IgA MONOCLONAL GAMMOPATHY: AN UNCOMMON CAUSE OF HIGH ANION GAP METABOLIC ACIDOSIS. A CASE REPORT

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Background: Metabolic acidosis is frequently encountered in emergency settings. It may recognize many different causes. Here, we report an uncommon case of severe high anion gap (AG) metabolic acidosis associated with a monoclonal gammopathy of renal significance (MGRS) due to monotypic IgA deposits.

Case Presentation: A 58-year-old woman was admitted to the emergency room for acute dyspnea.
ABG showed extremely severe metabolic acidosis with hyperkalemia and elevated anion gap: pH 6.9, pCO2 9, Bic 1.9, pO2 111, K+ 6.2, Cl 109, Ca++ 1.21, Lac 1.8, AG 32.
Lab exams also showed severe acute kidney injury (creatinine 20.4 mg/dL vs previous value 2.5 mg/dL) and severe anemia (Hb 6.4 mg/dL). She had no history of diabetes, hypertension, or other illness. The patient denied diarrhea or drugs intake. Because of rapidly worsening dyspnea, mechanical ventilation was started. In the meanwhile, CVVHD treatment was initiated and continued for 4 days, when it became possible to withdraw dialysis. Then, the patient was transferred to the nephrology ward.
Laboratory examinations showed increased IgA circulating levels (5 g/L). A renal biopsy resulted in the diagnosis of monotypic immunoglobulin IgA lambda deposition disease (bone marrow biopsy was negative for myeloma).
Then, therapy with Dexamethasone and Bortezomib was started with the progressive recovery of the renal function (two months after creatinine was 2.5 mg/dL, eGFR 20 mL/min). The patient is still in follow-up; the renal function is stable and ABG normal.

Conclusion: High anion gap acidosis is potentially associated with IgA gammopathy. Indeed, it has been proved that IgA paraproteins (in particular IgA lambda chains), having an isoelectric point slightly below physiologic pH, may act as anions in the serum. Monoclonal gammopathies should be included in the diagnostic workup of unexplained metabolic acidosis.