Horner's Syndrome Secondary to Hydromediastinum*
A Complication of Extravascular Migration of a Central Venous Catheter
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We report the findings in a patient who developed Horner's syndrome as the first manifestation of mediastinal migration of a central venous catheter that resulted in hydromediastinum and hydrothorax. The pathogenesis of this complication of central venous catheterization is discussed. (Chest 1988; 94:1093-94)

Horner's syndrome results from interruption of the ipsilateral sympathetic pathway at any site along its course between the hypothalamus and the eye. Since the preganglionic fibers traverse the superior mediastinum, Horner's syndrome can result from any insult to this region, such as lymphadenopathy or tumor. We present a case of Horner's syndrome from hydromediastinum due to migration of a central venous catheter.

CASE REPORT
A 58-year-old white man was transferred to the Charleston (SC) Veterans Administration Hospital 24 hours following admission to an outlying hospital for severe headaches. A lumbar puncture confirmed subarachnoid hemorrhage, and cerebral angiography identified two leaking cerebral aneurysms. A craniotomy was performed, and both aneurysms were clipped successfully. During the perioperative period, an 8 French percutaneous introducer was placed by the neurosurgical team in the right subclavian vein for central venous access. The chest roentgenogram after placement showed proper position of the catheter tip in the region of the superior vena cava; the roentgenogram showed a normal mediastinum and no pleural effusion.

The postoperative course was uneventful over the first three days, except for the development of hypotension, which was treated with 3 percent saline solution administered via the right subclavian catheter. On the fourth postoperative day the patient developed a right-sided Horner's syndrome with miosis, ptosis, and facial anhidrosis; this was initially attributed to a complication of the craniotomy. Over the subsequent 24 hours, the patient had progressive respiratory distress requiring intubation and mechanical ventilation. A chest roentgenogram following intubation (Fig 1) showed the right subclavian catheter unchanged in position, but new findings of mediastinal widening and a massive right and a small left pleural effusion. A right thoracentesis showed the following: serosanguinous fluid, white blood cell count, 375/ml; red blood cell count (RBC), 40,000/ml; total protein level, 2.3 g/dl; lactic dehydrogenase (LDH) level, 85 IU/dl; pH 7.46; and glucose level, 107 mg/dl. Gram stain and staining for acid-fast bacteria were negative, and cultures were sterile.

The right subclavian catheter was removed, and a pulmonary arterial catheter was placed via the left subclavian vein. The pulmonary capillary wedge pressure was 10 mm Hg. Without specific therapy, the right Horner's syndrome and pleural effusions resolved over the next 48 hours. The patient was weaned successfully from mechanical ventilation and had an uneventful recovery.

DISCUSSION
Numerous complications of central venous catheters have been reported, especially with their increased use over the last two decades as routes for hemodynamic monitoring and parenteral alimentation. Migration of a central venous catheter from the intravascular space into the mediastinum or pleural space, resulting in hydromediastinum or hydrothorax is a potentially serious complication that can result in respiratory compromise if not recognized early. Extravascular migration usually occurs hours to days after successful placement has been confirmed by venous blood return and radiographic localization. In two series reporting migration of central venous catheters, all six patients had severe respiratory compromise, and three required mechanical ventilation before a correct diagnosis of migration of the central venous catheter was established. Chest pain was the only reported symptom prior to dyspnea.14

Our patient developed Horner's syndrome prior to clinical or radiographic detection of hydromediastinum that could have provided a clue to migration of the central venous catheter. Horner's syndrome (unilateral ptosis, miosis, and facial anhidrosis) results from the interruption of the ipsilateral sympathetic pathway. The oculosympathetic outflow begins in the postero lateral hypothalamus, descends uncrossed through the brain stem, and synapses in the intermediolateral gray matter of the spinal cord between the eighth cervical and fourth thoracic vertebrae. The second-order or preganglionic neuron exits the spinal cord via the ventral roots to the paravertebral sympathetic chain. The fibers pass over the pulmonary apex and course through the

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Figure 1. Recumbent chest roentgenogram after intubation, showing widening of mediastinum and large right pleural effusion. Note presence of right subclavian catheter abutting superior mediastinum.
mediastinum before terminating in the superior cervical ganglion located at the angle of the jaw medial to the carotid artery and juxtaposed to the carotid sheath. The third-order or postganglionic nerves arise in the superior cervical ganglion and run on the common carotid and internal carotid arteries as a plexus ascending the internal carotid artery to enter the cavernous sinus joining the ophthalmic division for the trigeminal nerve to the orbit. The postganglionic fibers to the face that regulate sweating do not enter the skull but follow the external carotid artery.3

We postulate that this patient's transient Horner's syndrome resulted from either irritation to the nerve from instillation of hypertonic (3 percent saline) fluid into the mediastinum or from pressure on the nerve in the mediastinum prior to spontaneous decompression of blood and fluid into the pleural space. An alternative explanation is that the central venous catheter itself caused direct neural irritation.

As described previously,4 the presence of a relatively stiff, large-bore (8 French) percutaneous introducer used for central venous access most likely predisposed the patient to this complication. Although seen most commonly with catheterization of the external jugular vein, migration into the mediastinum has been reported with insertion of a central venous catheter from any approach. The right subclavian approach, in particular, has an acute angle at the point where the right subclavian vein meets the right brachiocephalic vein, making this a likely point for extravascular migration.

Horner's syndrome is a recognized complication of carotid arterial catheterization and has been reported infrequently following internal and external jugular cannulation. This usually is ascribed to pressure on the postganglionic fibers in the carotid plexus.4 Other iatrogenic causes are those associated with manipulation of the pulmonary apex, such as with tube thoracostomy.4 To our knowledge, there are no reports of Horner's syndrome from subclavian catheterization or hydromediastinum, but this may be underdiagnosed, particularly in critically ill patients.

The pleural fluid was sampled to exclude other potentially serious causes of pleural effusion, such as empyema. The presence of an RBC of 40,000/ml was likely caused by hemorrhage from the involved vessel. The levels of protein (2.3 g/dl) and LDH (85 IU/ml) are consistent with a transudative effusion, although both were considerably higher than the infusate, which contained no protein. This may have resulted from increased permeability of the mediastinal pleural vessels induced by the hypertonic infusion and the subsequent equilibration with the interstitial spaces of the mediastinum and pleura.

The diagnosis of migration of a central venous catheter may be subtle and, if not discovered, can progress to respiratory failure or vascular compromise with infusion of fluid into the mediastinum and pleural space. The acute occurrence of an ipsilateral Horner's syndrome without obvious cause may be an early clue to this diagnosis and warrants further investigation. Absence of blood return and the observation of fluid similar to the infusate on aspiration of the central venous catheter is diagnostic of extravascular migration of the catheter. A chest roentgenogram should be obtained to assess the location of the central venous catheter and to search for mediastinal widening or pleural effusion. When the diagnosis is established the infusion should be stopped and the central venous catheter removed.

REFERENCES

Endobronchial Hamartoma Associated with Bronchioalveolar Cell Carcinoma*

Dominique Mompoint, M.D.; Odile Groussard, M.D.; Philippe Grenier, M.D.

Computed tomographic findings in a 60-year-old man with lobular bronchioalveolar cell carcinoma distal to endobronchial hamartoma are described. (Chest 1988; 94:1094-96)

Computed tomography (CT) findings in a case of central, endobronchial hamartoma associated with lobular bronchioalveolar carcinoma in the subtended lung parenchyma are described. This pathologic association has not, to our knowledge, been previously reported.

CASE REPORT

A 60-year-old male smoker, with a history of chronic obstructive pulmonary disease, presented with loss of weight and hemoptysis of three months' duration following an episode of bronchial infection. Chest roentgenograms showed a persistent ill-defined alveolar consolidation in apical-posterior and inferior lingular segments of the left upper lobe (Fig 1). Bronchoscopic examination revealed a polypoid, elastic, mobile mass arising from the posterior wall of the left upper lobe bronchus, extending to its superior division and occluding the lingular bronchus. Bronchoscopic biopsy was negative for cancer. Computed tomographic scans showed a smooth-edged endobronchial intraluminal left upper lobe mass, 2 cm wide, extending to the lingular lumen; thin-section scans revealed small foci of fat inside the mass and no detectable calcium deposits (Fig 2). The diagnosis of endobronchial hamartoma was established by CT on the evidence of fat within the lesion. The alveolar consolidation visible in the upper and lower segments of the left upper lobe was considered obstructive pneumonitis.

Left upper lobectomy was performed. The examination of the specimen disclosed a pedunculated endobronchial tumor, 2 cm long, obliterating the apicodorsal bronchus. The tumor base was

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