Aortic aneurysm caused by schistosomiasis

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There has not been a previous report of schistosomiasis producing an aortic aneurysm. There is one report of schistosomiasis producing a pulmonary artery aneurysm, in which the author suggests possible mechanisms for such dilatation of the pulmonary artery. In the case reported here extensive infestation of the left upper lobe by ova of Schistosoma mansoni caused extension of the disease into the adjacent pleura and aortic sheath, producing endarteritis obliterans of the vasa vasorum of the aorta and subsequent formation of an aneurysm.

Case report

A well nourished 19 year old African woman was admitted to hospital with haemoptysis, left sided chest pain, and hoarseness of the voice of one month's duration. There was no hepatosplenomegaly and the left branchial pulse was absent. There was a soft systolic bruit in the left upper chest, and an opacity within the left upper lobe was demonstrated by plain radiography (fig 1). There were no acid fast bacilli in the sputum and a Mantoux reaction was grade 1. There was no history of trauma, and all other pulses were present and equal. Computed tomography showed a mass in the left upper lobe, inseparable from the aortic arch and descending thoracic aorta (fig 2). On enhancement with intravenous Conray 325 at least part of the mass was shown to contain blood in continuity with the aortic lumen. Aortography showed an arch aneurysm.

The haemoglobin concentration was 9.7 g/dl, and the white blood cell count 16×10^9 /l with an eosinophilia of 10%. There was blood in the urine but no ova of *Schistosoma* were identified. Stools showed the ova of *S mansoni*, as did a biopsy specimen of the rectal mucosa. Serological tests for syphilis (Venereal Disease Research Laboratory, Wassermann reaction) were negative. The results of liver function tests, including the prothrombin index, were normal.

A left thoracotomy was made and extended transsternally to the right inframammary fold. The left upper lobe was firm, shrunken, and airless, and densely adherent to the aneurysm. There was a granulomatous inflammatory reaction in the lung, extending through the pleura and inseparably merging with abnormal aortic wall. The aorta was aneurysmal from 5 cm proximal to the brachiocephalic ar-

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tery to the descending aorta at the level of the 5th thoracic vertebra. Repair of the aneurysm was undertaken with cardiopulmonary bypass and profound hypothermia (17°C), with circulatory arrest of 15 minutes' duration. The visible part of the aneurysm consisted mainly of a clot filled false of aneurysm, which communicated with the aorta through a defect in the arch at the level of the left subclavian artery. The defect was oval, measuring 7 × 2 cm with firm fibrous The defect was closed by direct suture, with reinforcement by closure of the false aneurysm. The post-operative aortogram was normal.



Fig 1 Chest radiograph on admission to hospital.

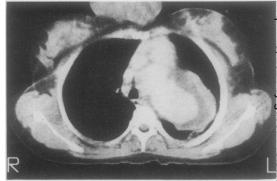


Fig 2 Computed tomographic section of chest.

Discussion

The parasite responsible for this disease is the trematode fluke, which lives in blood vessels of the human host and passes the intermediate (miracidial, redial, and circarial) stages of its life cycle in the fresh water snail.

The pulmonary manifestations of schistosomiasis have been well described.² The route by which both eggs and flukes reach the pulmonary circulation is not known with certainty, and several possible routes have been postulated.³ Live worms cause little or no reaction within the vessel wall but produce large numbers of eggs, which cause a range of pulmonary changes. Dead worms, however, produce severe toxic reactions, including thrombosis of the artery and necrosis of surrounding lung tissue.

It has been estimated that in 65% of cases of *S mansoni* infestation the lungs are affected,⁴ and this occurs in the absence of appreciable hepatic infestations.

Eggs lodge in branches of the pulmonary arterioles ranging from 50 to 100 μ m in diameter, where they cause an intravascular and perivascular granulomatous reaction. The arteriole becomes surrounded by histiocytes, giant cells, eosinophils, and lymphocytes, which produce the typical schistosomal granules that eventually destroy the whole or part of the vessel wall. Enzymes and antigenic substances are released from the eggs, which sensitise the host lymphocytes; these then migrate to the areas of egg deposition. Other cells

are recruited through secretion of lymphokines and result in a compact cellular infiltrate. Raised concentrations of IgG, IgM, and IgE and specific antischistosomal antibodies are produced, suggesting that immune complex reactions contribute to cellular damage.⁵

Histological examination confirmed a granulomatous reaction within and surrounding the lung, with histocytes, giant cells, eosinophils, lymphocytes, and the ova of S mansoni. Eggs were found in large numbers within the lung tissue and outside the visceral pleura within the aneurysm wall. Numerous eggs were seen even in the outer layers of laminated thrombus in the aneurysm. We believe that schistosomiasis was the cause of the aortic aneurysm in this case.

References

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