Fatal Cerebral Embolism Following Aorto-Coronary Bypass Graft Surgery

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SUMMARY In a case of fatal cerebral embolic infarction follow-
ing aorto-coronary bypass graft (ACBG) surgery, postmortem ex-
amination 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 milli-
meters 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 oc-
currence 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 abnor-
nalities 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 in-
farction) 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, hypothermia, hypox-
ia, microembolization of particulate matter, etc.) are equally
operational during ACBG surgery.

Since a high proportion of the patients with recognized
neurologic impairment were normal by the time of dis-
mission from the hospital, studies such as those mentioned
above — which are based primarily on follow-up after
ACBG surgery — are likely to give a falsely low impression
of the incidence of cerebral complications.

Though neurologic abnormalities following ACBG sur-
gery have not been studied in detail, ophthalmologic com-
plications of the surgical procedure are well documented.
Vukov and Wayne5 reported a 12% incidence of ocular ab-
normalities in a prospective study, and Gutman and
Zegarra6 reported 17% in a similar study. Of the 150
patients included in those two studies, only one had
homonymous hemianopia or other defect suggestive of a

cerebral lesion. Apparently cerebral complications do occur,
but infrequently. Prospective studies that carefully assess
neurologic function outside the visual system would be ex-
pected to increase the number of cerebral complications
identified.

In spite of improved management (prompt treatment for
hypotension, electroencephalographic monitoring, and use
of Millipore filters) to reduce the incidence of cerebral injury
at operation, the reported incidence of cerebral com-
plications following open-heart surgery in one series still is
7.4%.7

The following case is an example of fatal cerebral em-
bolism following ACBG surgery and illustrates a patho-
genetic 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 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 cor-

onary, 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 in-
travenously 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 quadrant anopia (fig. 1). In-
travenous 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
cerebral complication associated with other types of open-heart surgery, the location of the vein grafts in these patients appears to offer considerable opportunity for systemic and cerebral embolism. The graft is sewn directly into the aorta, and the suture line is a possible site for thrombus formation. In addition, because the vein-graft lumen is in direct continuity with the aortic lumen, a thrombus forming within the vein could easily gain access to the systemic circulation by retrograde propagation to the vein-aorta anastomosis.

A carefully designed prospective 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.


FIGURE 5. Vein-graft thrombus. Hematoxylin and eosin; X16.

FIGURE 6. Ventral surface of brain, showing marked swelling of right hemisphere.
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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 angio-
grams 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 angiog-
raphy 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 hemmoraghes in the area supplied
by the middle cerebral artery, and neither hematoma nor microaneu-
rysm 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 EXTRAVASATION of the contrast
medium in various kinds of intracranial hemorrhagic lesions
has been reported previously by many authors regarding
cases of hypertensive intracerebral hemorrhage,1 ruptured
aneurysm,2 or arteriovenous malformation,3 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.8 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 autopsied brain displayed
typical hemorrhagic infarction in the infarcted area. We
believe this case represents the first angiographical
demonstration of extravasation in hemorrhagic infarction.

Case Report

A 63-year-old woman had a history of chronic heart dis-
 ease associated with mitral valve failure for more than ten
years previously. She was doing fairly well until she
suddenly lost the use of her right arm and leg and within 30
minutes had some confusion. On arrival at the hospital by
ambulance three hours after the onset, she was somnolent
and somewhat restless, with motor and sensory aphasia and
right-sided hemiplegia. Her blood pressure was 140/90 mm
Hg and her pulse was 68 and irregular. External evaluation
showed no injuries. Physical examination revealed a com-
plete right hemiplegia with a central facial palsy and aphasia
and positive Babinski sign. The arm and leg were equally
paralyzed, and only a slight degree of motion was preserved
in the shoulder and the hip joints. Pinprick sensation was
diminished on the right, but other sensory modalities seemed
to be intact. Auscultation of the heart disclosed diastolic
rumbling and the ECG showed atrial fibrillation, suggesting
the presence of mitral stenosis. The ocular fundi showed no
papilledema. Lumbar puncture showed colorless CSF and
the pressure was 140 mm H2O. Serial left carotid arteriog-
raphy, using 7 ml of 60% Amidotrizoate, demonstrated
occlusion of the middle cerebral artery of its proximal por-
Fatal cerebral embolism following aorto-coronary bypass graft surgery.
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