Reconstruction of Heavily Calcified Aorta and Its Visceral Branches without Extracorporeal Circulation

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SUMMARY

A 61-year-old Japanese female was referred to our hospital for surgical treatment of a localized heavily calcified abdominal aorta. Preoperative angiograms and computed tomograms revealed severe stenosis of the aorta, resembling a slit. Bypass grafting between the thoracic and abdominal aorta was successfully performed together with the reconstruction of the celiac artery, superior mesenteric artery, and bilateral renal arteries without extracorporeal circulation. Postoperative angiograms showed patency of the graft and branches. A localized heavily calcified abdominal aorta is relatively rare, and the cause of this entity might be Takayasu's aortitis. (Jpn Heart J 2001; 42: 651-655)

Key words: Aortitis syndrome, Aortic calcification, Grafting

CASE REPORT

A 61-year-old Japanese female visited a local hospital complaining of lumbago in December, 1996. Chest and abdominal roentgenograms and computed tomograms (CT) revealed a localized heavily calcified aorta. The internal space of the aorta was severely stenotic, and there appeared to be a slit (Figure 1-A, 1-B, 1-C, 2). The patient had no particular family history, but had undergone previous operations for glaucoma and cataracts at the ages of 49 and 52 years old, respectively. The patient had been medicated for hypertension since 60 years of age. Preoperative values of laboratory examinations such as serum calcium, phosphorus, C-reactive protein, immunoglobulin, parathyroid hormone, calcitonin, nitric oxide (NO), and interleukin-6 (IL-6) were within normal limits. Preoperative angiograms revealed that the celiac artery (CA), superior mesenteric artery (SMA) and bilateral renal arteries (RA) were not stenotic, but that the thoraco-abdominal aorta itself was severely calcified and stenotic (Figure 3). Renograms demonstrated sufficient renal blood flow, and ankle brachial pressure
Figure 1-A, 1-B: Chest X-ray showing a localized heavily calcified aorta. 1-C: Abdominal X-ray showing a localized heavily calcified aorta.

Figure 2. Computed tomograms (CT) show that the internal space of the aorta is severely stenotic, which appears to be a slit and is almost obstructed.

indexes were slightly reduced (rt.; 88%, lt.; 85%). In order to avoid the possibility of a fatal aortic obstruction, a bypass operation between the thoracic and abdominal aorta with reconstruction of the CA, SMA, and bilateral RAs was performed in July, 1997.
With a Stoney's spiral incision associated with a left thoracotomy through the 5th intercostal space, the thoracic and abdominal aorta were approached via a retroperitoneal route. Despite marked calcification of the aorta at the level of the diaphragm, the proximal and distal aorta were not calcified. End to side anastomoses were performed by partial clamping of the aorta between the descending thoracic aorta and the abdominal aorta using a branched woven Dacron prosthesis (GELWEAVE®, φ 22mm ). Extracorporeal circulation (ECC) was not used. The visceral branches such as CA, SMA and RAs were also reconstructed with end to end anastomoses.

The resected aortic wall showed diffuse thickening of the intima which contained lipids and calcification, however, atheroma plaques were not found in the pathological study. The postoperative course was uneventful and the patient was discharged on the 27th postoperative day. Postoperative angiograms showed patency of the graft and visceral branches (Figure 4). Three years after surgery, the patient has been well without any problems.
Figure 4. Postoperative angiogram showing patency of the graft and visceral branches. CHA; common hepatic artery, SA; splenic artery, SMA; superior mesenteric artery, R-RA; right renal artery, L-RA; left renal artery.

DISCUSSION

Variable causes of aortic calcification have been reported. The development of atheroma brings about an extensive destruction of elastic fibers and muscular cells, and subsequently, other components of the extracellular matrix such as collagen, nonuronic sugars, water, and lipids are increased and occupy their places. Remarkable calcification occurs in this way.1-3) This is probably due to the fact
that aging and the development of atherosclerosis bring about an increase in elastase activity. Hypercholesteremia and hyper-low density lipoprotein (LDL) -cholesteremia, hypertension, obesity and aging may also promote calcification of arteries, and many complicated mechanisms are involved altogether. In our patient, only moderate hypertension was present. The extent of calcification was limited to an extremely narrow segment of the aorta, suggesting that the cause of calcification might not be atherosclerosis. On the other hand, Ohyama, et al reported that plasma levels of NO$_2^-$/NO$_3^-$ and IL-6 were elevated in patients on maintenance hemodialysis, and were significantly correlated with the aortic calcification index (ACI) and the ankle brachial pressure index (API). They proposed an underlying mechanism involving increased production of NO to be intimately associated with atherosclerotic change. Koh, et al reported that hypercalcemia, reduced levels of serum VitD$_3$ concentration, and increased levels of parathyroid hormone and calcitonin were relevant to aortic calcification. In our patient, these parameters were within normal limits.

A chronic change of the post-inflammatory state of atypical aortitis syndrome, which may induce coarctation of the thoraco-abdominal aorta, is strongly suspected as the cause of aortic calcification in our patient. Histological findings of the aortic wall were consistent with the characteristics of arteriosclerosis in aortitis syndrome characterised by transmural inflammation and fibrosis.

ECC is often necessary at the time of surgical treatment for a thoraco-abdominal aorta. In our patient, because the proximal and distal aorta were not calcified, bypass grafting was successfully performed under partial clamping of the aorta.

REFERENCES