Secondly, the patient's chest pain was of concern. An electrocardiogram in the emergency room revealed no acute change consistent with myocardial infarction. Consequently, the differential of expanding aortic aneurysm vs dissection was entertained. In a marfanoid patient with significant aortic regurgitation and chest pain, aortography was not used due to the concern over the large amount of contrast load that would be required to visualize the dilated aorta. The vasodilating property of the dye might further compromise coronary flow. Also, the large amount of contrast load regurgitating into the left ventricle may have caused severe myocardial depression. Magnetic resonance imaging, which uses the natural contrast created between flowing blood and the vessel wall, was the diagnostic modality of choice and revealed the fusiform aneurysm with no evidence of ascending or descending aortic dissection. The information was vital in the preoperative analysis to determine the extent of the disease, urgency, and type of operation to be performed.

In conclusion, to our knowledge, this is the first case report of acute hypertension in a marfanoid patient with an expanding ascending aortic aneurysm treated with intravenous labetalol. Also, this case report shows MRI as the initial diagnostic modality to differentiate aortic aneurysm vs dissection. This imaging technique was the only test required to give the surgeon all the information to perform the aortic valve and aortic root replacement successfully. This approach deserves further study as a possible alternative to traditional management in this disorder.

REFERENCES

Pulmonary Malignant Angioendotheliomatosis

Presentation with Fever and Syndrome of Inappropriate Antidiuretic Hormone

John T Pellicone, M.D.; Howard B. Goldstein, M.D.

A 64-year-old man presented with protracted fever, hypotension, and mononeuritis multiplex. Inappropriate antidiuretic hormone secretion was established. The absence of pulmonary infiltrates precluded any lung biopsy. Autopsy revealed malignant angioendotheliomatosis involving multiple organs including the alveolar septa and pulmonary vasculature. An early diagnosis of MAE in the setting of fever and SIADH may be possible via transbronchial biopsy. (Chest 1990; 98:1292-94)

The syndrome of inappropriate antidiuretic hormone secretion is associated with several infectious and neoplastic disorders. The concomitant presence of fever does not always confirm infection as the cause. We present a case of a rare hematolymphoid neoplasm with diffuse pulmonary vascular involvement characterized by prolonged fever and SIADH.

CASE REPORT

A 64-year-old man was admitted to the hospital for evaluation of weakness and confusion. The patient had been well until three months prior to admission when he began to have progressive aching and weakness of the lower legs.

On the evening of admission, the patient had become confused. The temperature was 97°F (99°F), and results from the physical exam were normal except for moderate weakness of the distal leg muscles. Pertinent lab values included a sodium level of 119 mmol/L and a creatinine value of 0.7 mg/dL.

On the third hospital day, the temperature rose to 38.4°C (101.2°F). Electromyographic studies of the legs revealed a pattern of mononeuropathy multiplex. Urine osmolality was 558 mosm/L while serum osmolality was 255 mosm/L. A CT scan of the brain and chest were normal. A lumbar puncture was normal.

On the 30th hospital day, the patient remained febrile with a fever of 40°C (104°F). The patient was empirically started on therapy with isoniazid and rifampin. On the 32nd hospital day, a fiberoptic bronchoscopic examination was performed revealing normal airways.

On the 58th hospital day, an angiographic study of the celiac plexus and right renal artery revealed normal results with no evidence of vasculitis. On the 65th hospital day, he developed hypotension and dyspnea requiring dopamine. He was given antibiotics intravenously. Due to persistent hypotension and dyspnea, a Swan-Ganz pulmonary artery catheter was inserted. Normal chamber pressures were recorded. On the 71st hospital day, the patient required endotracheal intubation and mechanical ventilation. He lapsed into unconsciousness and died on that day.

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Pathologic Findings

The autopsy revealed widespread intravascular proliferation of large mononuclear cells some of which were multilobar (Fig 1 and 2). Immunoperoxidase stains showed these cells to be positive for leukocyte common antigen and negative for cytokeratin. The histologic features were those of a high grade non-Hodgkin's malignant lymphoma. The organs involved included the mesenteric and retroperitoneal lymph nodes, hepatic sinusoids, both adrenal medullas, the kidneys, and lungs.

The adrenals were particularly involved exhibiting medullary hemorrhagic gray neoplastic tissue. No other organs had features suggesting mass lesions. The autopsy permission did not allow examination of the brain. The major thoracic spinal roots grossly appeared to be normal but were microscopically found to be infiltrated by the neoplastic cells described. The spinal cord was not involved. Alveolar septa were widened by infiltrating lymphoma cells (Fig 3), and several glomeruli were also filled by these cells. Mesenteric lymph node involvement was diffuse and associated with acute hemorrhage. A section of the skin did not reveal the presence of lymphoma cells.

Discussion

Malignant angioendotheliomatosis is a rare condition characterized by intravascular proliferation of tumor cells within the small vessels of many organs. Although Pfieger and Tappeiner originally described it as a cutaneous small vessel neoplasm because of the predilection for skin involvement, it has since come to be known as a systemic disorder with a particular affinity for the central nervous system.

Most case reports describe focal neurologic deficits with progressive dementia and skin lesions. Unlike many malignant lymphoproliferative disorders, significant fever is uncommon as a presenting symptom in cases of MAE until the terminal stages when an obvious secondary infection is present. We believe our case represents the first report of MAE presenting with protracted fever together with SIADH.

The malignant cells of MAE were initially thought to be of endothelial origin since they were confined to the vascular lumen and there appeared to be a transition from normal endothelial cells to malignant cells within the involved blood vessels. Simultaneous reports of MAE occurring in patients with primary malignancies led to the suspicion that MAE might be the vascular manifestation of these solid tumors. It was Ansell et al who forwarded the possibility that MAE may be an unusual manifestation of lymphoma since EM analysis showed no transitional change and no Wiebel-Palade bodies. Bhawan et al found no factor VIII-related antigen (FVIII-Ag), (an endothelial marker) on MAE cells but did find T and B cell markers. Wrotnowski et al reported that MAE cells stained strongly for leukocyte common antigen but negatively for FVIII-Ag and Ulex europaeus agglutinin 1. The B cell lineage has been confirmed by Sheibani et al and by Wick et al who used monotypic surface-bound IgM markers.

Carroll et al speculated that MAE cells lack specific surface receptors that enable these malignant cells to migrate extravascularly. Persistent showers of tumor emboli throughout the vasculature may account for the protracted fever without microorganism infection seen in our patient.

Reports of lung involvement by MAE are usually found as brief comments in autopsy reports with associated clinical symptoms. Strouth et al reported a patient in whom the pulmonary vessels were involved with tumor and the alveolar walls were thickened by engorged capillaries. Dolman et al described pulmonary vascular involvement by MAE as well as pulmonary emboli. Fulling and Gernell were the first to report pulmonary lymphatic channel involvement by

Figure 1. Pulmonary arteriole filled with neoplastic cells (hematoxylin-eosin, original magnification × 400).

Figure 2. Multilobated malignant lymphoma cells in lymph node (hematoxylin-eosin, original magnification × 1,000).

Figure 3. Alveolar septum infiltrated by lymphoma cells (hematoxylin-eosin, original magnification × 500).
MAE. Wick et al. reviewed 15 cases of MAE, 14 of which showed pulmonary vascular involvement. However, in none of the case reports did the premortem clinical signs and symptoms direct the physician to the lung for biopsy and diagnosis.

In the series by Wick et al. reporting 15 patients, 11 had fever of unknown origin and two had hyponatremia. Massive infiltration of the intraadrenal blood vessels leading to adrenal insufficiency has been forwarded as a possible explanation for fever, hyponatremia, and hypotension in MAE patients. We feel the clinical evidence in our case does not suggest hypoadrenalism, but is consistent with SIADH.

SIADH is a well-known consequence of both malignant tumors and pulmonary disease. Infected lung tissue may synthesize ADH, most notably in tuberculosis. The consideration of pulmonary tuberculosis in our patient prompted the bronchoscopy. The absence of significant pulmonary infiltrates precluded a transbronchial biopsy. No evidence of tuberculosis was discovered at autopsy, but pulmonary vascular infiltration by the cells of MAE was found. The availability of a specimen of lung tissue might well have led to a premortem diagnosis of MAE and a trial of chemotherapy. We suggest that SIADH in the setting of fever may warrant the consideration of a transbronchial biopsy despite the absence of parenchymal infiltrates.

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Electrocardiographic J Waves after Resuscitation from Cardiac Arrest*

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A patient was monitored prior to, during, and after cardiac arrest with a Holter monitor and an electrocardiograph. The arrest occurred without any premonitory signs on the ECG. At the onset of the arrest, torsades de pointes ventricular tachycardia occurred, which quickly degenerated into ventricular fibrillation. After a successful second defibrillation, the patient developed Osborn waves, which subsided within a few minutes. (Chest 1990; 98:1294-96)

The report of the findings in a patient who was monitored with multilead ECGs during cardiac arrest and successful resuscitation is presented.

Case Report

A 53-year-old white man suffered a lateral wall myocardial infarct one month prior to this event. Coronary angiography revealed that the left anterior descending coronary artery had a 100 percent occlusion, the diagonal artery had a 95 percent occlusion, and the posterior descending branch had a 50 percent occlusion. There was a small area of hypokinesia in the inferior wall. The left ventricular ejection fraction was 65 percent. The patient weighed 79 kg and was receiving diltiazem (80 mg four times daily). He was brought for aortocoronary bypass surgery.

Following informed consent for perioperative monitoring, a Holter monitor (Marquette 8500) was used to record a modified CM lead with the positive electrode at V1 and the negative electrode below the right clavicle in the midclavicular line. Modified leads 3 and 1 with electrodes on the torso were also monitored. A digital electrocardiograph (Marquette MAC-12) was also used to record modified limb leads with electrodes on the torso and at V1 and V6.

On the morning of surgery, the patient received 10 mg of diazepam orally at 6 AM and 1 mg of lorazepam intravenously at 7:12 AM and again at 7:17 AM, during the placement of an introducer in the right internal jugular vein. A few minutes later, he was noted to be cyanotic, unresponsive, and in ventricular fibrillation. Cardiopulmonary resuscitation was performed, and defibrillation was attempted with 200 J and was successful with 300 J. Sinus rhythm returned, and satisfactory blood pressure was obtained. Infusion of nitroglycerin was started at 1 μg/kg·min.

Central nervous system damage was expected to be unlikely due to the arrest. It was decided to proceed with surgery. Fentanyl (25 μg/kg) and pancuronium (0.1 mg/kg) were administered, and the patient was intubated. The cardiopulmonary bypass was started 65 minutes later and lasted 90 minutes. A myocardial infarct was ruled out on the basis of intraoperative inspection of the myocardium and postoperative cardiac enzyme levels and ECGs. No neurologic or other deficits were noted after surgery.

Periarrest ECG

This monitored cardiac arrest is different in several respects from those reported earlier.‡ This patient had no previous history of

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