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Nephrogenic Diabetes Insipidus induced by Liposomal Amphotericin B: a case report

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BACKGROUND:

- Nephrogenic diabetes insipidus (NDI) results from the inability of the late distal tubules and collecting ducts to respond to vasopressin.
- The lack of ability to concentrate urine results in polyuria and polydipsia.
- NDI is almost always drug induced; however, there are other causes of it that are acquired, such as electrolyte abnormalities as hypokalemia or hypercalcemia.

OBJECTIVE:

- To describe a case of nephrogenic diabetes insipidus (NDI) associated with the long-term use and high-dose liposomal amphotericin B.

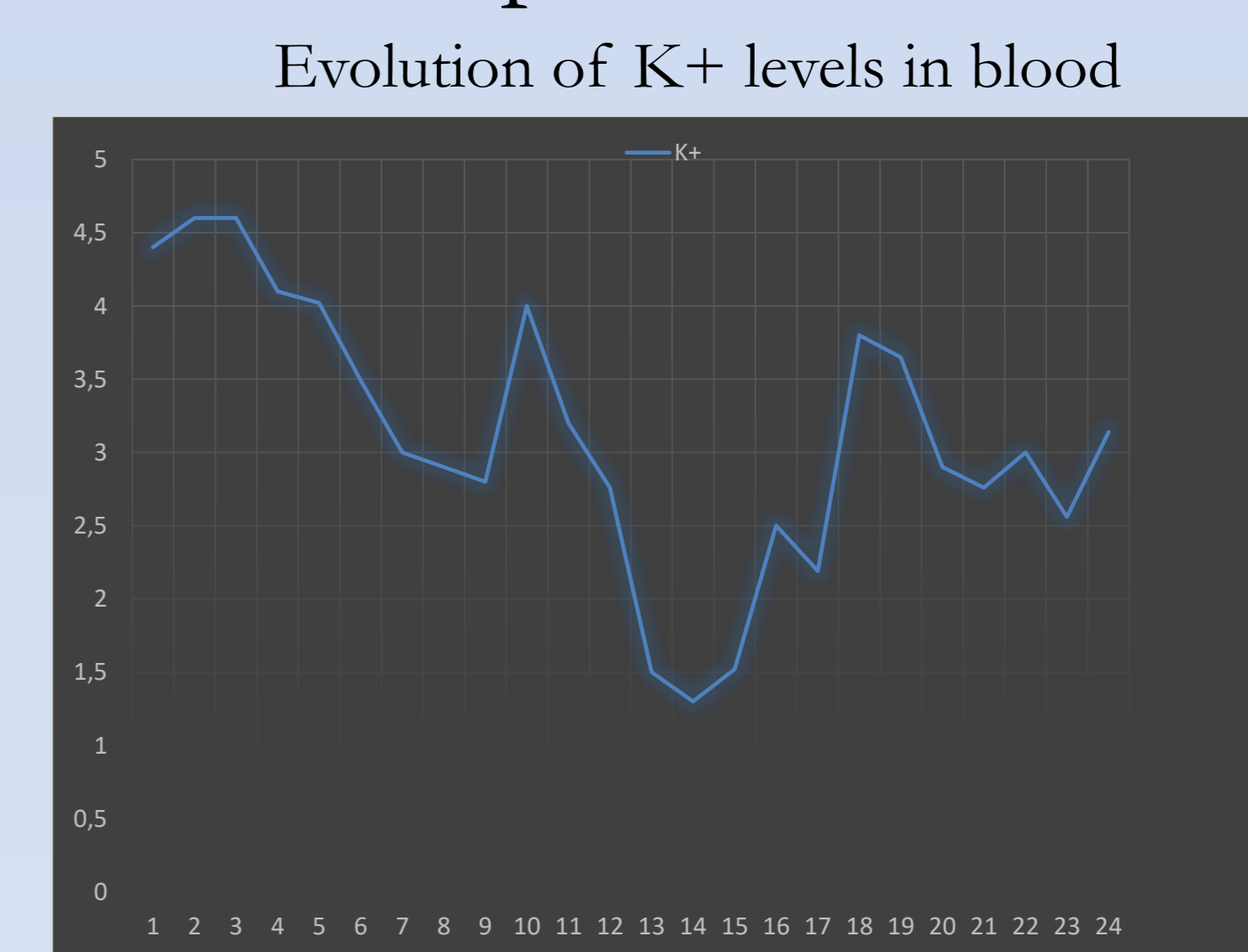
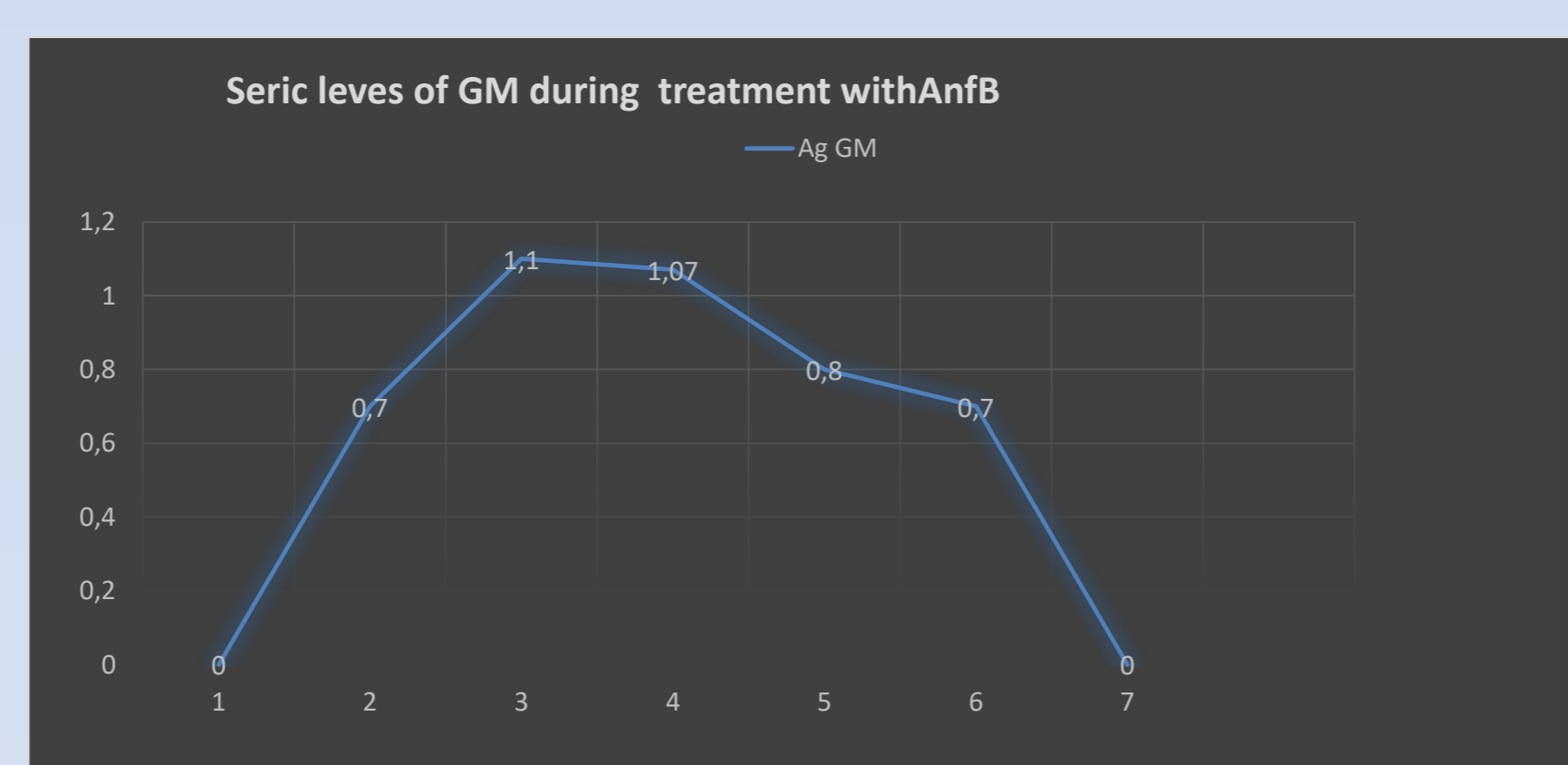
METHODS:

- Data were obtained from electronic medical records



RESULTS:

A 39 years old man diagnosed with diffuse large B-cell non-Hodgkin Lymphoma underwent an allogeneic bone marrow transplant. After a few months, the disease's progression was detected and immunosuppressive treatment was suspended (February 2014). Rescue treatment was: Gemcitabine and Vinorelbine. In May, the patient was admitted to the hospital with graft-versus-host disease grade III, intestinal form. He was treated with: cyclosporine, micophenolate, sirolimus, methylprednisolone, oral and rectal beclametasone. Additionally, as antimicrobial prophylaxis were meropenem, acyclovir, levofloxacin, cotrimoxazole and caspofungine. In June, serum galactomannan antigen test was positive and invasive pulmonary aspergillosis was diagnosed. *Aspergillus fumigatus* and *Aspergillus flavus* were isolated. The patient was started on liposomal amphotericin B 6mg/kg (440 mg) daily during 41 days (accumulated dosage: 18.04g). Voriconazol was rejected because of concomitant treatment with sirolimus. During the treatment, his serum potassium was decreased $<1.5\text{mEq/L}$ and it was difficult to maintain $>3\text{ mEq/L}$ during the treatment with significant polyuria (urine output was $>6\text{litr/day}$). Nephrogenic diabetes insipidus(NDI) was diagnosed. The patient was started on desmopressin 10mcg/12h nasal drops plus hydrochlorothiazide 50mg/24h and spironolactone 50mg/24h. Finally, Amphotericin treatment was withdrawn, and serum potassium level returned to normal. According to the Naranjo algorithm, this would be classified as a possible reaction because of the temporal correlation between NDI after long term and high dose liposomal amphotericin. Several cases were reported related to NDI induced by Amphotericin B1–3, regardless of formulation.



CONCLUSIONS

It is very important to understand the etiology and symptoms associated with nephrotoxicity and NDI. Despite of the use of liposomal amphotericin B, NDI was developed. The association of others nephrotoxic drugs and persistent hypokalemia also contributed to this event. Specific intervention is required to prevent the nephrotoxicity in patients receiving Amphotericin B.

References:

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