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CASE REPORT | PANCREAS

Spontaneous Tumor Lysis Syndrome Secondary to Metastatic Pancreatic Adenocarcinoma

Jason Gusdorf, MD[1], Netana H. Markovitz, MD[2], Cleveland Ricketts, MD[3], Yuho Ono, MD[3], Barbara D. Lam, MD[4], Jonathan Berry, MD[4], Jacob Dockterman, MD, PhD[2], and Sheela Reddy, MD[5]

[1]*Department of Internal Medicine, Beth Israel Deaconess Medical Center, Boston, MA, United States of America*
[2]*Department of Internal Medicine, Beth Israel Deaconess Medical Center, Boston, MA, United States of America*
[3]*Department of Pathology, Beth Israel Deaconess Medical Center, Boston, MA, United States of America*
[4]*Division of Hematology and Hematologic Malignancies, Department of Internal Medicine, Beth Israel Deaconess Medical Center, Boston, MA, United States of America*
[5]*Division of Gastroenterology, Department of Internal Medicine, Beth Israel Deaconess Medical Center, Boston, MA, United States of America*

ABSTRACT

Spontaneous tumor lysis syndrome (STLS) secondary to metastatic pancreatic adenocarcinoma is a rare clinical phenomenon. An 86-year-old woman with a history of pancreatic cysts presented to the emergency department with progressive fatigue, transaminitis, elevated lactate dehydrogenase, and acute kidney injury of unclear etiology. Abdominal imaging and celiac lymph node biopsy were consistent with metastatic pancreatic adenocarcinoma. Her clinical status deteriorated requiring intensive care unit transfer, and her laboratory results were found to be consistent with STLS. Despite treatment, she entered multisystem organ failure and died shortly after. This case adds to the literature of STLS in pancreatic adenocarcinomas.

KEYWORDS: Spontaneous tumor lysis syndrome; pancreatic adenocarcinoma; pancreatic cyst; acute kidney injury; oliguric renal failure; uric acid; gastrointestinal malignancy; pathology

INTRODUCTION

Spontaneous tumor lysis syndrome (STLS), characterized by spontaneous destruction of tumor cells with release of intracellular contents including uric acid, potassium, and phosphate, can lead to death from electrolyte abnormalities and end-organ damage. Typically, STLS occurs with leukemias and lymphomas. It is seldom observed in solid malignancies, with only 3 reported instances arising from pancreatic adenocarcinoma.[1–3] This report details a fourth case of STLS secondary to a metastatic pancreatic adenocarcinoma.

CASE REPORT

An 86-year-old woman with hyperlipidemia, hypertension, and pancreatic cysts presented with a month-long history of progressive fatigue. In the emergency department, vitals were within normal limits, and her examination showed normal mental status, no acute distress, and an unremarkable cardiopulmonary and abdominal examination. Laboratory findings were notable for a creatinine of 3.2 (baseline of 1.0), alanine aminotransferase of 146 U/L, aspartate aminotransferase of 338 U/L, alkaline phosphatase of 377 U/L, total bilirubin of 3.7 mg/dL, lactate dehydrogenase (LDH) of 2,350 U/L, carcinoembryonic antigen (CEA) of 2,316 ng/mL, and carbohydrate antigen 19-9 (CA 19-9) of 468 U/mL. The patient was admitted for evaluation of unexplained transaminitis and acute kidney injury (Tables 1 and 3).

Abdominal computed tomography showed a 7.2 × 5.9 × 6.9 cm hepatic hypodensity and multiple pancreatic cysts, without abnormalities in the small bowel, colon, and rectum (Figure 1). Renal ultrasound displayed normal renal morphology with a 6 mm nonobstructive left renal calculus.

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Correspondence: Jason Gusdorf, MD (jgusdorf@bidmc.harvard.edu).

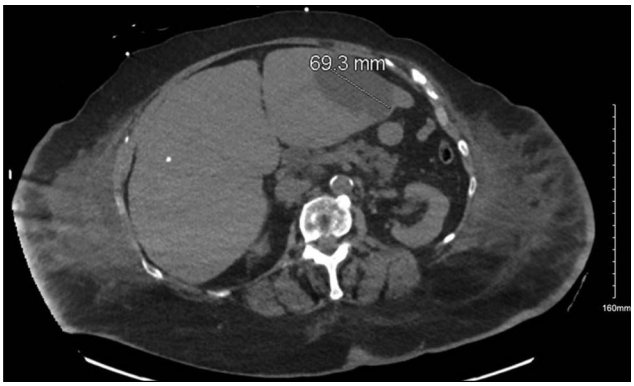

Figure 1. Computed tomography scan demonstrating a large 7.2 × 5.9 × 6.9 cm hypodensity in the patient's liver.

On routine screening 4 years before admission, a colonoscopy had shown a single 5 mm sessile serrated adenoma that was removed. A pancreatic cyst fluid sample 1 year before admission yielded a CEA level of 37.5 ng/mL, not consistent with a mucinous cyst, which is defined by a CEA level above 192 ng/mL.

Endoscopic ultrasound was performed for further assessment of the pancreas and identified main pancreatic duct dilation up to 1 cm and multiple large cysts without a discrete mass, the largest of which was 4.0 cm (Figure 2). A fine needle aspiration of the pancreatic cyst and a fine needle biopsy of a celiac lymph node were obtained during the procedure. The pancreatic cyst sample was nondiagnostic. Histologic evaluation of the lymph node sample showed malignant glands in fibrotic stroma, consistent with adenocarcinoma (Figure 3). The tumor cells demonstrated high nuclear-to-cytoplasmic ratio, with large, ovoid pleomorphic nuclei, and some with foamy cytoplasm (Figure 3). By immunohistochemical stains, the tumor cells were positive for CK7, CK20, and CDX2 (Figure 3). They were negative for Thyroid Transcription Factor-1, Napsin-A, estrogen receptor, GATA Binding Protein 3, and Paired Box Gene 8. The overall morphologic and immunophenotypic features, in accordance with the clinicoradiological findings, were compatible with a pancreatic primary. No other sources of malignancy were identified.

On day 4 postadmission, the patient developed oliguric renal failure, with creatinine rising to 4.7 mg/dL and urine output under 150 mL/d. Laboratory results showed an increased LDH at 3,410 U/L, uric acid at 21.8 mg/dL, phosphate at 5.4 mg/dL, sodium at 129 mmol/L, potassium at 5.7 mmol/L, calcium at 8.8 mg/dL, and bilirubin at 6.7 mg/dL with 5.5 mg/dL direct. These findings raised suspicion for STLS secondary to pancreatic adenocarcinoma (Tables 1 and 2).

The patient was treated for STLS with 6 mg intravenous rasburicase, 300 mg allopurinol, lactated Ringer solution, as well as sodium zirconium cyclosilicate, insulin, dextrose, and intravenous furosemide for hyperkalemia.

Figure 2. Endoscopic ultrasound showing a massively dilated pancreatic duct and numerous large cysts. The largest cyst measured approximately 4 cm in the head/neck. No discrete pancreatic mass is seen.

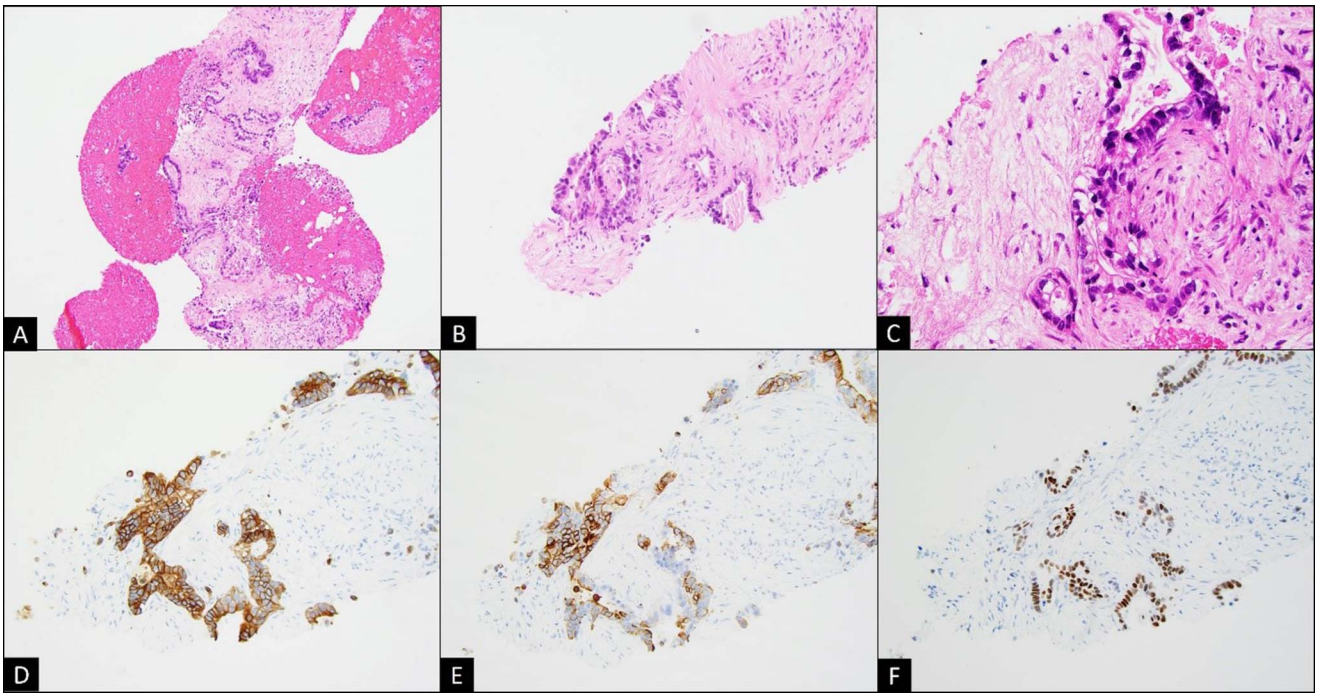

Figure 3. Lymph node biopsy specimen showing adenocarcinoma (A–C). The tumor is immunoreactive for CK7 (D), CK20 (E), and CDX-2 (F), compatible with pancreatic origin.

Table 1. Laboratory results

Laboratory result	Day of admission	Day of ICU transfer
White blood cell	10.6	16
Hemoglobin	9.7	9.3
Platelets	220	187
Glucose	99	86
Blood urea nitrogen	40	64
Creatinine	3.2	4.6
Sodium	134	127
Potassium	4	5.2
Chloride	97	92
Bicarbonate (HCO3)	19	15
Anion gap	18	20
Alanine Aminotransferase (ALT)	146	155
Aspartate Aminotransferase (AST)	338	483
LD (LDH)	2,350	3,410
Total bilirubin	3.7	6.8
Calcium	8.6	8.9
Phosphate	4.4	5.7
Magnesium	1.9	2.2
Uric acid	—	16
Lactate	—	3.5
Hepatitis B Surface Antigen (HBsAg)	Negative	—
Hepatitis B Surface Antibody (HBsAb)	Positive	—
Hepatitis C Surface Antibody (HBcAb)	Positive	—
Hepatitis B Virus Viral Load (HBV VL)	Negative	—
Hepatitis A Virus Antibody (HAV Ab)	Negative	—
Acetylsalicylic Acid (ASA)	Negative	—
Ethanol	Negative	—
Acetaminophen	Negative	—
Anti-smooth muscle antibody	Negative	—
Anti-mitochondrial antibody	Negative	—
Liver kidney microsome type 1 (Lkm-1) antibody	Negative	—
Anti-nuclear antibody	Negative	—

ICU, intensive care unit; LDH, lactate dehydrogenase.

The patient's condition deteriorated rapidly, necessitating intensive care unit transfer. Subsequent multiorgan failure ensued, attributed to an aggressive malignancy and STLS-induced oliguric renal failure. After discussions with her family, care was transitioned to comfort measures only. The patient died 4 days later.

DISCUSSION

This is the fourth known case report of STLS secondary to a pancreatic adenocarcinoma. Although renal failure from

Table 2. Tumor lysis syndrome laboratory results

Laboratory result	Day of admission	Day of ICU transfer
Potassium	4	5.2
Calcium	8.6	8.9
Phosphate	4.4	5.7
Uric acid	—	16
LD (LDH)	2,350	3,410

ICU, intensive care unit; LDH, lactate dehydrogenase.

other causes can lead to a secondary rise in uric acid, potassium, and phosphate, the extremely high uric acid levels and escalating levels of LDH in this case are more consistent with the rare but documented phenomenon of STLS. No other causes of renal failure were identified. While none of the pancreatic samples showed direct evidence of carcinoma, there were multiple findings that make pancreatic adenocarcinoma the most likely underlying malignancy. These include elevated serum CEA and carbohydrate antigen (CA) 19-9 levels and a celiac lymph node biopsy which showed adenocarcinoma with immunomorphologic features most consistent with pancreatic origin in the absence of other sources of malignancy. The patient had a normal colonoscopy done 4 years before presentation, and abdominal and pelvic computed tomography (CT) at the time of admission showed a normal caliber small bowel, colon, and rectum.

In such a patient who has high-risk findings of disseminated neoplastic disease with acute kidney injury, obtaining a uric acid level early is important in identifying TLS. This case adds to the literature of STLS in pancreatic adenocarcinomas and suggests that STLS should be considered in patients with widespread metastatic disease presenting with otherwise unexplained acute kidney injury.

Table 3. Malignancy markers

Laboratory result	Day of admission
Carcinoembryonic Antigen (CEA)	2,316
alpha-fetoprotein (AFP)	6.71
Carbohydrate antigen (CA) 19-9	468

DISCLOSURES

J. Gusdorf: primary writer and editor, and is the article guarantor. NH Markovitz: secondary writer and editor. C. Ricketts: writer of pathology section; BD Lam: Heme/onc consultants, editor; J. Berry: Heme/onc consultants, editor; Y. Ono: writer of pathology section. J. Dockterman: writer and editor. S. Reddy: primary GI supervising attending and editor.

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Informed consent was obtained for this case report.

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