. Infrequently, sudden sharp increase in the intrapitoneal pressure by severe trauma or by prolonged strenuous childbearing may be the causal factor. Unilateral paralysis of the diaphragm as one of the manifestations of herpetic zoster was reported first by Halpern, S L et al (Arch Int Med 84:907, 1949). Subsequent sporadic reports confirmed the occurrence of this clinical entity. Only 84 primary neoplasms of the diaphragm were reported during a hundred year period according to Olafsson, G et al (Chest 59:568, 1971). Benign tumors include angiolipoma, angiofibroma, chondroma, fibroma, fibromyoma, fibroangioidothelioma, hemangioidothelioma, lymphangioma, myoma, neurofibroma, rhabdomyofibroma. The following malignant tumors have been recorded: endothelial sarcoma, fibromyosarcoma, leiomyosarcoma, mesothelioma, myosarcoma, neurofibrosarcoma, polymorphous sarcoma, round cell sarcoma, rhabdomyosarcoma, undifferentiated sarcoma. No encyclopedic coverage of the subject has been contemplated within the framework of this presentation.

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