Air Embolus in Liver Transplantation

G. Mazzoni, L. Koep, and T. Starzl

AMONG adult recipients of orthotopic liver homografts, a high incidence of acute and profound neurologic disability has been recognized shortly after operation.[1-2] A clinical neuropathologic survey of these central nervous system complications has been reported in detail[2] and will be summarized here. Particular emphasis has been placed on the possible etiologic role of air emboli originating from the hepatic homograft and on the avoidance of such emboli.

MATERIALS AND METHODS

Forty-eight adult patients were treated between March 1963 and April 1976. Within the group of 48 adults, 9 patients sustained clinically significant neurologic damage during or soon after operation. All 9 patients had end-stage cirrhosis and evidence of portal hypertension. Three were alert just prior to operation. The other six had evidence of altered consciousness, ranging between confusion and coma. Postoperatively, all nine patients developed irreversible and widespread neurologic damage, which contributed to or caused death.

During the life of these patients, clear identification of the causative factors of these complications was not possible. Standard liver functions tests usually were not very abnormal. There was no evidence in any patient of early posttransplantation liver necrosis. Cerebral angiography in one patient suggested thrombotic or embolic disease. However, most coagulation factors in this and other patients, as exemplified by the prothrombin time, were only moderately subnormal. Cortical ischemia area with dying neurons and microcystic changes, gliosis related to demyelimination, capillary proliferation, injured neurons, and gliosis were noted in almost all our cases.

EVIDENCE OF AIR EMBOLUS

Although the neuropathologic changes could have been caused by air embolus, they were nonspecific. However, evidence of air embolus was definitely observed in some cases, including one of our early patients who developed bilateral partial leg paralysis. Another patient had a cardiac arrest from which he was promptly resuscitated. Air was withdrawn from a right atrial catheter immediately afterwards.

Another patient awakened promptly after transplantation and was completely normal for several hours. He then suddenly had a focal seizure, never regained consciousness, and died 2 days later. At autopsy, the cavernous sinus and several cerebral arteries contained gross air.

PATHOPHYSIOLOGY OF AIR EMBOLUS

Thus, cerebral air embolism, derived from residual air in the homograft vena cava or hepatic veins, was suspected to have been responsible for at least some of the neurologic complications. Appreciation of this concept has been slow and delayed by an imprecise understanding of how air could regularly by-pass the lung, go to the left heart and systemic circulation, and then proceed to the brain.

In actuality, the ability of the air to readily cross the lung barrier is understandable. Even in normal human lungs, potential connections exist between the pulmonary artery and venules. These anastomotic channels become patent under various conditions and particularly with chronic liver disease. In addition, communications may exist between the portal and azygos veins.

It appears reasonable to believe that air could easily make its way by anyone of several routes to the systemic circulation and lodge within the central nervous system, producing

From the Department of Surgery, Denver Veterans Administration Hospital and the University of Colorado Medical Center, Denver, Colo.

Supported in part by research grants from the VA Hospital, Denver, Colo.; by NIH Grants AM-17260 and AM-07772; and by Grants RR-00051 and RR-00069 from the General Clinical Research Centers Program of the Division of Research Resources, NIH.

Reprint requests should be addressed to T. Starzl, Department of Surgery, Denver Veterans Administration Hospital, Denver, Colo.

0041-1345/79/1101-0058$01.00/0

focal damage. In patients with the right-to-left shunts of liver disease, air presumably could quickly by-pass the lungs, enter the pulmonary veins and left heart, and by lodging in these latter locations, could remain a threat to the brain and other organs for many hours or even days. The fact that air bubbles may persist for a long time before a fatal movement to the brain could explain the lucid interval in some of our patients before the onset of seizures, paralysis, and coma.

PREVENTION OF AIR EMBOLI

An infusion technique now being used during liver transplantation apparently has eliminated the source of air emboli in the high-risk adult patients. The portal perfusion that is used to chill the organ is continued as the upper and lower vena caval anastomoses are performed, care being taken to float out any residual bubbles as the anastomoses are completed.[2] The portal venous and hepatic arterial anastomoses are then performed in the usual way. With revascularization, after completion of either three or all four anastomoses, the anesthesiologist has listened with the esophageal stethoscope for auscultatory evidence of air embolization. None has been detected since the use of this technique. Furthermore, no neurologic complications have been encountered in the additional 28 adults so treated since April 1976.

REFERENCES

1. Starzl TE, Schneck S, Mazzoni G, et al: Ann Surg 2:184, 1978

2. Starzl TE, Porter KA, Putnam CW, et al: Surg Gynecol Obstet 142:487, 1976