Metastatic pancreatic adenocarcinoma presenting with immune thrombocytopenic purpura is a very rare association. To date, only 1 case report found in the literature delineates such an association. We present a case of a patient with newly diagnosed, biopsy-proven metastatic pancreatic adenocarcinoma with new-onset immune thrombocytopenic purpura. The patient’s platelet count returned to normal limits after being treated with oral corticosteroid therapy. In conclusion, immune thrombocytopenic purpura can be associated with metastatic pancreatic adenocarcinoma and responds well to corticosteroid therapy.

Pancreatic cancer is the fourth most common cause of adult cancer death in the United States. The high mortality rate from pancreatic cancer is a result of often-subtle clinical manifestations and a high incidence of metastatic disease at the time of diagnosis.

Pancreatic cancer manifesting with associated immune thrombocytopenic purpura has been previously reported in 1 case report in the literature. We present a case of a patient who presented with newly diagnosed metastatic pancreatic cancer with new-onset immune thrombocytopenic purpura.

A 60-year-old man who was previously healthy presented to the Emergency Department (ED) with 1 month of fatigue and a 20-pound unintentional weight loss. Over the past week, his friends had noticed yellowing of his skin, and he had noticed prolonged bleeding after shaving. The patient was on no medications prior to presentation. He denied any history of alcohol or tobacco use and reported no family history of malignancy. Review of systems was otherwise negative.

On physical examination, the patient was afebrile and hemodynamically stable. He was alert and oriented, pleasant and cooperative, but his skin was visibly jaundiced with associated scleral icterus, and his face had scattered cuts from the morning’s shave. Otherwise, there were no petechiae or ecchymoses appreciated. There was no hepatosplenomegaly, no fluid wave, and no stigmata of chronic liver disease. A neurologic examination revealed no focal deficits, and cardiac examination revealed a regular rate and rhythm, +S1S2, no murmurs, rubs, or gallops, and his lungs were clear to auscultation bilaterally.

Liver tests revealed a direct hyperbilirubinemia with a total bilirubin of 13.2 (0.3-1.9 mg/dL), a direct bilirubin of 11 (0-0.3 mg/dL), an alkaline phosphatase of 323 (39-117 U/L), elevated transaminases with an aspartate aminotransferase of 430 (5-40 U/L), and an alanine aminotransferase of 82 (7-56 U/L). A complete blood count revealed a severe thrombocytopenia with a platelet count <10,000 (150,000-450,000 platelets/microliter), a mild anemia with a hemoglobin of 11.9 (12.5-16 grams/dL), and a white blood count within normal limits. A chemistry panel revealed serum creatinine of 0.7 (0.7-1.4 mg/dL) and mild hyponatremia with a sodium of 133 (136-145 mEq/L).

A computerized tomography (CT) scan of the abdomen revealed a mass at the head of the pancreas, multiple liver lesions, trace ascites, no splenomegaly, and extensive lymphadenopathy.

To further work up the patient’s thrombocytopenia, a peripheral blood smear was obtained, and this revealed occasional elliptocytes. As the patient was...
afebrile, had baseline normal kidney function, no neurologic findings, no thrombotic events, and no schistocytes on blood smear, a diagnosis of thrombotic thrombocytopenic purpura was not entertained. A diagnosis of Evans syndrome was similarly excluded because the patient’s anemia was very mild, and there were no spherocytes appreciated on the blood smear. The patient was not bleeding spontaneously, or oozing from any intravenous sites, and as there were no red blood cell fragments appreciated on the blood smear, thus making the diagnosis of disseminated intravascular coagulation much less likely. An international normalized ratio was sent, and this returned within normal limits at 1.05 (0.8-1.2), which further argued against a potential diagnosis of disseminated intravascular coagulation. There was no platelet clumping on the blood smear, ruling out pseudothrombocytopenia.

Other diagnostic considerations included medication-induced thrombocytopenia; however, the patient was not on any medications prior to coming into the hospital and had received nothing in the ED before having his first complete blood count.

Two final diagnoses for consideration included immune thrombocytopenic purpura and a primary bone marrow disorder. Given the relatively isolated nature of this patient’s thrombocytopenia and lack of any spontaneous bleeding events, immune thrombocytopenic purpura was considered more likely. Additionally, given the platelet count <10,000 platelets/mL, bone marrow biopsy was not deemed the next safest step in diagnosis.

The patient was treated with oral prednisone 1 mg/kg by mouth daily, and over the next 2 days the patient’s platelet count increased from <10,000 to 48,000 platelets/mL. A presumptive diagnosis of immune thrombocytopenic purpura was made on clinical grounds, as other causes of thrombocytopenia, including thrombotic thrombocytopenic purpura, disseminated intravascular coagulation, pseudothrombocytopenia, medication-induced thrombocytopenia, and bone marrow infiltration would not be expected to improve with corticosteroid therapy. Additionally, the patient’s mild anemia did not improve with corticosteroids, arguing against Evans syndrome.

Given improvement in platelet counts, a biopsy of the largest, most accessible liver lesion was then able to be performed safely as identified on CT, revealing adenocarcinoma consistent with metastatic pancreatic cancer. Endoscopic retrograde cholangiopancreatography was then performed, displaying simultaneous strictureing of the proximal portions of the common bile duct and pancreatic duct with distal dilation. A metal stent was placed in the common bile duct for palliation, and the patient was discharged to home hospice. Given the patient’s poor prognosis and normalization of platelet count with corticosteroid therapy, anti-platelet antibodies and a bone marrow biopsy were not performed to confirm a diagnosis of immune thrombocytopenic purpura. Unfortunately, the patient died within 1 month of presentation.

**DISCUSSION**

This case is important for a few different reasons. First, it shows that metastatic pancreatic adenocarcinoma can present with associated immune thrombocytopenic purpura and can respond to corticosteroid therapy. Second, immune thrombocytopenic purpura is a common paraneoplastic manifestation of other malignancies including chronic lymphocytic leukemia, Hodgkin’s lymphoma, and various solid tumors, including breast, lung, and ovarian cancer. The association with leukemia and lymphoma commonly has been reported, and there have been approximately 20 cases of immune thrombocytopenic purpura reported with the various solid tumors.17

To date, it is very difficult to prove that immune thrombocytopenic purpura is a direct paraneoplastic manifestation of any malignancy as there are no specific antibodies for malignancy-related immune thrombocytopenic purpura. There is a serologic test for platelet-bound antibodies, however, the sensitivity of this test is only 49%-66%, and the specificity is 78%-92%. Again, this test simply tests for antibodies associated with immune thrombocytopenic purpura and is not specific for malignancy-related immune thrombocytopenic purpura.17 In this case report, a clinical diagnosis was made based on the temporal correlation of the patient’s presentation.

The association of metastatic pancreatic cancer with immune thrombocytopenic purpura is very rare. After a thorough review of the literature, we were only able to find 1 other case report documenting such an association.2 Typically, when thrombocytopenia is incidentally discovered with metastatic pancreatic cancer, it is attributed to disseminated intravascular coagulation, chemotherapy-induced thrombocytopenia, or medication-induced thrombocytopenia.

This case report proves valuable because this association may be more prevalent than previously thought. In the 2006 case report and this case, it appears the pancreatic adenocarcinoma was diffusely metastatic by the time paraneoplastic immune thrombocytopenic pur-
purpura was discovered. Perhaps the association of immune thrombocytopenic purpura with pancreatic adenocarcinoma is indicative of metastatic disease and could be a harbinger of poor prognosis. More research in this area would prove beneficial in delineating the prognosis of this association and amenability to various treatment options.

CONCLUSION
Metastatic pancreatic adenocarcinoma can present with associated immune thrombocytopenic purpura, which can respond to corticosteroid therapy. Performing invasive procedures such as biopsies of metastatic lesions and endoscopic retrograde cholangiopancreatography with palliative biliary stent placement can be safely performed once the platelet count improves.

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REFERENCES
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