CASE 6

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Clinical Abstract:

M.L. was a 75 year old white man who was in excellent health
until three years antemortem when he began to note progressive
bilateral multiple joint pain and swelling. He was initially treated
with NSAID, but he subsequently required Prednisone therapy for
approximately one year antemortem. His past medical history was
notable for >100 pack years of cigarette smoking.

M.L. presented to clinic 2 months antemortem with a 6 month
history of progressive general malaise, anorexia, dyspnea on
exertion and a 60 pound weight loss. He denied fevers, chills or
night sweats. A chest x-ray revealed a poorly-defined nodular
density in the lateral right upper lobe. The lung fields were
otherwise clear. An EGD revealed candidal esophagitis attributed to
chronic steroid therapy. He was treated with a 2 week course of
oral Nystatin.

M.L. was last admitted 6 weeks antemortem for further
evaluation of weight loss. A repeat EGD showed resolution of the
esophagitis. He was anergic to PPD and control skin test antigens.
An abdominal CT scan revealed only pleural and pericardial
effusions. An ECG showed first degree AV block and bundle branch
blocks. Shortly after admission, he had an episode of high anterior
chest pain of 30 minutes duration without radiation or diaphoresis.
A repeat ECG showed second degree AV block necessitating placement
of a pacemaker. A myocardial infarct was ruled out by serial
cardiac isoenzymes. High resolution electrocardiography revealed
possible intra-Hisian disease. An echocardiogram showed fused,
thickened aortic valve cusps and a moderate to large pericardial
effusion.

M.L. continued to have recurrent episodes of atypical chest
pain attributed to angina, pericarditis, or recurrent esophagitis.
Serial chest x-rays showed persistent bilateral effusions and
atelectasis. He subsequently had a fatal cardiorespiratory arrest.

The patient had no clinical signs or symptoms of neurologic
disease throughout his course. The brain weighed 1430 gm. The
cerebral dura and falx cerebri were thickened and had focal
nodularity. There was diffuse, firm, homogeneous, yellow-white
thickening of the leptomeninges along the medial surfaces of the
cerebral cortex adjacent to the falx.

Material submitted: One H&E and one unstained slide from the left
medial parietal lobe

Points for discussion: 1. Diagnosis
2. Associated systemic findings
3. Clinicopathologic correlations