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Acute Kidney Failure Complicating Administration of Immunoglobulins During Guillain Barré Treatment: Case Report and Literature Review

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Abstract

Drug induced nephrotoxicity is a common problem, and the incidence of drug related kidney failure may be as high as 20% of etiologies of acute kidney failure. Therapy with intravenous immunoglobulin (IGIV) was introduced to replace natural deficiencies of immunoglobulin. The indication for such treatment have broadened to include a wide number of clinical disorders. Anuria, oliguria, associated with kidney failure appeared to occur more commonly than any other disorders. The extend and complexity of the installation is dose-depending.

Acute kidney failure (AKF) usually occur within 2 days after the initiation of IGIV and may persist for 15 days. Although risk factor like preexisting kidney impairment, diabetes, vascular injury, may predispose to AKF, yet sucrose and saccharose are the main caises of anuria associated an acute kidney failure.

Keywords: *Nephrotoxicity, Intravenous immunoglobulin, Acute kidney failure.*

Introduction

Acute Renal Failure (ARF) is defined by a sudden, sustained and reversible reduction in glomerular filtration occurring within hours to days.

It results in the retention of metabolic waste products and disruption of hydroelectrolytic and acid-base balance. Acute drug-induced renal failure accounts for 20% of the etiologies of AKI. Its incidence is increasing due to the emergence of new potentially nephrotoxic molecules and the multiplication of drug prescriptions. However, this incidence is most certainly underestimated given the fact that symptoms are often silent [1].

Medicated ARFs are classically considered to have a better prognosis than ARFs of other origins. This notion must be qualified in the hospitalized patient. Drug-induced ARF, especially if it occurs with other visceral defects, is associated with excess mortality with a 5.5-fold increase in the risk of death in some studies [1,2].

It is therefore essential to recognize them and establish a causal relationship between the drug and the adverse event. In clinical practice, the Naranjo Probability Scale and the World Health Organization (WHO) rating system are the most widely accepted and widely used methods for assessing causality [3].

We report in this publication a case of GUILLAIN barré complicated by acute anuric renal failure in post immunoglobulin therapy.

Case report

Mister, J, B, 30 years old, without any particular pathological antecedent, hospitalized in medical intensive care at the CHU IBN ROCHD of Casablanca for suspicion of Guillain barré.

The history of his disease goes back to one week before his admission by the appearance of a muscular weakness initially felt at the level of the lower limbs and complicated by a motor deficit with a type of paresis of ascending

evolution going up to the upper limbs, without sensory deficit, without sphincter disorders with notion of ineffective cough and swallowing disorders with normal thoracic amplification without other associated dysautonomic disorders. All of this evolves in a context of apyrexia without any notion of infectious episode in the weeks preceding the symptomatology.

The physical examination finds a patient with apyrexia, weight 70 kg;

NEUROLOGICAL EXAMINATION finds a patient conscious 15/15 on the Glasgow scale, pupils symmetrical and reactive, Muscular test 3/5 with

- Upper extremity : Proximal : 1/5 Distal: 2/5
- Inf > Proximal : 0/5 Distal: 1/5

Respiratory: patient presents a respiratory discomfort with ineffective cough, saturation at 94%, FR: at 16 cycles per minute, no audible rales at the two pulmonary fields.

On the hemodynamic AND CIRCULATORY level: BP: 12/07cmHg (MAP: 65mmHg) CF: 100bpm, Diuresis preserved: 1.2 L/24h, absence of oedema of the lower limbs.

Pulmonary pleuropneumon auscultation: absence of rales in both pulmonary fields. The patient had initially benefited from a cerebral CT scan, and a frontal chest X-ray with normal results. Biologically normal on admission:

*A lumbar puncture was performed on 03 /09/2019, showing albuno-cytological dissociation with : Proteinorachy: 0.69g/l , Glycorachy : 0.6 g/l , Leucorachy : < 3 element /mm3
A lhémogramme : Hb :16,4g/dl Plq :185000
Its objective ionogram: Creatinine: 76.1 umol/l, Urea: 7.52umol/l, Na+: 125 meq/L K+: 4.5 meq/L, ALAT/ ASAT: 395/1985 UI ,Proteins/albumin: 51/28 g/L, Cl- :93mmol/l.

➢ Haemostasis assessment: TP 105% ,TCA:25.2 sec
➢ INFECTIOUS BALANCE: CRP: 104mg/l, Procalcitonin: 2.05 ng/l, GB: 9750/mm3, ECBU: hematuria 25600 GR/mm3 germ-free
Therapeutically, the patient was initially put on :
➢ PPI: 40mg/d, benfotiamine 100mg, LMWH 0.4 IU/d
➢ Immunoglobulin Human (Tegeline) IV 1g/kg /day for 2 days, for a total dose of 140g.

The evolution was marked by the appearance at D4 of hospitalization (at D2 after tegeline administration) of a total anuria with worsening of renal function Urea: 24.9 mmol/l ,Na+: 119 meq/L ,K+: 5.5 meq/L.

Creatinine: 721umol/l with a DFG A ZERO ,ALAT/ ASAT : 357/837 (24 times the normal one) ,Proteins / albumin: 67/25 g/l ,Cl- : 90mmol/l Lipasemia : 66 IU/L, CRP : 160 mg/L vs 104 at PCT admission : 0.5 µg/l , with Hb : 12,1g/dl ,Plq : 113000 , GB: 4420/mm3.

The vesico-renal ultrasound showed: absence of obstacle on the excretory tract with kidneys of normal size, regular contours, fairly well differentiated, without dilatation of the excretory cavities.

The therapeutic approach was to perform a saline serum filling test with 2L of crystalloid. In case of persistent anuria and high kalemia at 6.5 mmol/l, a hemodialysis session was indicated (duration=1h30, UF=1kg500).

The evolution was favorable after 7 hemodialysis sessions with a creatinine level of 124.7umol/ a diuresis preserved.

Discussion

The occurrence of acute renal failure following immunoglobulin (IGIV) administration has been frequently reported since 1987 [4,5].

There are approximately 30 cases of renal failure attributed to immunoglobulin available in the literature. The onset and regression time ranges from 4 to 15 days and is frequently oligoanuric renal failure (74%) requiring one or more hemodialysis sessions. Three-quarters of patients have pre-existing renal insufficiency, even moderate, and one out of two have diabetes [5].

Other studies have shown that acute renal failure due to IVIG occurs within 2.6 ± 1.8 days after the start of treatment. It is most often oligoanuric or anuric, of variable intensity, and requires the use of extra-renal purification in less than half of cases (<50%). In the vast majority of cases, the time to return to basal creatinine is less than 15 days. The mechanism involved is debated; the role of osmotic nephrosis due to the sugars contained in certain IVIG preparations, notably Sandoglobulins, is currently being studied [7, 8-9].

Our patient presented, 2 days after a treatment with IGIV (tegeline) based on 2g/kg in 2 days for the disease of guillain barré, an acute anuric renal insufficiency, regressive after 7 sessions of hemodialysis, our patient also presented a risk factor predisposing to the speed of installation of the symptomatology namely a pre-existing renal insufficiency.

Excessive infusion rate may be an additional risk factor, [6] increased oncotic pressure in the glomerular capillary and vasoconstriction are factors observed during nephrotoxicity.

Immunoglobulins are responsible for tubular damage mainly obstructive osmotic nephrosis or secondarily acute interstitial nephritis of allergic origin [3].

Carbohydrate excipients and in particular sucrose contained in immunoglobulin preparations are the main factors promoting this nephrotoxicity. The development of ARF is due to intratubular precipitation of immunoglobulins, or to the deposition of immune complexes that can lead to glomerulonephritis [11].

In our case the immunoglobulin used is based on 100MG/ML sucrose. After the penetration of sucrose into the tubular cell, there is a swelling of the proximal tubular cells leading to a tubular obstruction. The renal cells do not possess disaccharidases, the enzyme necessary for the digestion of sucrose, so that its accumulation leads to a call of water inside the cell by oncotic effect, explaining the increase in cell volume and the obstruction of the tubular lumen, hence the anuria observed in the cases reported in the literature and in our case.

The risk factors that expose to the occurrence of ARF: extracellular dehydration, hypovolemia, heart failure, pre-existing renal failure, age over 60 years, high dosage of nephrotoxic product, coexistence of diabetes and vascular damage. [10]
Our patient has two risk factors: pre-existing renal insufficiency, IGIV infusion rate, and a high risk of death.

The risk factors related to the molecule:The type of preparation / The dose administered > normal dose: 2g/kg : (1g/kg/d x 2d or 400 mg/kg/d x 5d) / The infusion rate: 1ml/kg/hr but not exceeding 4ml/kg/hr. The first injection must be particularly slow.

Therefore, it is recommended that the presence of these risk factors, including the creatinine level, be systematically evaluated in patients at risk.

Conclusion

Nephthotoxicity of drugs remains a daily concern in medical practice. The prescription of a potentially nephrotoxic agent should take into account the report of benefit/risk, The nephthotoxicity of polyvalent immunoglobulins seems to come from two mechanisms. The first is induced by the sudden increase in oncotic pressure after infusion of high doses of immunoglobulin. The second would come from a direct tubular toxicity of sucrose, This likely dose-dependent mechanism would allow the design of more rational prevention and treatment strategies to reduce the incidence and severity of nephthotoxicity.

The degressivity of the renal symptomatology suggests the mechanism of tubular cell regeneration, the observed polyuria must be compensated by sufficient intake to avoid dehydration.

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