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Case Report

Acute tumor lysis syndrome after proximal splenic artery embolization

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ABSTRACT

Preoperative splenic artery embolization for massive splenomegaly has been shown to reduce intraoperative hemorrhage during splenectomy. We describe a case of tumor lysis syndrome after proximal splenic artery embolization in a patient with advanced mantle cell lymphoma and splenic involvement. The patient presented initially with hyperkalemia two days after embolization that worsened during splenectomy. He was stabilized, but developed laboratory tumor lysis syndrome with renal failure and expired. High clinical suspicion of tumor lysis syndrome in this setting is advised. Treatment must be started early to avoid serious renal injury and death. Lastly, same day splenectomy and embolization should be considered to decrease the likelihood of developing tumor lysis syndrome.

Introduction

Open splenectomy for massive splenomegaly (drained splenic weight, >1000 g) is associated with a high morbidity and mortality [1,2]. The most common complication of this surgery is uncontrolled intraoperative hemorrhage [2]. Preoperative splenic artery embolization has been shown to reduce intraoperative bleeding and has been widely used for both open and laparoscopic techniques [1,2]. There are 2 major modalities of splenic artery embolization: proximal splenic artery embolization, defined as embolization of the main trunk of the splenic artery; and distal splenic artery embolization, defined as embolization of one or more of the terminal branches of the splenic artery [3]. Proximal splenic artery embolization has a lower risk of splenic infarction compared with distal splenic artery embolization because of the extensive collateral arteries (eg, short gastric arteries) supplying the spleen [4]. However, most patients with massive splenomegaly are late in their disease course, conferring a high rate of hematologic complications (eg, anemia, thrombocytopenia, leukopenia, and pancytopenia) [5]. Here, we present a case of acute tumor lysis syndrome after proximal splenic artery embolization.

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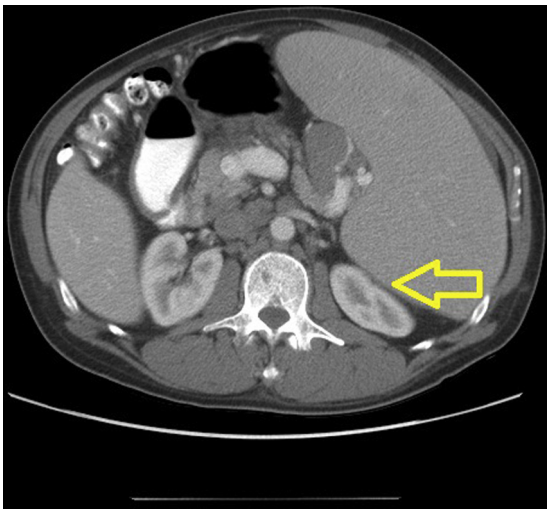

Fig. 1 – A 58-year-old man with massive splenomegaly. Axial contrast-enhanced CT of the abdomen showing splenomegaly with compression of the left renal parenchyma (arrow).

Case report

Our patient is a 58-year-old man with a medical history of mantle cell lymphoma refractory to chemotherapy with 8 months of worsening splenomegaly and thrombocytopenia. He presented to the emergency department with a 5-day history of persistent left upper quadrant abdominal pain. The spleen was palpable on examination, and measured 23 × 22 × 10 cm^3 on computed tomography scan (Fig. 1), with an approximate volume of 2964.8 cm^3 using the splenic volume formula [6]. On the morning of the procedure, the laboratory values were significant for leukocyte count of 11.6/mm^3, platelet count of 46,000/μL, potassium of 3.0 mEq/L, phosphate of 1.5 mg/dL, blood urea nitrogen (BUN) of 37 mg/dL, and creatinine (Cr) of 1.27 mg/dL. Pretreatment lactate dehydrogenase and uric acid were not available. Proximal splenic artery embolization was performed with an Amplatzer plug (St. Jude Medical, St. Paul, AK) and coils. Additional coils were necessary because of persistent forward flow 15 minutes after plug deployment (Fig. 2A and B).

Two days after the procedure, the patient's leukocyte count was 32.5/mm^3, potassium was 6.1 mEq/L, phosphate was 3.6 mg/dL, BUN was 28 mg/dL, and Cr was 1.52 mg/dL. Calcium gluconate 1 g intravenous was administered for the hyperkalemia. The patient then underwent an open splenectomy. One liter of ascites was seen and suctioned on entering the peritoneal cavity. The spleen was then exposed, revealing multiple areas of infarction. The hyperkalemia continued to worsen throughout the surgery, prompting the anesthesia team to transfuse insulin, glucose, and bicarbonate. Potassium rose to a maximum of 7.8 mEq/L intraoperatively before stabilizing at 5.0 mEq/L the following day. Of note, pathology of the spleen was significant for a gross weight of 3880 g, and diffuse effacement of normal parenchyma by neoplastic cells.

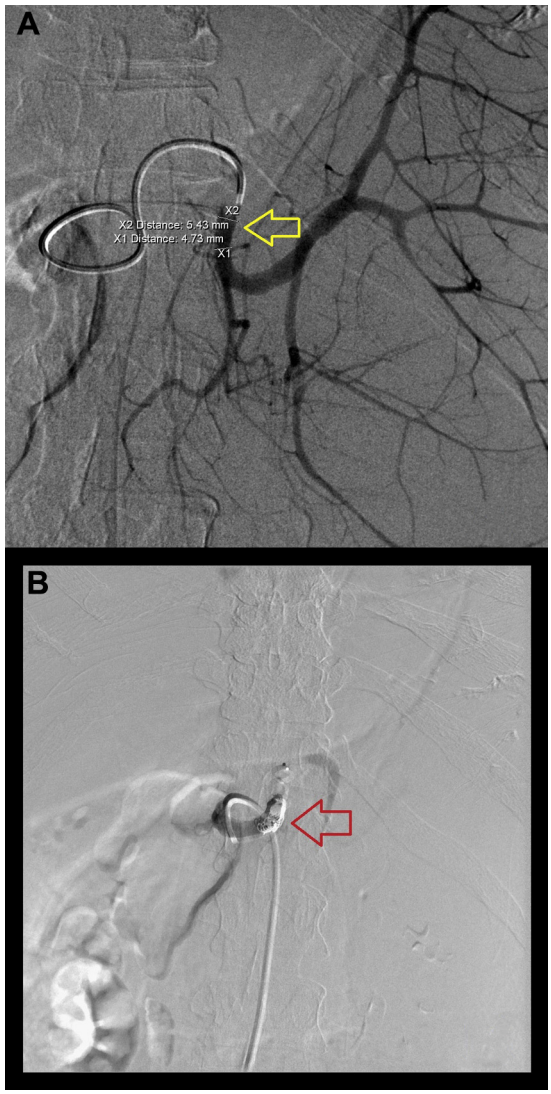

Fig. 2 – (A) Preembolization angiogram showing tip of catheter proximal to the first branch of the splenic artery (arrow). (B) Postembolization angiogram showing coil (arrow) at main trunk of splenic artery with minimal distal flow.

Postoperatively, the patient remained intubated on continuous positive airway pressure support for 24 hours and was extubated with no neurologic sequelae. Immediate postoperative course was complicated by nonoliguric renal failure confirmed by rising BUN and Cr. Renal function improved with maintenance D5W at 150cc/H and albumin boluses as needed. Leukocytosis also improved but began to rise again on the fourth postoperative day. The patient also developed concomitant massive ascites and bilateral pulmonary edema with effusions seen on chest radiographs. Furosemide was started, but he subsequently developed respiratory distress on the 12th postoperative day. An 8-French pigtail catheter was emergently placed in the right lower quadrant to improve the ascites, which immediately drained chylous fluid with a triglyceride level of 461 mg/dL.

Laboratory results on postoperative day 11 were significant for uric acid of 15.7 mg/dL, potassium of 5.4 mEq/L, phosphorous of 4.9 mg/dL, BUN of 64 mg/dL, and Cr of 2.65 mg/dL. Rasburicase was started for suspected tumor lysis syndrome. On postoperative day 15, the patient experienced asymptomatic oxygen desaturation and was intubated. Emergent hemodialysis was started for severe volume overload, but he went into cardiac arrest and expired.

Discussion

Tumor lysis syndrome refers to a metabolic dysregulation caused by the rapid breakdown of malignant cells. It is most commonly seen after the initiation of chemotherapy for highly proliferative lymphomas and leukemias [7]. The precipitous destruction of neoplastic cells leads to an uncontrolled release of intracellular contents, causing hyperuricemia, hyperkalemia, hyperphosphatemia, hypocalcemia, and acute renal failure [7].

In our case, we hypothesize that acute tumor lysis syndrome occurred after proximal splenic artery embolization with consequent infarction of splenic tissue with significant neoplastic invasion. Based on the Cairo–Bishop definition of tumor lysis syndrome after chemotherapy [7], only hyperkalemia (7.8 mEq/L) was met within 3 days before to 7 days after proximal splenic artery embolization. There was a >25% increase of baseline inorganic phosphorous (1.5-3.8 mg/dL), but this was after supplemental phosphate infusions. Acute renal failure (BUN: Cr < 20:1) was noted 2 days after the procedure; however, Cr (1.58 mg/dL) was less than the Cairo–-Bishop parameter. Contrast-induced renal injury remains a probable cause. Nonetheless, the parameter for tumor lysis syndrome was fulfilled 13 days after the procedure: hyperuricemia (15.7 mg/dL), hyperphosphatemia (4.9 mg/dL), and increased Cr (2.65 mg/dL). The delayed onset of tumor lysis syndrome may be due to a slower progression of tumor lysis syndrome after splenic artery embolization versus tumor lysis syndrome after chemotherapy.

It is also possible that proximal splenic artery embolization precipitated a spontaneous form of tumor lysis syndrome. Spontaneous tumor lysis syndrome, defined as tumor lysis syndrome in the absence of cytotoxic therapy, is exceedingly rare but occurs most commonly in hematologic malignancies [8,9]. The low incidence of splenic infarction associated with proximal splenic artery embolization, and the absence of splenic infarction on surgical pathology support this claim. However, hyperphosphatemia is less common in spontaneous splenic artery embolization, as is hypocalcemia [9]. The phosphate released in spontaneous tumor lysis syndrome is reused by dividing neoplastic cells rather than binding with extracellular calcium [9].

Only one previous case of tumor lysis syndrome after splenic artery embolization is known. Leibowitz et al [10] report a case of tumor lysis syndrome in a patient with chronic lymphocytic leukemia about 3 hours after splenic artery embolization, presenting as hyperkalemia with cardiac arrhythmia during laparoscopic splenectomy. Similar to our case, the patient had massive splenomegaly with malignant involvement. However, proximal embolization was performed in our case.

In summary, our case represents the second reported incident of acute tumor lysis syndrome after splenic artery embolization. Tumor lysis syndrome should be especially considered after proximal or distal splenic artery embolization in patients with advanced hematologic malignancies. Potassium, phosphorous, calcium, and uric acid should be monitored closely. Any abnormalities should be managed promptly, and fluids and allopurinol should be given liberally to avoid renal injury. At last, same day splenectomy and embolization should be considered to decrease the likelihood of developing tumor lysis syndrome.

REFERENCES

[1] Poulin E, Thibault C, Mamazza J, Girotti M, Côté G, Renaud A. Laparoscopic splenectomy: clinical experience and the role of preoperative splenic artery embolization. Surg Laparosc Endosc 1993;3:445–50.
[2] Hiatt JR, Gomes AS, Machleder HI. Massive splenomegaly. Superior results with a combined endovascular and operative approach. Arch Surg 1990;125:1363–7.
[3] Ekeh AP, Khalaf S, Ilyas S, Kauffman S, Walusimbi M, McCarthy MC. Complications arising from splenic artery embolization: a review of an 11-year experience. Am J Surg 2013;205:250–4.
[4] Schnüriger B, Inaba K, Konstantinidis A, Lustenberger T, Chan LS, Demetriades D. Outcomes of proximal versus distal splenic artery embolization after trauma: a systematic review and meta-analysis. J Trauma 2011;70:252–60.
[5] Goldstone J. Splenectomy for massive splenomegaly. Am J Surg 1978 Mar;135(3):385–8.
[6] Prassopoulos P, Daskalogiannaki M, Raissaki M, Hatjidakis A, Gourtsoyiannis N. Determination of normal splenic volume on computed tomography in relation to age, gender and body habitus. Eur Radiol 1997;7:246–8.
[7] Cairo MS, Bishop M. Tumour lysis syndrome: new therapeutic strategies and classification. Br J Haematol 2004;127:3–11.
[8] Kekre N, Djordjevic B, Touchie C. Spontaneous tumor lysis syndrome. Can Med Assoc J 2012;184(8):913–6.
[9] Weeks AC, Kimple ME. Spontaneous tumor lysis syndrome: a case report and critical evaluation of current diagnostic criteria and optimal treatment regimens. J Investig Med High Impact Case Rep 2015;30(3).
[10] Leibowitz AB, Adamsky C, Gabrilove J, Labow DM. Intraoperative acute tumor lysis syndrome during laparoscopic splenectomy preceded by splenic artery embolization. Surg Laparosc Endosc Percutan Tech 2007;17:210–1.