7. Ford GT, Whitelaw WA, Rosenal TW, Cruse PJ, Guenter CA: Diaphragm function after upper abdominal surgery in humans. Am Rev Respir Dis 1983; 127:431-6

8. Katagiri M, Young RN, Platt RS, Kieser TM, Easton PA: Respiratory muscle compensation for unilateral or bilateral hemidiaphragm paralysis in awake canines. J Appl Physiol 1994; 77:1972-82

9. Easton PA, Fleetham JA, de la Rocha A, Anthonisen NR: Respiratory function after paralysis of the right hemidiaphragm. Am Rev Respir Dis 1983; 127:125-8

10. Arborelius M, Lilja B, Senyk J. Regional and total lung function in patients with hemidiaphragmatic paralysis. Respiration 1975; 32:253-64

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Intraoperative Immediate Diagnosis of Acute Obstruction of Tricuspid Valve and Pulmonary Embolism Due to Renal Cell Carcinoma with Transesophageal Echocardiography

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PATIENTS with the tumor thrombus in the inferior vena cava (IVC) have a great risk of pulmonary embolism[1,2] and obstruction of tricuspid valve[3] during operative management. There has been several reports about intraoperative cardiac arrest and death due to such situations.[1,4] Therefore, immediate diagnosis and the subsequent management is an important challenge for anesthesiologists.

This case report describes a patient with renal cell carcinoma that extended into the IVC and caused acute obstruction of tricuspid valve and pulmonary tumor embolism intraoperatively, which were immediately diagnosed by transesophageal echocardiography (TEE) and were successfully managed by the rapid institution of cardiopulmonary bypass (CPB).

Case Report

A 35-yr-old woman, weighing 53 kg, was admitted to our hospital with appetite loss and left abdominal pain. Physical examination revealed a palpable mass in the left upper quadrant. Laboratory results showed a hematocrit of 30% and the elevation of leukocyte count (11,500), alkaline phosphatase(378 U/l), and lactic acid dehydrogenase (2,786 U/l). Radiologic examinations, including computed tomography, inferior venocavograms (fig. 1), and transthoracic echocardiography revealed a left renal mass with a tumor thrombus extending upward to near the right atrium. The chest radiograph revealed the metastatic lesions in the left lower lung area. This patient

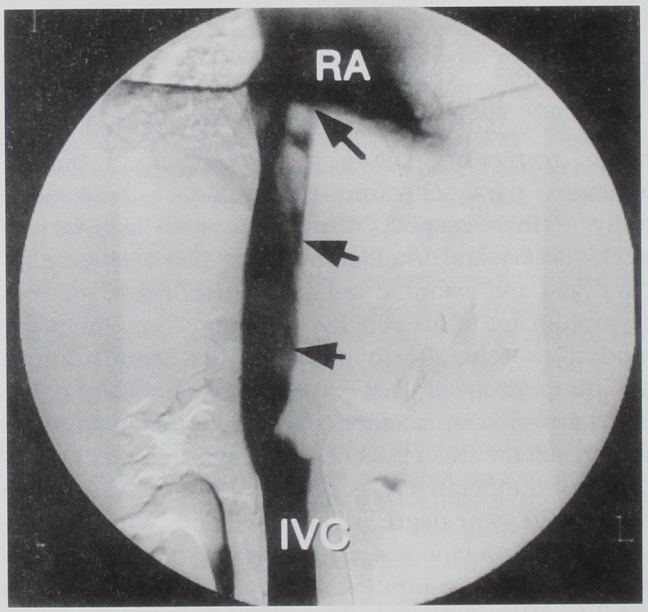

Fig. 1. Inferior venocavograms demonstrating the tumor thrombus extending upward to near the right atriumin (RA). The arrows indicate extention of the tumor thrombus in the inferior vena cava (IVC).

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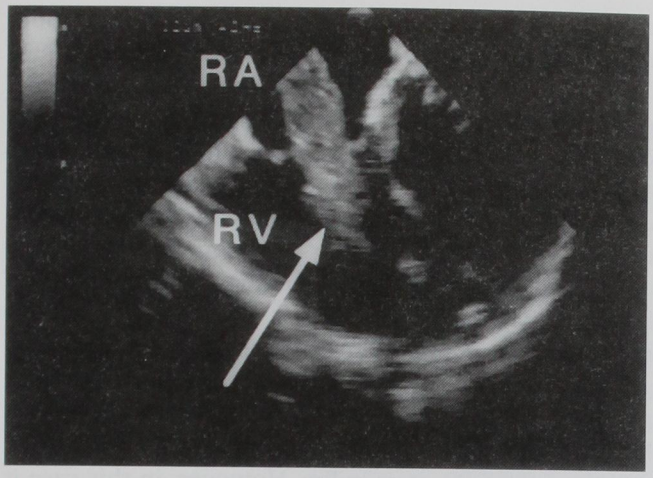

Fig. 2. Long-axis four-chamber view of the heart. The arrow shows the thrombus, which occludes the tricuspid valve and moves between right atrium (RA) and right ventricle (RV).

was scheduled for left nephrectomy, dissection of lymph nodes, and removal of the tumor thrombus using cardiopulmonary bypass (CPB).

Anesthesia was induced with intravenous fentanyl (0.2 mg), midazolam (5 mg), and thiopental (100 mg) after previous placement of an epidural catheter. Anesthesia was maintained with 66% nitrous oxide in oxygen, fentanyl, and isoflurane. Monitoring included electrocardiography, arterial and central venous pressure, pulse oximetry, end-tidal carbon dioxide monitor, and rectal temperature measurement. Two-dimensional transesophageal echocardiography (TEE; SDD870, Aloka, Tokyo, Japan) probe was placed to monitor the tumor thrombus. Tumor thrombus floating freely in the IVC near the right atrium, but no mass were observed in the four-chamber view of the heart.

With the patient in the supine position, the left nephrectomy was started by urologists. Operation and anesthesia proceeded uneventfully. About 5 h after abdominal incision, the left kidney and renal artery were isolated. At this time, blood gas analysis showed the pHa was 7.36; PaO_2, 169 mmHg; $PaCO_2$, 40 mmHg; base excess, −2.4 (FiO_2, 0.3). While the surgeons were dissecting lymph nodes around the renal artery, arterial blood pressure suddenly decreased from 94/61(70) mmHg to 49/39(42) mmHg. Concurrently end-tidal carbon dioxide decreased from 31 to 26 mmHg. An increase in the central venous pressure from 5 to 13 mmHg immediately followed. Immediately the inspired oxygen concentration was increased to 100%, and ephedrine (8 mg) was administrated, but there was no reaction. At this time, the embolization of tumor thrombus was suspected, and TEE was performed to search for the embolization. The tumor thrombus was discovered in the four-chamber view of the heart. The thrombus occluded the tricuspid valve and was moving between right atrium and ventricle (fig. 2). Although the tricuspid valve was moving, it could not prevent regurgitation. Gradually it appeared that thrombus separated into parts and moved into pulmonary artery (fig. 3). At this time, ephedrine (5 mg), methoxamine (2 mg), dopamine (10 $\mu g \cdot kg^{-1} \cdot min^{-1}$), and dobutamine (10 $\mu g \cdot kg^{-1} \cdot min^{-1}$) were administered, but failed to restore the blood pressure. Blood gas analysis showed that the pHa was 7.46; PaO_2, 374 mmHg; $PaCO_2$, 27 mmHg; and base excess, −3.6. Eight minutes later, cardiac arrest developed, and external cardiac massage was commenced. While the right femoral artery and vein were exposed, sternotomy was performed, and subsequently open cardiac massage was commenced. After heparinization (250 U/kg) and cannulation of the right femoral artery and vein, CPB was instituted. A second venous cannula in the superior vena cava was added. The time from the beginning of hypotension to commencement of CPB was 28 min. A pulmonary arteriotomy was performed, and the tumor thrombus was removed from the left, right, and the main pulmonary artery. Next, the right atrium was opened, and the tumor thrombus was removed. Residual tumor in the IVC was removed by using a Fogarty catheter. At this time, no residual tumor was observed by TEE. Finally, the left nephrectomy was completed. After insertion of a Swan-Ganz catheter, CPB was discontinued uneventfully. The operation was completed without additional complication.

The patient was transferred to the intensive care unit where she recovered without myocardial injury and neurologic complication. She initially did well, but subsequently died of metastatic disease.

Discussion

Although massive pulmonary tumor embolism from renal cell carcinoma occurs relatively rarely,[5,6] tiny emboli appear to occur more commonly. Winterbauer *et al.*[7] and Arkless[8] reported pulmonary tumor emboli in 31% and 26% of patients with renal cell carcinoma, respectively. The possibility of pulmonary embolism from renal cell carcinoma is considerably enhanced when tumor extends into the renal vein or IVC.[6] Therefore, it is considered that the risk of pulmonary embolism is high during surgical treatment in patients with renal cell carcinoma extending into the IVC because the hemodynamics are unstable and because the IVC is

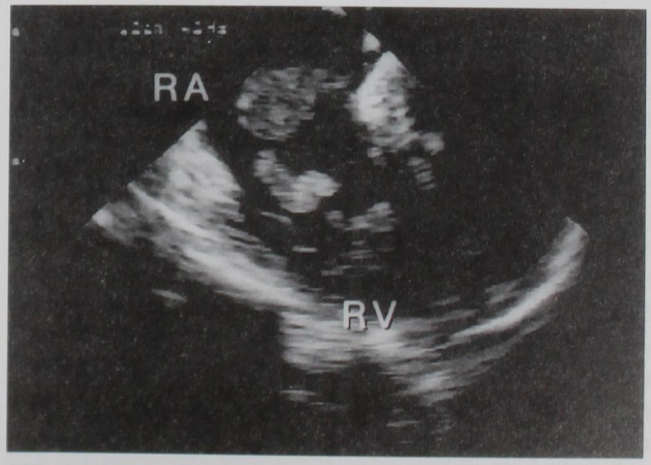

Fig. 3. Long-axis four-chamber view of the heart. The thrombus appeared to separate into parts.

manipulated intraoperatively.[5] Wilkinson *et al.*[2] reported a patient with renal cell carcinoma extending above the diaphragm who developed massive pulmonary embolism during a radical nephrectomy. Milne *et al.*[1] also reported a case of massive intraoperative pulmonary tumor embolus from renal cell carcinoma invading the IVC, who developed cardiac arrest and required open cardiac massage and institution of CPB.

Intraoperative symptoms associated with pulmonary embolism include hemodynamic changes such as a decrease in arterial pressure and increase in central venous pressure, hypoxemia, and a reduction in end-tidal carbon dioxide tension with a large increase of the arterial to end-tidal carbon dioxide tension gradient. However, diagnosis of pulmonary embolism may be difficult in the operating room because diagnostic procedures such as pulmonary scintigraphy, transthoracic echocardiography, or angiography cannot be performed usually. Therefore, TEE has become the procedure of choice in the evaluation of the patient with embolic events.[9,10] Langeron *et al.*[9] reported a case in which the thromboembolus migrating from the right atrium to the right ventricle was observed with TEE, and pulmonary embolism was intraoperatively diagnosed. However, as the limitation of TEE, there is the possibility of missing small emboli or those that have already passed into the pulmonary vascular bed.[11] Nevertheless, Kasper *et al.*[12] reported that there is a close correlation between size of the right pulmonary artery and mean pulmonary artery pressure in patients with pulmonary embolism and changes in the right-to-left ventricular end-diastolic area ratio correlate with severity of pulmonary obstruction.

Obstruction of tricuspid valve is another possible intraoperative fatal complication in patients with renal cell carcinoma. Utley *et al.*[3] reported a patient with Wilms' tumor extending into the IVC, in which acute obstruction of the tricuspid valve occurred, and circulation was arrested. However, diagnosis of obstruction of the tricuspid valve was made only after the immediate sternotomy and institution of CPB. Wilson *et al.*[11] reported a patient undergoing pelvic surgery for bleeding fibroids, in which obstruction of tricuspid valve caused by massive thrombus was diagnosed by using TEE intraoperatively. To our best knowledge, we believe that this is the first report in which acute obstruction of tricuspid valve from renal cell carcinoma invading the IVC was intraoperatively diagnosed by using TEE. Diagnosing intraoperative tumor events in the IVC, atrium, ventricle, and pulmonary artery without any delay can allow us immediate treatment of patients including interruption of the vena cava and embolectomy under the CPB.

Once the fatal complication such as massive pulmonary embolism and obstruction of tricuspid valve occurred, institution of CPB may be mandatory to save the patient. Milne *et al.*[1] reported a patient with renal cell carcinoma invading the IVC, in which cardiac arrest occurred during a radical nephrectomy with CPB available on standby, and the time from the beginning of hypotension to commencement of CPB was approximately 10 min. Wilkinson *et al.*[2] also reported that emergency institution of CPB was required for salvage of a patient with intraoperative massive pulmonary embolism, and the time to commencement of CPB was within 20 min, with CPB on standby. However, in this case it took approximately 30 min to commence CPB from the beginning of hypotension. With more delay, the patient might have been in jeopardy. Therefore, we believe that it is important to prepare for CPB with it on standby.

In conclusion, we present a case in which massive pulmonary embolism and obstruction of tricuspid valve from renal cell carcinoma invading the IVC were intraoperatively diagnosed by TEE. TEE is inevitably necessary for intraoperative monitoring of tumor events in the IVC, atrium, ventricle, and pulmonary artery in such patients.

References

1. Milne B, Cervenko FW, Morales A, Salerno TA: Massive intraoperative pulmonary tumor embolus from renal cell carcinoma. ANESTHESIOLOGY 1981; 54:253–5

2. Wilkinson CJ, Kimovec MA, Uejima T: Cardiopulmonary bypass in patients with malignant renal neoplasms. Br J Anaesth 1986; 58:461–5

3. Utley JR, Mobin-Uddin K, Segnitz RH, Belin RP, Utley JF, Ky L: Acute obstruction of tricuspid valve by Wilm's tumor. J Thorac Cardiovasc Surg 1973; 66:626–8

4. Masson AHB, Bramwood AW: Sudden operative death due to tumor embolism. BMJ 1955; 1:1514–5

5. Kubota H, Furuse A, Kotsuka Y, Yagyu K, Kawauchi M, Saito H: Successful management of massive pulmonary tumor embolism from renal cell carcinoma. Ann Thorac Surg 1996; 61:708–10

6. Novick AC, Daughtry JD, Stewart BH, Groves LR: Pulmonary embolus presenting as initial manifestation of renal cell carcinoma. Urology 1978; 12:707–9

7. Winterbauer RH, Elfenbein IB, Ball WC: Incidence and clinical significance of tumor embolization to the lung. Am J Med 1968; 45:271–90

8. Arkless R: Renal carcinoma: How it metastasizes. Radiology 1968; 84:496–501

9. Langeron O, Goarin JP, Pansard JL, Riou B, Viars P: Massive intraoperative pulmonary embolism: Diagnosis with transesophageal two-dimensional echocardiography. Anesth Analg 1992; 74:148–50

10. Nixdorff U, Erbel R, Drexler M, Meyer J: Detection of thromboembolus of the right pulmonary artery by transesophageal two-dimensional echocardiography. Am J Cardiol 1988; 61:488–9

11. Wilson WC, Frankville DD, Maxwell W, Carpenter T, Hastings RH: Massive intraoperative pulmonary embolus diagnosed by transesophageal echocardiography. ANESTHESIOLOGY 1994; 81:504–8

12. Kasper W, Meinertz T, Kerting F, Lollgen H, Limbourg P, Just H: Echocardiography in assessing acute pulmonary hypertension due to pulmonary embolism. Am J Cardiol 1980; 45:567–72

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Fiberoptic Intracranial Pressure Monitoring during Magnetic Resonance Imaging

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MAGNETIC resonance imaging (MRI) provides high quality diagnostic information in many central nervous system disorders.[1,2] During MRI, patients are exposed to a large static magnetic field (*e.g.*, 1.5 Tesla) and radiofrequency (RF) pulse sequences.[1–3] It has long been appreciated that the presence of metallic objects within the magnetic field can lead to image distortion or, worse yet, patient injury.[1–7]

Magnetic resonance imaging centers typically keep compatibility registers of commonly used biomedical devices.[5,6] Unfortunately, with the growing number of biomedical devices, it is often difficult to keep such registers up to date.[6]

We report a case in which a commonly used intracranial pressure (ICP) monitor was found to be MRI incompatible. We also discuss a practical two-step method for performing *ex vivo* MRI compatibility screening.

Case Report

A 24-yr-old man with a severe closed head injury requiring intubation and ventilation underwent an emergent computed tomography (CT) scan of his head and neck. There was no evidence of intracranial pathology requiring surgery, although due to his injury, a fiberoptic ICP monitoring catheter (Camino Laboratories, San Diego, CA, Model 110-4B) was placed in the parenchyma of the right frontal lobe *via* a small burr hole. The initial ICP reading was 9 mmHg. The cervical spine CT scan was positive for fractures of C5 and C6, with subluxation of C6 on C7.

To better characterize the extent of the neck injuries, an MRI examination of the cervical spine was scheduled, and the neuroanesthesiology service consulted for patient management during the scan. As part of routine patient screening, the radiology department queried whether the patient had any ferromagnetic devices that would preclude exposure to a magnetic field. This initial screen was negative, and the patient was brought to the MRI suite. Before scanning, the ICP monitor was noted, and imaging was deferred until MR compatibility of the indwelling monitor could be determined. Neither the Mayo Clinic MRI database nor the catheter product insert had information pertaining to its MRI compatibility. Additionally, a search of the current MR safety literature was devoid of compatibility information for this monitor. Lastly, we contacted the manufacturer *via* telephone and were informed that MRI compatibility testing had not been performed on this device.

Because we were unable to determine the safety of this device in a 1.5 Tesla magnetic field, the fiberoptic catheter was electively removed by the neurosurgical service. As part of the ferromagnetic screening process, the fiberoptic catheter was then exposed and strongly attracted to a hand-held ring magnet. Based on this observation, the catheter was not replaced, and the MR examination proceeded without incident. Subsequently, the ICP monitor was sent to our Magnetic Resonance Imaging Research Laboratory for further compatibility testing.

Testing was done using a two-stage process. The first stage was a simple screen for ferromagnetism using a hand-held ring magnet, as had been done previously (fig. 1). Any perceptible deflection was considered positive for ferromagnetic properties. The second stage of testing involved calculating a deflective force at the bore of the imager magnet. This was achieved by suspending the biomedical implant from a fine silk string and exposing it to the 1.5 Tesla magnetic field of the MR imager (GE Medical Systems, Milwaukee, WI). Assuming the mass of the string is zero, the force acting on the object can be calculated using the following standardized[3,4] formula:

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Key words: Biocompatible material: chemistry; metals. Magnetic resonance imaging: adverse effects; contraindications. Material testing. Radiofrequency.