primary mediastinal seminoma. This is unlikely to be metastatic disease from the primary mediastinal seminoma, mainly due to the long interval between the diagnosis, and the absence of extracranial lesions. We offer this as additional proof of a genetic predisposition to GCTs, which is congruent with the current data available on the incidence of second primaries in treated testicular cancers. The 15-year actuary risk of a contralateral testis cancer has been reported to be of 2.4% by a Dutch tumor registry.[6] The occurrence of metachronous GCTs is very rare and has been reported in isolated case reports.[7-14]

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The authors indicated no potential conflicts of interest.

Intravascular Hemolysis As a Complication of Clostridium Perfringens Sepsis

After presenting with acute onset painless obstructive jaundice, a previously healthy 49-year-old man was diagnosed with a small cell-like neuroendocrine variant of metastatic pancreatic cancer to the liver. Two weeks after receiving his first cycle of cisplatin/etoposide chemotherapy, the patient contacted our service with a 2-day history of feeling generally unwell. Given the absence of fever or focal symptoms, he declined clinical assessment at that time. Two hours later, the patient's sister called and described a significant clinical deterioration. He now complained of diffuse chest and abdominal pain, myalgias, nausea, and rapidly progressive dyspnea. On emergency assessment, the patient appeared unwell, diaphoretic, tachypneic, severely jaundiced, and extremely restless. On requestioning, his sister confirmed that the jaundice was a new development over the previous 2 hours. His blood pressure was 130/70, his heart rate was regular at 132 beats per minute, and his oxygen saturation was 95% on room air. He was now febrile at 38.3°C (101°F). His respiratory rate was 46 breaths per minute and he was unable to recline from an upright position because of worsening dyspnea. His chest was clear. Cardiac examination was unremarkable and he was euvolemic. His abdomen was diffusely tender but soft with no peritoneal signs. Neurologic examination was normal. Urgent bloodwork and plain films of the chest and abdomen were arranged.

Soon after the blood was collected and the imaging was completed, laboratory personnel reported that they were unable to process any of the requested bloodwork because of gross hemolysis. The blood film showed gross intravascular hemolysis with reddish background staining secondary to gross hemoglobinemia, many spherocytes (Fig 1, arrows), numerous ghost erythrocytes (Fig 1, arrowhead), and toxic changes in neutrophils with Dohle bodies. No organisms were identified. The CBC was estimated as follows: hemoglobin, less than 50; hematocrit, 0.06; neutrophils, less than 1.0; and platelets, less than 150. Within minutes of this report, the patient collapsed. A pulseless electrical activity arrest quickly ensued. Despite resuscitation efforts, the patient died shortly thereafter. Of note, the time from presentation to death was less than 90 minutes. As illustrated in Figure 2, the chest x-ray (which became available postmortem) demonstrated pneumoperitoneum. Clostridium perfringens bacteremia was reported by blood culture 2 days later.

C. perfringens is an anaerobic Gram-positive rod which produces at least a dozen exotoxins, including hemolysin. It has an extraordinary potential for rapid proliferation with an approximate doubling time of only 7 minutes.[1] The *clostridial* hemolysin is an α-toxin, which hydrolyzes phospholipids in RBC membranes. The resulting spherocytes are exquisitely sensitive to osmotic lysis.[2] Classically, *C. perfringens* sepsis and hemolysis have

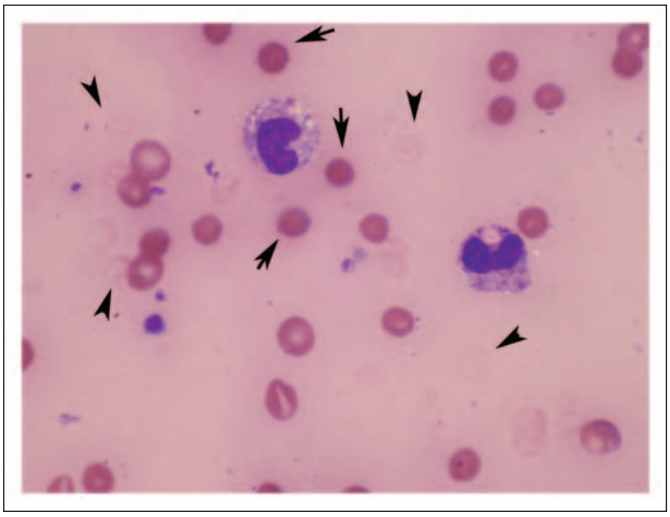

Fig 1.

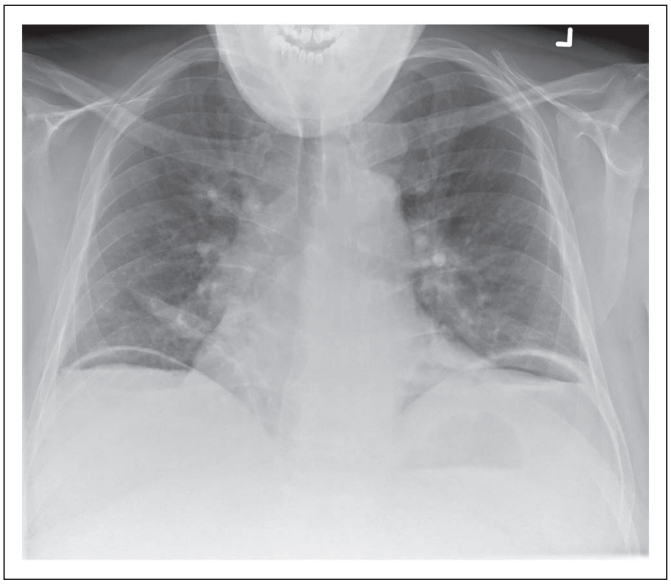

Fig 2.

been reported in cases of postpartum or postabortion infections, but today are more frequently observed with malignancies or intra-abdominal infections.[3]

This case demonstrates the dramatic nature of *C. perfringens* sepsis with associated massive hemolysis. The specific incidence of this entity is unknown. However, only two cases of massive hemolysis were identified among the 136 cancer patients at M.D. Anderson Cancer Center (Houston, TX) with *Clostridial* bacteremia over a period of 11 years.[4] Of these patients, one had acute myeloid leukemia, and the other had colorectal cancer. Similarly to our patient, both of these patients died within hours of presentation. There are very few additional cases of *C. perfringens* associated massive hemolysis reported in the literature, and the mortality rate of these rare cases approaches 100% in patients with an underlying malignancy.[5,6] Of the few cases reported, gastrointestinal and genitourinary malignancies are the most commonly associated solid tumors. To our knowledge, there has never been a documented neuroendocrine or small cell tumor association.

Due to the dramatic natural history of *C. perfringens* associated massive intravascular hemolysis, the therapeutic window for instituting medical and/or surgical treatment is extremely narrow. Therefore, *C. perfringens* sepsis should always be considered in the differential diagnosis of a cancer patient presenting with fever and hemolysis.

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Paraneoplastic Erythropoietin-Induced Polycythemia Associated With Small Lymphocytic Lymphoma

A 64-year-old man presented with a 3-month history of intermittent abdominal pain with cramps, documented fevers of 39.5C, drenching night sweats, and small lumps on the right side of his neck for 1 year. Past medical history was unremarkable; the patient is a life-long nonsmoker and has no prior history of respiratory or cardiac disease. Physical examination revealed palmar erythema, peripheral lymphadenopathy, and a palpable large central abdominal mass. He had no hepatosplenomegaly. Computed tomography (CT) scan of the abdomen and pelvis revealed extensive retroperitoneal lymphadenopathy, with a normal spleen. Lymph node biopsy and bone marrow examination showed morphologic and immunohistochemical features consistent with small lymphocytic lymphoma. Pretreatment hemoglobin of 19.7 g/dL (normal range, 13.0 to 18.0 g/dL), hematocrit of 59% (normal range, 40% to 50%), with normal platelet and white cell/differential counts. He had a routine CBC 2 years previously that showed a normal hemoglobin and hematocrit at 13.5 g/dL and 40%, respectively. Pretreatment evaluation to identify the cause of the patient's polycythemia was undertaken. Serum erythropoietin (EPO) level was 93.0 mU/mL (normal range, 3.3 to 16.6 mU/mL), and stem cell culture assay revealed no evidence of EPO-independent erythroid colony growth, both findings rule out polycythemia rubra vera (PRV). No other causes of secondary polycythemia were identified; normal CT scan of the head (ruling out cerebellar hemangioblastoma), no evidence of renal, adrenal, or liver tumors. He had no history of

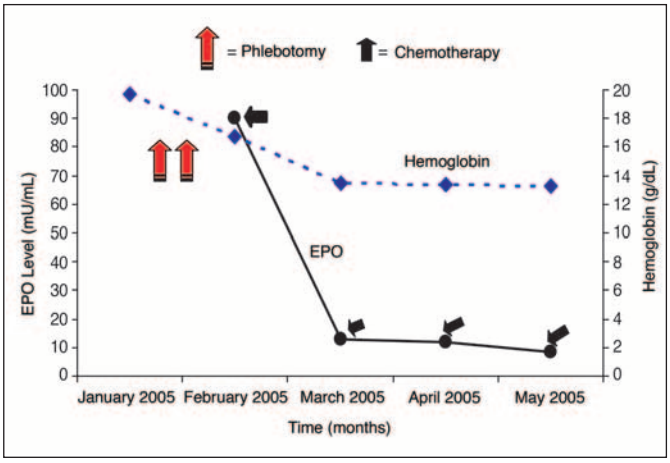

Fig 1.