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Brain Circulatory Disturbance in a Central Venous Catheter-inserted Child

Tomoyuki Matsuda, M.D., Yoshifumi Tanaka, M.D., Ph.D.†*

Hickman-Broviac central venous catheters often are used for prolonged vascular access, particularly in children. Despite their advantages, complications associated with their long-term use can occur. We describe here a case in which such a catheter, in combination with insertion of a second central venous catheter and head rotation, was associated with serious neurologic complication in a child undergoing abdominal surgery.

Case Report

A 13-month-old, 8-kg, girl was scheduled for right hepatic lobectomy as a result of hepatic hamartoma. She was cachectic resulting from malnutrition, and a 6.6-French Broviac silicone single lumen catheter was inserted into the right external jugular vein 33 days before surgery for nutritional support. The tip of the catheter was confirmed in the superior vena cava by radiographic examination. Preoperatively, the patency of the inserted Broviac catheter was confirmed, and the catheter was used for infusion of fluids and medications. Anesthesia was induced by thiopental and was maintained with fentanyl, 0.5–2.0% isoflurane, and 67% nitrous oxide in oxygen. Vecuronium was used to facilitate intubation and for abdominal relaxation. After induction, the head was rotated to the right, and a 4-French oxygen saturation catheter (Opticath®, Abbott Laboratories, North Chicago, IL) was inserted into the superior vena cava through a 5-French catheter sheath in the left internal jugular vein (IJV). The insertion site was covered with a compressive gauze dressing to prevent bleeding from the skin. After insertion of this catheter, the head remained rotated to the right by about 60°. The operation was performed in a supine position over a period of 6 h. Transient massive bleeding occurred for about 1 h during surgery, at which time the patient's systolic blood pressure was between 50 and 70 mmHg.

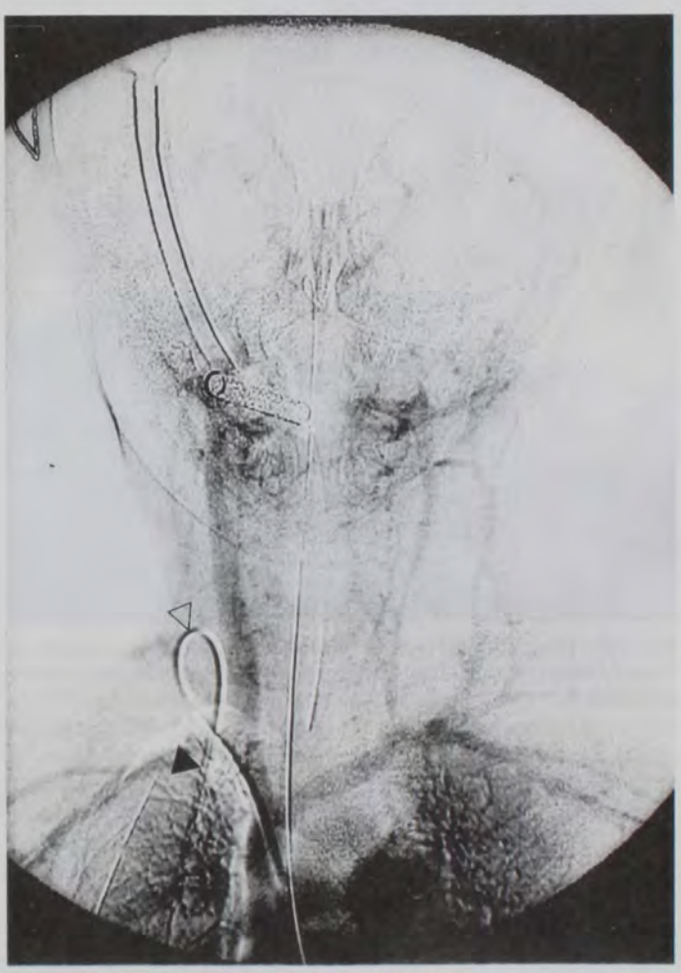

Fig. 1. Angiography shows intact right internal jugular vein flow at the mid-head position. A Broviac catheter, inserted into the external jugular vein, forms a subcutaneously concealed loop (between open and closed triangles).

During anesthesia, pulse oximetry hemoglobin saturation and the blood gas data were consistently normal. The venous hemoglobin saturation ranged from 40% to 55%, responding to blood transfusion during the massive bleeding episode. After surgery, the patient did not regain consciousness. There was no response to pain stimuli in the immediate postanesthesia period, but a cough reflex and sluggish light reflex were observed. She was transferred to the intensive care

* Research Assistant.

† Professor of Anesthesiology.

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Address reprint requests to Dr. Matsuda: Department of Anesthesiology, Kyoto Prefectural University of Medicine, Kawaramachi-Hirokoji, Kamigyoku, Kyoto 602, Japan.

Address electronic mail to: matsuda@koto.kpu-m.ac.jp

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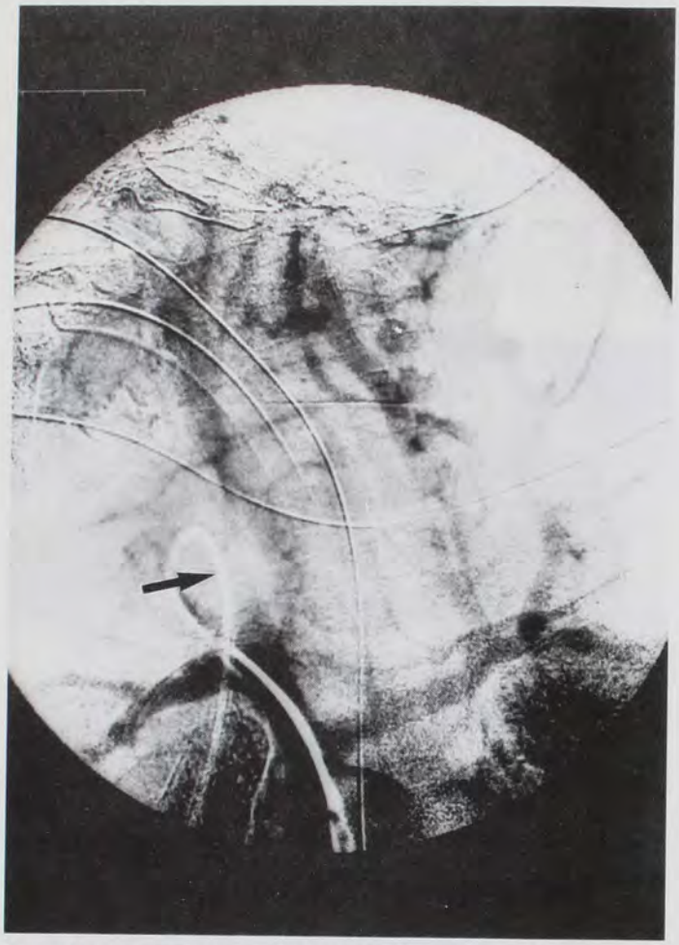

Fig. 2.On head rotation to the right side, angiography confirms interruption of right interior jugular vein flow by the Broviac catheter (*arrow*).

unit and ventilated overnight. The following morning, the patient remained unconscious. Light reflex had gradually disappeared, and her pupils were widely dilated by afternoon. Bilateral papilledema were observed at that time. An electroencephalogram showed pronounced diffuse slow-wave activity. Cranial computed tomography (CT) scan revealed global cerebral edema. Digital subtraction angiography (DSA) was performed, and results showed intact right IJV flow at the mid-cranial position (Fig. 1), but on head rotation to the right side by 60°, ipsilateral IJV flow from the brain was blocked by compression resulting from the subcutaneously concealed loop of the Broviac catheter (Fig. 2). The left jugular venous catheter-sheath had already been removed 3 h before angiography, and small left IJV flow was observed by DSA. At this point, we suspected that cerebral circulation might have been compromised during surgery as a result of the combination of vascular catheterization, head positioning, and low perfusion pressure. Three days after surgery, brain infarction was definitely observed in cerebral extensive area, but it was not focal infarction. Her clinical picture deteriorated progressively until brain death was declared 5 days after surgery.

Discussion

The development of severe neurologic dysfunction after surgery and anesthesia is rare in children and typically results from hypoxia, emboli, or hemorrhage in children with congenital heart abnormalities. The neurologic complication described here, however, occurred in a child without cardiac disease, and in the absence of hypoxia. The child was hypotensive for a 1-h period, but the recorded low blood pressure (50–70 mmHg) alone would not be expected to produce global cerebral ischemia. The complication seen in this child is thought to be the result of bilateral jugular venous occlusion combined with hypotension. The left IJV was occluded by the catheter-sheath in combination with a pressure dressing, whereas the right IJV was compressed after neck rotation by the invisible subcutaneous loop of the Broviac catheter.

Blood drains from the brain by two major routes: the jugular veins and vertebral venous plexus. The vertebral venous plexus is a major source of cerebral venous drainage only in the upright position,[1,2] and it is generally assumed that in the supine position, the jugular veins are the main cerebral venous drainage routes. It has been shown in animals that ligation of IJVs affects venous return from the cranial cavity and results in intracranial hypertension.[3] Similar events have been seen in humans.[4–6] It is likely that impairment of venous outflow is the main cause of these abnormal changes within the cranium. Further, impairment of venous return is likely to influence the reabsorption of cerebral spinal fluid possibly contributing further to intracranial hypertension and edema.[7] In the supine position, head rotation alone has an effect on IJV flow and increases the intracranial pressure (ICP), particularly with rotation 90° to the right. Because the left IJV often is smaller than the right IJV, there is a greater increase in ICP with head rotation to the right in patients with a baseline ICP greater than 10mmHg compared with that on head rotation to the left.[1,8] Studies using the range-gated Doppler technique have shown that rotation of the head 90° to the side completely obstructs jugular venous flow on that side.[8] Early recognition of jugular venous obstruction is necessary to avoid progressive cerebral injury.

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Intravenous Erythromycin Promotes Gastric Emptying Prior to Emergency Anesthesia

Vincent J. Kopp, M.D., David C. Mayer, M.D.,† Nicholas J. Shaheen, MD, ‡*

Emergency anesthesia in a patient with full stomach presents a risk of pulmonary aspiration of gastric contents, and many strategies have been developed to minimize the risk of this life-threatening complication before emergency anesthesia.[1] We report a case of endoscopically confirmed total gastric emptying after intravenous administration of erythromycin lactobionate before induction of general anesthesia for emergency esophagogastroduodenoscopy (EGD).

Case Report

A 15-yr-old, 50-kg, American Society of Anesthesiologists' (ASA) IV-E boy with cystic fibrosis, heptatobiliary cirrhosis, portal hypertension, abdominal ascites, hypersplenism, anemia, thrombocytopenia, and hydronephrosis presented to our institution in hypovolemic shock as a result of bleeding esophageal varices. After hemodynamic stabilization and volume resuscitation with packed erythrocytes, platelets, and fresh frozen plasma, he underwent emergency EGD in the pediatric intensive care unit. Five bleeding esophageal varices proximal to the gastroesophageal junction were identified, and endoscopic variceal band ligation was performed. On the second hospital day, he was transferred to the ward and resumed a regular diet and oral medications.

On the fourth hospital day, he developed signs and symptoms of recurrent gastrointestinal bleeding, principally hematemesis, hematochezia, and melena. Although hemodynamically stable, his hemoglobin and platelet counts decreased, his prothrombin and partial thromboplastin times increased, and his fibrinogen level remained stable. The decision was made to perform EGD emergently to identify and manage new or recurrent sites of bleeding. General anesthesia in the operating room was requested.

Before the decision to perform emergency EGD, the patient took oral medications and ate a meal. Six hours before the EGD, oral intake included multivitamin, vitamin K and E preparations, and pancrease tablets. Medical management of bleeding included one oral dose of particulate antacid and one intravenous dose of ranitidine 3.5 h and 2.5 h before induction, respectively. Within 2.5 h of induction, he ate a meal of meat broth, potatoes, corn kernels, and whole milk. After the decision was made to perform EGD, the patient received one intravenous dose of erythromycin lactobionate (100 mg) 80 min before induction as a gastric prokinetic agent. Particulate antacid was repeated 30 min before induction independent of anesthesiology staff instructions. Finally, immediately before induction, 30 ml of sodium citrate solution was orally administered.

Induction of anesthesia and oral endotracheal intubation were accomplished with propofol (2.0 mg/kg) and succinylcholine (1.0 mg/kg). Cricoid pressure was applied until the endotracheal tube cuff was inflated and until mid-tracheal position was confirmed. Mainte-

* Assistant Professor, Departments of Anesthesiology and Pediatrics.

† Assistant Professor, Departments of Anesthesiology and Obstetrics and Gynecology.

‡ Clinical Instructor, Department of Medicine.

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Address reprint requests to Dr. Kopp: Department of Anesthesiology, University of North Carolina, CB# 7010, 223 Burnett-Womack Building, Chapel Hill, North Carolina 27599-7010. Address electronic mail to: vkopp@aims.unc. edu

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