Therefore, the antemortem diagnosis of RATT is important.

Diagnostic methods for the RATT associated with the HCC include echocardiography, angiography, and radioisotope scintigraphy. However, no echocardiographic changes of the tumor thrombus before and after transarterial embolization therapy have been reported previously. This is the first report of such changes. In our case, the tumor within the IVC and RA was not embolism, but the true tumor thrombus because of selective celiac angiographic and inferior cavaographic findings, and the diminishing of the true tumor thrombus after chemoembolization therapy was clearly detected by echocardiography, but necrosis of the tumor thrombus shown by CT was barely evident on echocardiography. Though the mechanism of the diminution of RATT is thought to be necrosis and fragmentation, fragmentation is unlikely because a pulmonary embolic event was not detected clinically throughout his course.

Such thrombi associated with HCC are fed from tumor vessels derived from the hepatic artery. In our case, pooling of Lipiodol within the tumor thrombus was seen on CT scan after chemoembolization therapy. This also shows hepatic arterial supply of the tumor thrombus. Therefore, chemoembolization therapy is considered to be an effective treatment against the secondary Budd-Chiari syndrome due to the tumor thrombus of the venous system.

**CLINICAL LIMITATION**

In our reported case, autopsy was not performed, and histologic diagnosis could not be established. However, the tumor was diagnosed as HCC by morphologic and physiochemical findings.

**REFERENCES**


**Neurogenic Pulmonary Edema after Trigeminal Nerve Blockade**

Robert S. Wright, M.D.; Tony Feuerman, M.D.; and Julie Brown, R.N.

Acute neurogenic pulmonary edema developed immediately after injection of bupivacaine hydrochloride into the trigeminal cistern of a 28-year-old man with atypical facial pain and no prior history of cardiopulmonary problems. This complication of trigeminal nerve blockade has not been reported previously, to our knowledge. Associated neurologic deficits suggest a key role for the brain stem in the pathogenesis of this disorder. (Chest 1989; 96:436-39)

Neurogenic pulmonary edema is a process that typically occurs after severe, and often devastating, CNS events. While the pathogenesis of the pulmonary edema is uncertain, two widely divergent hypotheses have been proposed. Some investigators think that marked hemodynamic alterations in the pulmonary circulation are of paramount importance, while others believe that neuroendocrine factors mediate a pulmonary capillary leak process. CNS events that have been associated with neurogenic pulmonary edema include severe head trauma, hemorrhagic strokes, generalized seizures, and various operative interventions. We report a case of short-lived neurogenic pulmonary edema occurring immediately after injection of the trigeminal

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Cistern with bupivacaine hydrochloride (HCl). This complication has not been reported previously, to our knowledge.

**Case Report**

A 32-year-old man with bilateral atypical facial pain was scheduled for diagnostic injection of the left trigeminal nerve with bupivacaine.

He was entirely well, with no prior history of cardiopulmonary disease, until eight years before admission, when he began to experience facial pain. The pain became progressive and was diagnosed as trigeminal neuralgia. Routine medical measures failed to palliate his symptoms. Subsequently, two surgical procedures to decompress the posterior fossae were performed with poor relief of pain. Three months before admission, he underwent percutaneous injection of the right trigeminal cistern with bupivacaine HCl. The injection reduced his pain by approximately 60 percent. A diagnostic bupivacaine injection of the left trigeminal cistern was scheduled in an attempt to provide similar reduction in pain on the left side. The plan was to perform glycerol injection of the trigeminal nerves if the bupivacaine blockade was successful.

Before the left trigeminal nerve injection, the patient was feeling well, except for his usual facial pain. Vital signs showed a blood pressure of 140/80 mm Hg and a heart rate of 82 beats/min. Heart and lung examination results were normal. A chest X-ray film (Fig 1) was within normal limits.

After being placed supine in a pneumoencephalography chair, the patient was given 0.625 mg IV droperidol, 2 mg IV midazolam, 200 mg IV methohexital HCl, and 50 µg IV fentanyl citrate uneventfully; 3 ml of lidocaine was then injected at a site 2 cm lateral to the left corner of the mouth. Vital signs remained stable. A 22-gauge spinal needle was then directed toward the foramen ovale and punctured the dura. After its position had been confirmed by fluoroscopy to be in the inferior portion of the trigeminal cistern, 0.5 ml of bupivacaine HCl 0.75 percent was injected uneventfully. The needle was then advanced slightly, and an additional 1.25 ml bupivacaine was injected. At this point, the patient suddenly developed tachycardia (heart rate, 150 beats/min) and complained of severe dyspnea. His blood pressure transiently increased to 150/85 mm Hg. Oxygen saturation was measured continuously by finger pulse oximeter and fell to 76 percent. He became confused, and his pupils became fixed and dilated. Complete ophthalmoplegia was noted. Endotracheal intubation was performed, and the patient received mechanical ventilation with an F1O2 of 1.0. On further examination, he appeared fully oriented, but his pupils remained fixed and dilated. He continued to have complete ophthalmoplegia with absent corneal reflexes. Chest examination results showed diffuse crackles. An ECG showed sinus tachycardia without any ischemic changes. Chest X-ray film (Fig 2) showed acute pulmonary edema. Within minutes of intubation, his airways became flooded with copious amounts of serosanguinous fluid. The patient was given 10 mg IV of furosemide (Lasix) and IV fentanyl and midazolam for sedation. Positive-pressure ventilation was continued with the addition of approximately 5 cm PEEP. Six hours after intubation, he was extubated. His ophthalmoplegia resolved completely the same day, and he had no permanent neurologic deficits. A chest X-ray film taken the following day showed marked improvement. He was discharged home two days after the procedure.

**Discussion**

Neurogenic pulmonary edema occurred in our patient within minutes of a bupivacaine injection of the trigeminal cistern. Although other complications of trigeminal nerve blockade have been reported, this complication has not been reported, to our knowledge.

The pathogenesis of the pulmonary edema in this patient is not clear. However, altered pulmonary capillary integrity is suspected on the basis of the copious amount of serosanguinous fluid that suddenly flooded the airways. The fluid resembled blood on gross appearance and RBCs may have entered the airways through a leaky endothelium. Previous clinical and experimental reports have suggested such a possibility.

Since pulmonary artery catheterization was not performed, an alteration in pulmonary hemodynamics cannot be discounted. However, he was borderline hypertensive for a very brief period, so acute left ventricular failure is highly unlikely. Nevertheless, the possibility of acute and severe pulmonary venoconstriction was not excluded and has been suggested by other investigators. In this patient, there was roentgenologic evidence of right ventricular volume overload, which could have been precipitated by acute pulmonary hypertension. In this regard, it has been suggested that sympathetic discharge and, particularly, the
release of norepinephrine might exert intense venoconstriction at the level of the pulmonary venule with resultant pulmonary edema.4,5

The concomitant neurologic deficits in this patient suggested that alterations in brain stem activity may have precipitated the pulmonary edema. Previous animal studies have produced pulmonary edema by causing deficits in the medulla oblongata. In this patient, the neurologic findings suggested a deficit at the level of the pons. Moreover, cervical cord transection has been shown experimentally to block the onset of neurogenic pulmonary edema.4

Treatment in this patient was primarily supportive. The relatively low dose of Lasix did not alter fluid balance to any significant extent and would not be expected to be of significant benefit in this disorder. PEEP was transiently used, and its use must be tempered by the knowledge that it can potentially increase intracranial pressure and worsen pulmonary edema. As such, it is generally recommended that basic supportive measures be used alone, possibly along with an α-adrenergic blocking agent if severe hypertension is present.9

This report should make physicians aware of the possibility of acute neurogenic pulmonary edema after anesthetic injections in the region of the brain stem.

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Numerous Mesothelial Cells in Tuberculous Pleural Effusions*

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Tuberculous pleural effusions are characterized by the absence or paucity of mesothelial cells. Two cases of pleural tuberculosis are reported in which significant numbers of mesothelial cells were found. (Chest 1989; 96:439-39)

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FIGURE 1. Posteroanterior view of chest in case 1 shows a rightsided pleural effusion and enlarged right paratracheal lymph nodes.