Invasion of the Kidneys

MUHC Medical Grand Rounds
Alex Khoury
PGY-2 Internal Medicine
MGH
Case Presentation

• A 33 year old man from Guinea presented in Oct 2016 to the MGH ER with malaise, vomiting, and diffuse abdominal pain.

• PMHx /PSHx: None
• Meds: None
• Habits: None
• Allergies: NKDA
Physical Exam

• On presentation, he was hemodynamically stable.

• Physical exam revealed mild confusion, cachexia & signs of volume depletion.

• Remainder of the clinical exam was unremarkable and no oliguria.
Laboratory Investigations

• Hemoglobin: 109, normal WBC and PLT
• Serum Na: 99, serum osmolality: 262 mosm/L.
• Creatinine: 1240, proteinuria: >10 g/L on spot U/A, serum albumin: 15 g/L and glycosuria with normal serum glucose.
• PO4 of 3.7, ionized calcium: 0.8, uric acid normal
• Urine Na: 51, urine osmolality: 221 mosm/L.
• LDH: 2223
• TSH, lactate, cortisol, LFTs, lipase/amylase and HIV negative
Further Investigations

- CXR: bilateral peri-hilar nodular opacities
- CT head and lumbar puncture were negative
- CT abdomen (no contrast)
Non-Infused CT Abdomen

- Enlarged kidneys and possible thickening of the descending colon.
- R kidney: 14.2 cm and L kidney: 14.8 cm.
- Normal liver, pancreas, no LAD and no obstruction.
Dialysis was initiated.

Given that he was not oliguric, it was deemed that he would likely not improve with volume management alone.
DDx bilateral nephromegaly

- Diabetes (most common)
- Amyloidosis
- Polycystic kidneys
- Bilateral Hydronephrosis
- Malignancy
Renal Biopsy

- Extra nodal NK/T-cell lymphoma nasal type with renal involvement and concomitant focal segmental glomerulosclerosis.
Renal Biopsy

• The renal biopsy revealed FSGS (collapsing type) and widespread acute tubular injury.

• A large area of the interstitial compartment was infiltrated by atypical medium to large lymphoid cells, with the immuno-histochemical profile of an extra-nodal NK-/T cell lymphoma, nasal type (Next Figure)
Light photomicrographs

- A: H & E stain -> prominent atypical lymphoid cells between tubular epithl cells.
- B: CD3+
- C: EBV+
- D: Glomerulus stained with Masson trichrome stain showing FSGS.
Further Investigations

- Normal autoimmune screen, negative TB screening, CMV titers, Hep B and Hep C.

- Epstein Barr Virus PCR revealed $7.62 \log_{10} \text{copies/mL}$. 
Evolution

• Subsequently developed a massive LGIB which required urgent operative resection of a fistulous tract between the jejunum and descending colon.

• Pathology → lymphomatous involvement.

• Continued to bleed despite maximal support and died in the ICU day 20.
Conclusion

• Our patient had a **rare** and **aggressive EBV-associated non-Hodgkin lymphoma** with involvement of the kidneys (by Carbone et. al)

• Collapsing focal segmental glomerulosclerosis which may be EBV-associated as described by Chandra et. al.
Conclusion

• Cause of his severe hyponatremia was multifactorial.
  Renal failure with nephrotic syndrome and contributory ADH release mediated by baroreceptor-stimulation (hypovolemia), his symptoms (nausea, vomiting and pain) and his lymphoma.

• Confused secondary to severe hyponatremia and renal failure.
References


Submitted to Kidney International

- **Authors:**
  - Catherine Courteau, Alex Al-Khoury MD, Rene P. Michel MD, FRCP(C), Catherine L. Weber MD, MHSc., FRCP(C)