Fatal Cerebral Embolism Following Aorto-Coronary Bypass Graft Surgery

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SUMMARY In a case of fatal cerebral embolic infarction following aorto-coronary bypass graft (ACBG) surgery, postmortem examination revealed thrombosis of the vein grafts to the left circumflex and left anterior descending coronary arteries. Contiguous with the thrombus in the graft to the circumflex artery was thrombotic material adherent to the aortic sutures and extending several millimeters into the lumen of the aorta. A nonadherent thrombus of similar histologic character was found in the right middle cerebral artery, associated with localized brain infarction. In addition to the risks of cerebral complication associated with other types of open-heart surgery, the location of the vein grafts in patients undergoing ACBG operations seems to offer a unique mechanism for the occurrence of systemic and cerebral embolism, which may be operational in other cases.

CEREBRAL COMPLICATIONS constitute a major hazard of open-heart surgery for repair of congenital abnormalities or valve replacement. One retrospective study of 417 cases indicated that 19.2% of patients had impairment of consciousness, voluntary movement, or vision within the first three postoperative days.1 Prospective studies specifically searching for neurologic abnormalities suggest the rate to be even higher: Tufo et al.2 reported their presence in 44% of patients surviving the operative period. Operative mortality in their series was 15%, and it is likely that cerebral complications contributed to that mortality.

In contrast, the incidence of cerebral complications following aorto-coronary bypass graft (ACBG) surgery is reported to be quite low. Assad-Morell et al.3 mentioned only one instance of neurologic abnormality (cerebral infarction) in their follow-up of 500 patients from 18 to 42 months after surgery, and Sheldon and Grinfeld4 made no mention of neurologic complications in any of their 300 patients followed up for 21 to 46 months. This is surprising, because — although the risk of air embolism is much less during ACBG operations — other pathogenetic mechanisms implicated in the production of cerebral symptoms during open-heart surgery (i.e., hypotension, electroencephalographic monitoring, and use of Millipore filters) to reduce the incidence of cerebral injury at operation, the reported incidence of cerebral complications following open-heart surgery in one series still is 7.4%.5

The following case is an example of fatal cerebral embolism following ACBG surgery and illustrates a pathogenetic mechanism, unique to this type of surgery, which may be operational in other cases.

Report of Case

In April 1975, a 45-year-old white man was admitted to the Mayo Clinic for evaluation of intractable angina pectoris. All results of a complete physical examination and routine laboratory studies, including electrocardiography, were normal. The patient was admitted to the hospital, where coronary angiography showed widespread moderately severe disease. The right coronary artery was occluded at the junction of the proximal and middle thirds, and the marginal branch of the left coronary artery was narrowed by 60% to 70% at its origin and by 70% to 80% more distally. The main left anterior descending branch was normal; but the continuing portion was narrowed by 90% and 85% at two places, the distal segment being normal.

An ACBG procedure was performed, which included saphenous-vein grafts from the aorta to the distal right coronary, left anterior descending, and left circumflex arteries. Total pump time was 132 minutes. No complications were noted during the procedure or in the immediate postoperative period. Intermittent heparin was given intravenously as a routine precaution against peripheral venous thromboembolism.

On the eighth postoperative day, the patient noted the onset of left-sided visual field loss, which was defined by perimetric testing as left inferior quadrantanopia (fig. 1). Intravenous administration of heparin was continued, and no further symptoms were noted until postoperative day 13, when left hemiparesis developed and progressed rapidly to hemiplegia, accompanied by a progressive decline in level of
consciousness. Computerized transaxial tomography showed decreased density of a large portion of the right cerebral hemisphere (fig. 2) and a marked shift of midline structures. The findings were thought to be indicative of a large cerebral infarct with associated edema. Despite intravenous administration of dexamethasone and glycerol, the decrease in the level of consciousness continued. Signs of uncal herniation became evident, and death occurred 24 hours after the onset of the hemiparesis.

Postmortem examination revealed thrombosis of vein grafts to the left circumflex and left anterior descending coronary arteries. Contiguous with the thrombus in the graft to the left circumflex artery, there was thrombotic material adhering to the aortic sutures and extending several millimeters into the lumen of the aorta (fig. 3). The chambers of the heart were devoid of thrombus, and there was no evidence of recent myocardial infarction.

Examination of the cerebral blood vessels revealed a large nonadherent thrombus in the right middle cerebral artery just proximal to its trifurcation (fig. 4). The cerebral thrombus was similar in gross appearance to that at the orifice of the vein graft (fig. 5). The remainder of the cerebral blood vessels showed only mild atherosclerotic narrowing; there was no evidence of ulcerated atherosclerotic plaques in the extracranial vessels.

Examination of the brain parenchyma showed most of the right hemisphere to be softened by infarction (fig. 6).

Light microscopy confirmed the histologic similarity between the thrombus extending from the vein graft and that in the middle cerebral artery. In addition, the typical nonadherent character of the distal clot indicated its probable embolic origin.

**Comment**

This case is an example of fatal cerebral embolism occurring after ACBG surgery and illustrates a general mechanism of thromboembolism that seems a possibility in many post-ACBG patients. In addition to the risks of
A careful study by Grondin et al. has shown the one-year graft occlusion rate to be 20.4%. In view of this significant frequency of occlusion, it is somewhat surprising that more of these thrombi do not extend proximally and eventually give rise to emboli.

We believe that a more critical evaluation is needed — in the form of a prospective study of neurologic function following ACBG surgery — to ascertain the true incidence of cerebral complications following this procedure. In addition, postmortem examination in cases with cerebral infarction must include not only study of the cerebral vasculature but also a search for the pathogenetic mechanism responsible for the brain lesion. In the meantime, we urge physicians to be aware of the possibility of cerebral embolism that must exist in some post-ACBG surgery patients.
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Angiographical Extravasation of Contrast Medium in Hemorrhagic Infarction
Case Report
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SUMMARY Leakage of the contrast medium was noted on angiograms of a patient whose autopsied brain disclosed typical pathological findings of hemorrhagic infarction. The case was a 63-year-old woman with mitral valve failure, who suddenly had loss of consciousness and right-sided hemiplegia. The left carotid angiography performed six hours after onset demonstrated middle cerebral arterial axis occlusion, and the second angiography performed three days after onset displayed recanalization of the initially occluded artery as well as extravasation of the contrast medium. Fourteen days after onset the patient died and an autopsy was performed. The brain demonstrated perivascular punctate hemorrhages in the area supplied by the middle cerebral artery, and neither hematoma nor microaneurysm was disclosed pathologically.

A short discussion is given on the possible relationship between recanalization and hemorrhagic infarction. The clinical assessment of hemorrhagic infarction has not been established successfully.

ANGIOGRAPHICAL EXTRAVERSATION of the contrast medium in various kinds of intracranial hemorrhagic lesions has been reported previously by many authors regarding cases of hypertensive intracerebral hemorrhage, ruptured aneurysm, or arteriovenous malformation, and epidural hematoma.* However, as yet, no cases have been described concerning angiographical extravasation in patients with occlusive cerebrovascular diseases, in spite of the fact that hemorrhagic infarction develops frequently in the clinical course of such patients. The present case demonstrated leakage of the contrast medium on the angiograms obtained after spontaneous recanalization of the occluded middle cerebral arterial axis, and the autopsy brain displayed typical hemorrhagic infarction in the infarcted area. We believe this case represents the first angiographical demonstration of extravasation in hemorrhagic infarction.

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Fatal cerebral embolism following aorto-coronary bypass graft surgery.
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Stroke. 1976;7:611-614
doi: 10.1161/01.STR.7.6.611

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