disease and the coronary arteries were not affected. On day 24 of the illness, however, mitral regurgitation with an audible murmur occurred suddenly, resulting in the rapid development of severe heart failure. Doppler echocardiography demonstrated prolapse of the posterior leaflet of the mitral valve, which before operation was attributed to rupture of the papillary muscle in the setting of recurrent myocarditis. Rupture of the chordae tendineae was confirmed at the time of operation; however, there was no evidence of valvulitis. The valve was successfully repaired by mitral valvuloplasty with total circular annuloplasty.

Results of this case suggest that initial mild mitral regurgitation may be attributable to papillary muscle dysfunction caused by recurrent myocarditis. Severe mitral regurgitation, however, may be caused by chordal rupture that occurs because of functional impairment of the valve apparatus. This is the first report of mitral regurgitation resulting from ruptured chordae tendineae in a patient with Kawasaki disease. Evaluation of similar patients will be necessary to investigate in greater detail mechanisms responsible for chordal rupture.

REFERENCES

LATE FAILURE OF INTERNAL THORACIC ARTERY GRAFTS CAUSED BY SEQUELAE OF MEDIASTINITIS OR ITS TREATMENT

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Media stinitis is a severe complication after cardiac operations, leading to higher morbidity and mortality rates and to greatly increased hospital costs. The risk for erosion and hemorrhage of bypass grafts and the heart is known, but it is generally accepted that mediastinitis does not affect long-term graft patency. The following case report, however, is suggestive of occlusion of internal thoracic artery (ITA) grafts as a late result of mediastinitis or its treatment.

In 1987, a 56-year-old man underwent elective primary myocardial revascularization with both ITAs: left ITA to the diagonal branch (side to side) and left anterior descending coronary artery (end to side), the right ITA, retroaortic to the circumflex coronary artery, and a single vein graft to the right coronary artery. The first postoperative day, the patient was extubated and discharged from the intensive care unit. Six days after operation, the patient had an unstable sternum. There were no signs of infection (fever, leukocytosis, wound drainage), and a refixation was performed. Four days later, the patients was septicemic, with a temperature of more than 40°C and leukocytosis. The sternal wound was producing purulent fluid. Antibiotic therapy was continued. There was a good response to this therapy. A clean wound developed, and secondary closure of the wound was performed 22 days after the last intervention. The patient was discharged from the hospital after 38 days. The wound was healed without evidence of infection. The patient was seen regularly in the outpatient clinic. There were no wound problems. In 1989, the patient reported atypical retrosternal pain. An angiogram showed three patent grafts. In January 1995, the patient reported angina (Canadian Heart Association class 2 to 3) and received medical
therapy with nitrates, calcium-channel blockers, and β-blockers. Despite this medication, angina was progressive and a new angiogram was performed. This angiogram showed a diseased coronary artery system and a patent but diseased venous graft to the right coronary artery. The right and the left ITAs were both occluded (Figs. 1 and 2) several centimeters below their origin.

A coronary reoperation was performed. A median sternotomy was performed. There were no signs of infection; the greater omentum was hard and encircled the heart like an eggshell. After this “eggshell” was split, the heart was free. After adequate heparinization, extracorporeal circulation was started, and the patient was cooled to a core temperature between 26°C and 28°C. Myocardial protection was accomplished with retrograde infusion of cold (4°C) blood cardioplegia. A venous graft was placed to the diagonal branch and the left anterior coronary artery, a venous graft was placed to the circumflex coronary artery, and a new venous graft was also placed to the right coronary artery. The postoperative course was uncomplicated.

During the operation, both ITA pedicles were identified and visualized up to their origin. Intrapericardially (intrapericardially), the ITAs were occluded. Extrapericardially (extraorally), however, both pedicles were patent, as shown by the angiogram.

In this patient, the return of angina 8 years after the operation and the angiographic and clinical findings of an occlusion of the ITA grafts at the entrance to the pericardium (omentum) were suggestive of late occlusion of the ITA grafts as a result of the previous mediastinitis or the treatment with the wrapped greater omentum. The treatment of mediastinitis with a pedicled greater omental flap, richly vascularized and highly immunoreactive, is well known. Omentum’s qualities in protecting infected organs and vascular or even prosthetic grafts has been proved. In our search in the literature, no information could be found about late complications leading to strictures obstructions. In abdominal surgery, however, adhesions between abdominal organs and omentum eventually leading to bowel obstruction are well known. Some studies suggest a role of infection in the development of late postoperative adhesions.

In our opinion, this late ITA graft failure from an occlusion just at the entrance to the pericardium or wrapped omentum can only be the result of a late stricture formation of the omental flap and should be considered a late graft failure from mediastinitis.

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