Late Pleuropulmonary Metastases of a Cerebral Ependymoma*


A patient with a 12-year history of occipital ependymoma was found to have late pleuropulmonary metastases without recurrence of the primary tumor. The pleural metastases were diagnosed by histologic, ultrastructural features and finally by glial fibrillary acidic protein (GFAP) labeling positive reaction. This case is unique because of the long interval between occurrence of the initial tumor and the metastases, and because of the apparent quiescence of the cerebral lesion when the pleuropulmonary metastases were discovered. (Chest 1988; 94:1097-98)

We report herein a case of late pleuropulmonary metastases of an occipital ependymoma in a young woman.

CASE REPORT

A 27-year-old woman was admitted in February 1985 for evaluation of a right pleural effusion. Twelve years before, she had undergone surgery for a left occipital ependymoma (grade 4). Six subsequent intracranial recurrences were treated by surgery and radiotherapy. Central nervous system (CNS) computed tomography (CT) scan was normal in January 1985.

The patient was in no acute distress but had right pleural effusion and sequelae of right hemiparesis. A chest film disclosed right pleuritis and a regular, scalloped thickness of parietal pleura (Fig 1). Pleural fluid contained 4.0 g/dl protein, 240 cells/ml with 90 percent lymphocytes; cultures for Mycobacterium tuberculosis and for pyogens were sterile. After three unsuccessful pleural needle biopsies, a pleurectomy revealed a nodular pleura with right hemidiaphragmatic involvement and several pulmonary nodules.

Light microscopic examination disclosed a tubular pattern and perivascular pseudorosettes of spreading polygonal tumoral cells. An intracytoplasmic ciliary pattern and several zonae adherens were found by electron microscopy. A positive reaction to antibody raised against GFAP was obtained by immunocytochemistry, using a labeled biotin-avidin technique, and confirmed the ependymal origin of the pleural metastasis. A similar pattern was demonstrated on sections of the initial tumor specimen.

In spite of chemotherapy (Adriamycin-Lomustine), pleuropulmonary metastases worsened but remained isolated, as demonstrated by CT scan (Fig 2). One year later, pituitary, encephalic, and brain-stem metastases developed. The patient died 18 months after diagnosis of the pleural effusion and 14 years after initial diagnosis of ependymoma. An autopsy was not performed.

DISCUSSION

Ependymoma is a rare tumor of the CNS and is usually complicated by local recurrence or CNS metastases. However, 33 cases of extraneural metastases of ependymoma


FIGURE 2. Thoracic CT scan several months after right thoracotomy was performed.
Figure 1. Continuous record of lead 2.

complexes to the preceding sinus P or atrial premature beat, with its interference of possible retrograde impulse could be the explanation. When no P wave was encountered in front of the QRS complexes or the distance between the P wave and QRS complex was more than 0.50 second, a retrograde impulse would then encounter the atrium out of its refractory period, so that retrograde activation was possible. In strip IIa of Figure 1, the sixth complex had an upright P wave before it, with the P-R interval being 0.18 second; the R-R interval was shorter than the R-R interval, simulating a ventricular capture beat; however, the R-R interval equaled that of the R-R and R-R intervals, so R was possibly still an atrioventricular junctional escape beat, with the P wave in front of it being merely coincidental. In strip IIc of Figure 1, two different P waves were evident (ladder diagram); the 1st, 4th, 5th, and 6th P waves were of sinus origin and dome shaped with a duration of 0.01 second. The 2nd, 3rd, and 7th P waves were peaked, with a duration 0.08 second, suggesting third-degree intra-atrial block (Bachmann's bundle block), with the sinus impulses activating only the right atrium. A dome-shaped P' wave appeared after the third sinus wave prematurely, apparently not related to the following QRS complexes, presumably of high atrial origin. Reentry of P', probably occurred at the sinoatrial junction to form P'; the P-P interval was thus shorted to 0.96 second. The P' wave probably activated the sinus node retrogradely to reset the sinus rhythm. Electrocardiographic diagnosis was third-degree atrioventricular block, retrograde atrial activation, and intermittent atrio-atrial block.

Figure 2 was recorded from lead V'. The QRS complex exhibited an rS configuration with a duration of 0.08 second, evidently of junctional origin. Retrograde P waves after the 1st, 2nd, 4th to 6th, and 8th QRS complexes were noted, with intervals being 0.08 to 0.12 second. No retrograde P' waves were seen after 3rd and 7th QRS complexes, presumably because of the shorter intervals to the preceding sinus P waves (less than 0.50 second) so that retrograde conduction of the impulses to the atria would not be possible. Sinus P waves exhibited two different contours. The 1st, 2nd, and 4th sinus P waves were biphasic, with a duration of 0.12 second, with PTFV, being −0.04 mm/sec; the 3rd and 5th P waves were upright and peaked, with an amplitude of 2 mm and duration of 0.06 second. Intermittent third-degree intra-atrial block was considered to be present with only the right atrium activated, giving rise to the peaked contour. The 3rd and 4th sinus P waves were 3.48 seconds apart and three times the sinus cycle length; retrograde P' waves were apparent after the QRS complexes. Retrograde conduction of the junctional impulses with interference with the sinus impulses at the sinoatrial junction was probably the underlying mechanism; another possibility was the coexistence of Mobitz type 2 sinoatrial block.

**DISCUSSION**

Early in 1914, Cohn and Fraser observed retrograde conduction of the atria in a case of high-degree atrioventricular block. Only scattered reports were seen thereafter. The underlying mechanism of retrograde conduction in atrioventricular block is worth probing. Under physiologic conditions, the atrioventricular node is in a state of relative inhomogeneous inhibition, and inhibition in the proximal portion is far greater than that in the distal part. The reversion occurs in high-degree atrioventricular block; the degree of inhibition in the distal part would be greater. Conduction of the sinus impulse in an anterograde direction with decremental conduction would block the impulse when it reaches the distal part of the atrioventricular node, but when impulse conduction is retrograde, then impulses from a pacemaker below the area of block would first encounter and overcome the most depressed area and thereafter easily reach the atria. Another explanation is the so-called summation of impulses. When the bundle branches and fascicles are diseased, the anterograde impulses would be blocked, while retrograde impulses from the bundle branches and fascicles would overcome the blockade because
of summation of impulses which is strong enough to cross the block. This may play an important role in high-degree atrioventricular block. The previously mentioned two effects may act together to make retrograde conduction possible. As to ventricular contractions from the ectopic pacemakers, impulses below the block give rise to mechanical or electrotonus effects to activate the area above the block, and the hypothesis of dual conduction pathways is merely conjectural. 1.14

In the present case, defects of the atrioventricular junction as well as the atria were possible; when intra-atrial block is present, the following may occur: (1) sinus impulses reach the lower part of the right atrium and then cross over to activate the left atrium through the atrial muscle; and (2) the left atrium does not depolarize. 3 The latter probably was the case in the present report. In leads 2 and V1, narrow and peaked P waves intermittently appeared, denoting third-degree intra-atrial bundle-branch block; the peaked P waves signified that the right atrium was activated, while left atrium was not depolarized.

REFERENCES

Right-to-left Shunt Through Patent Foramen Ovale Complicating Right Ventricular Infarction

Successful Percutaneous Catheter Closure

Steven K. Krueger, M.D.; and Donald L. Lappé, M.D.

A patient with right ventricular infarction and severe hypoxemia secondary to right-to-left shunting through a patent foramen ovale is presented. A balloon tip catheter was positioned in the left atrium and retracted against the atrial septum and the hypoxemia resolved. (Chest 1988; 94:1100-01)

Right ventricular infarction is common and potentially lethal. Recognition and correct treatment frequently result in short-term survival which usually becomes long-term success. Therefore, optimal treatment is essential.

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Patent foramen ovale is common and benign unless right atrial pressure (RAP) exceeds left atrial pressure (LAP) and right-to-left shunting of blood or clot ensues. Right ventricular infarction frequently results in increased RAP and right-to-left shunting has been reported.

We report a patient with right ventricular infarction, patent foramen ovale, significant hypoxemia secondary to right-to-left shunting, successfully treated with short-term percutaneous catheter closure of the patent foramen ovale.

CASE REPORT

A 74-year-old man was admitted with acute inferior myocardial infarction with CK elevation to 2,563 with 16 percent MB. Temporary ventricular pacing was instituted for sinus bradycardia. Hypotension refractory to 2 L of intravenous fluid required therapy with dobutamine. Renal failure and confusion developed. Arterial blood gas determinations showed PaO2 = 65 mm Hg on 70 percent O2 by mask. There was no preexisting pulmonary disease. Physical examination was remarkable for confusion and jugular venous distension. Chest x-ray film showed pulmonary venous congestion. The electrocardiogram showed acute inferior myocardial infarction and 1 mm ST-T segment elevation in V1.

The next six days were marked by persistent hypotension requiring therapy with dobutamine. The cardiac index was 1.94, right atrial pressure = 23 mm Hg, and pulmonary capillary wedge pressure, 19 mm Hg and stable. An echocardiogram with contrast showed severe posterior and right ventricular hypokinesis and right-to-left crossing of contrast at the atrial level.

The PaO2 level dropped to 45 mm Hg on 100 percent O2 and the patient underwent cardiac catheterization (results in Table 1). Angiography showed inferior hypokinesis, 100 percent obstruction of the proximal right coronary artery (RCA), 60 percent stenosis of the first diagonal and 60 percent stenosis of the proximal left anterior descending (LAD).

A Gemini catheter was advanced through the patent foramen ovale and exchanged over a guidewire for a 5F Swan-Ganz catheter. The balloon was inflated with a dilute contrast medium and the catheter retracted until resistance was met. The PaO2 rose to 286 mm Hg on 100 percent FiO2. The catheter was anchored to the leg with a rubber band. Anticoagulation was accomplished with IV heparin infusion.

The next 12 days brought no significant change with right atrial pressure remaining higher than the left, and the patient remained in shock with cardiac index = 1.52, RAP = 25-12 mm Hg, LAP = 15-8 mm Hg despite adequate oxygenation. The PaO2 remained between 64 and 89 mm Hg on 50-60 percent O2.

Despite the poor prognosis, coronary bypass grafts to the first diagonal, LAD and RCA were constructed and the patent foramen ovale was sutured closed.

The patient did well initially. However, mild hypotension continued and did not improve with varying pacing rates, and therapy with dobutamine or isoproterenol. He developed pneumonia, sepsis, worsening hypotension and died.

DISCUSSION

Right-to-left shunting through the patent foramen ovale has been reported in the adult respiratory distress syndrome, pneumonectomy, and coronary artery bypass graft surgery as well as RV myocardial infarction. 1.14 The incidence of right-to-left shunting and RV myocardial infarction is not known, but could approach the incidence of patent foramen ovale (25 percent). Patients with RV myocardial infarction and hypoxia should be investigated for right-to-left shunting with contrast echocardiography, Doppler, indicator dye curves or O2 saturation studies.