# Spontaneous tumor lysis syndrome in a patient with significant metastatic tumor burden from hepatocellular carcinoma

Malashanka MD, Sofya; Shenoy MD, Ananth; Liskey MD, David; Finch MD, Kevin; Siddique MD, Asma
Virginia Mason Medical Center

## Introduction

Tumor lysis syndrome (TLS) is an oncologic emergency due to the lysis of cells typically associated with hematologic malignancies and active chemotherapy[1].

The Cairo-Bishop criteria for TLS includes hyperkalemia, hypocalcemia, hypophosphatemia, and hyperuricemia in the setting of high cellular proliferation and turn over[2, 3].

Prevention as well as timely recognition of TLS is critical due to the cardiotoxic effects of the associated electrolyte imbalances.

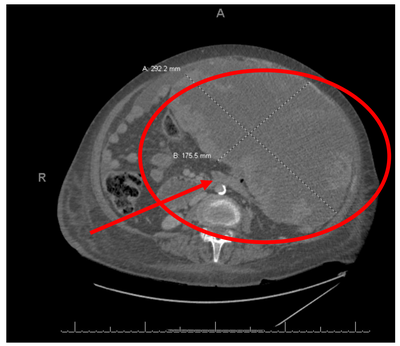


Figure 1a (left): Metastasis, axial view. 29.2 cm by 17.5 cm on the left side of the abdomen, displacing nearby structures.

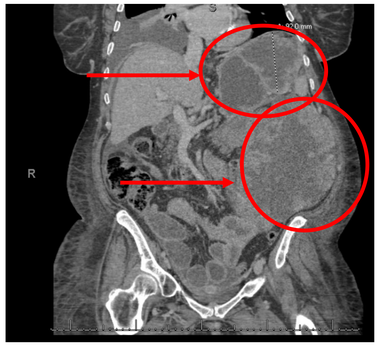


Figure 1b (right): coronal view of metastases. There is a necrotic metastasis in the left upper quadrant and another in the left lower quadrant compressing nearby structures.

## Case Description

A 61-year-old female with a history of Hepatis C cirrhosis, Hepatocellular Carcinoma (HCC, stage pT2 pNX) with abdominal metastases was found to be profoundly anemic prior to port placement for chemotherapy. On admission her hemoglobin was down to 5.2 from 8.4 three weeks prior.

Other laboratory abnormalities included mild hyperkalemia (5.0-5.5), and acute kidney injury (AKI) with creatinine 1.17-1.34 from 0.67. AKI was suspected to be pre-renal in the setting of blood loss. Hyperkalemia was attributed to spironolactone as well as AKI. Work up of anemia included upper endoscopy and CT angiogram that demonstrated significant interval increase of known intra abdominal metastases with diffuse hemorrhage leading to the development of hemoperitoneum. It was determined that the metastases were not amenable to embolization (Figure 1a, 1b).

The patient was offered palliative radiation but was unable to tolerate the sessions. In addition, the patient developed a small bowel obstruction, which was attributed to compression from metastases.

Over the course of hospitalization, potassium level continued to increase, and this was attributed to worsening AKI. Calcium as well as phosphate remained within normal limits. This patient's potassium level climbed to a critical high, at which point a uric acid level was checked and found to be elevated to 15.2 (Figure 2). There was high suspicion of spontaneous TLS, due to large burden of metastatic disease from HCC. Rasbiuricase was administered, with uric acid level decreasing to 5.0 (Figure 2).

Patient elected to proceed with comfort-based treatment and passed away, likely in the setting of fatal cardiac arrythmia.

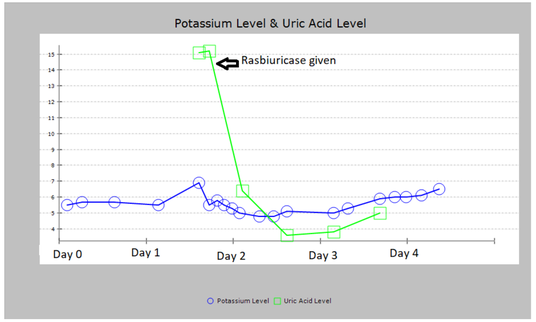


Figure 2: Potassium and uric acid levels. Potassium levels elevated from admission, uric acid level of 15.2 with subsequent drop after Rasbiuricase administration.

## Discussion

Spontaneous TLS has been documented with multiple types of solid malignancies, and rarely in those with metastatic hepatocellular carcinoma[4,5]. Another take away from this case is to be aware of anchoring bias. This rare condition can present with worsening renal function and electrolyte abnormalities. Moderate hyperkalemia and AKI in a patient with known metastatic disease and large tumor burden should prompt consideration of tumor lysis syndrome, which may include monitoring uric acid level as well as initiating preventative therapy.

## References

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## Contact

Sofya Malashanka, MD
Internal Medicine PGY-2
Virginia Mason Franciscan Health
Email: sonya.Malashanka@CommonSpirit.org