A 51-year-old woman originating from Africa presented to our institution with newly intermittent claudication, headache, and dyspnea associated with a 4-month history of weight loss and tiredness. Clinical examination showed hypertension and a marked pressure gradient between the upper and lower limbs (140/80 versus 80/50 mm Hg). There was no sign of digestive claudication or renal insufficiency.

Computed tomography angiography demonstrated an 80% segmental stenosis of the descending thoracic aorta associated with aortic wall thickening >95 mm in length (Figure A), together with pulmonary infiltrates and cervicomediastinal lymphadenopathies. Combined positron emission tomography-computed tomography showed a hypermetabolism of the aortic thickening (Figure B) and cervicomediastinal lymphadenopathies. The surgical biopsy of a cervical lymphadenopathy showed the presence of granuloma consistent with a tuberculosis infection.

The diagnosis of acquired coarctation attributable to the aortic involvement of disseminated tuberculosis was confirmed by a multidisciplinary team, and medical treatment with quadruple antituberculosis therapy was decided. After 12 months of antituberculosis therapy, the respiratory symptoms regressed, positron emission tomography-computed tomography activity disappeared (Figure D), but aortic thickening remained stable. A surgical biopsy of the aortic wall was performed through left minithoracotomy. Pathology showed the presence of inflammatory fibrotic tissue but the absence of Mycobacterium tuberculosis. Steroids were initiated and decreased in the aortic wall thickness. Area measurements of thoracic aortic lumen showed an increase from 14 (Figure A) to 25 mm² (Figure C). Antihypertensive therapy was lowered to a monotherapy 6 months after the initiation of steroids. The patient is being treated further by steroids 22 months after the diagnosis. She presents no symptoms of recurrent claudication.

Tuberculous aortitis is a rare entity that is invariably indicative of disseminated tuberculosis. It can involve the abdominal or thoracic descending aorta with equal frequency. Involvement of the ascending aorta and aortic arch is less frequent.1,2 The close spatial relationship between the thoracic aorta and the previously infected pulmonary segment might suggest a direct extension of the tuberculosis from the lung to the aorta. However, blood-borne seeding through the vasa vasorum have also been advocated,2 with no definitive evidence favoring 1 hypothesis over the other. Tuberculous aortitis might be associated with aneurysmal or stenotic aortic disease.3

Tuberculous aortitis is usually associated with the formation of a pseudoaneurysm that may lead to aortic rupture. Aneurysmal disease usually requires surgery by aortic bypass grafting.1,2 In the case of rupture, endovascular repair with a stent graft might be an option. A few years ago, endovascular repair was preferentially performed as a bridge to open surgery, because graft infection and secondary rupture have been described.4 The situation is evolving, because prolonged remission following endovascular repair has also been reported.

Only a few cases of tuberculosis aortitis causing coarctation have been reported to date.1 Most of these cases presented with symptoms of aortic coarctation. Surgical management has been constantly advocated in the literature, with endovascular and open approaches being equally associated with long-term remission. However, endovascular repair could not be used in our case because of the extension of aortic stenosis, and open surgical repair is associated with a high rate of major postoperative complications. We therefore advocated a medical treatment combining first-line antituberculosis quadritherapy followed by adjuvant steroid treatment, leading to disease remission and symptoms control.

In conclusion, the diagnosis of tuberculous aortitis should be considered in the case of acquired aortic coarctation, especially in patients with a history of recent pulmonary tuberculosis. Surgery is not always necessary in the case of stenotic lesions, and the disease might be successfully treated by medical approaches.


Figure. A, PET scan showing hypermetabolism of aortic thickening. B, CT angiography showing long stenosis of descending thoracic aorta with wall infiltration. C, disappearance of hypermetabolism after antituberculosis treatment on PET scan. D, reduction of aortitis with increase of aortic lumen diameter. CT indicates computed tomography; and PET, positron emission tomography.