NDT Plus (2008) 1: 32–33
doi: 10.1093/ndtplus/sfm028
Advance Access publication 24 December 2007

NDT PLUS
Nephrology Dialysis Transplantation

Case Report

A case of extreme azotaemia

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Keywords: azotaemia; BUN; renal failure

Introduction

Despite intensive therapy, the mortality rate in acute renal failure still exceeds 30% [1]. Advanced age, multiple organ failure, oliguria and high blood urea nitrogen (BUN) at the time of diagnosis are associated with a poorer prognosis [2]. Rapid diagnosis and prompt treatment are critical to prevent death and irreversible renal failure. Renal biopsy remains the golden standard for diagnosis. Glucocorticoids and cyclophosphamide are the cornerstones in the treatment of rapidly progressive glomerulonephritis; they dramatically ameliorate glomerular injury and induce remission in 75–80% of cases [3].

We present a patient with remarkable biochemical derangement, especially high BUN (Table 1). A case like this has not previously been seen in our department, nor described in the literature.

Case

A 42-year-old unemployed male was admitted to a local hospital with severe fatigue and oliguria. He lived with, and was supported by his father. When he was younger, he started university studies, but stopped after only 1 year. At admission, he had been in bed for a month and could barely walk to the lavatory. Earlier in the year, he had a tendency to crural oedema; at hospitalization he was severely dehydrated, but was well circulated. Blood samples revealed extreme azotaemia, hyperkalaemia, metabolic acidosis and anaemia (Table 1). No other organ failure was found. Ultrasonography showed normal size kidneys without hydronephrosis. He was subsequently transmitted to our department, where he received haemodialysis, blood transfusion, erythropoetin therapy, diuretics and antihypertensive therapy. Initially he received haemodialysis twice a day for 1 h, but after the first dialysis he developed generalized convulsions. Further blood analysis showed the presence of circulating antineutrophil cytoplasmic antibodies (ANCA) with specificity for proteinase-3. Anti-glomerular basement membrane antibodies were not detected. Renal biopsy revealed focal, segmental necrotizing glomerulonephritis with crescent formation.

Immunofluorescence showed absence of immunoglobulin. Treatment with glucocorticoids (75 mg prednisolone) and cyclophosphamide (150 mg) was started. After 1 month the patient was discharged. At this time he was on haemodialysis three times a week and had a normal diuresis (Table 1). Six months later, peritoneal dialysis was initiated, and a new renal biopsy revealed irreversible glomeruli destruction. The immunosuppressive treatment was discontinued.

Discussion

The patient presented with acute renal failure caused by rapidly progressive glomerulonephritis. He had two out of the four described risks factors for poor outcome [2], namely high BUN and oliguria.

BUN is a waste product of protein metabolism. It is synthesized in the liver and is excreted in the urine by glomerular filtration. Acute renal failure thereby results in progressive elevation of BUN. Other reasons for elevated BUN are gastrointestinal bleeding, acute ileus and dehydration. The high BUN in gastrointestinal bleeding and ileus is caused by increased fluid loss, decreased glomerulus filtration because of low blood pressure and increased protein metabolism from blood or transsudate in the intestine. In this case, the azotaemia was partly caused by renal failure and to a lesser extent by dehydration.

The highest level of BUN we were able to find in the literature was 93 mmol/l [4], which makes this case, with a value of 163.9 mmol/l, particulary interesting. A high elevation of BUN correlates with a poor prognosis and rapid dialysis is life saving. However, a slow drop in BUN is desirable because of a protracted drop in cerebrospinal fluid.

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[3]Contribution: Conception. Revising the article. Approval of the version to be published.

Table 1. Key laboratory parameters of the patient

	Day 1 (pre-dialysis)	Day 2 (post-dialysis)	Day 7	Day 13	Day 26 (discharge)	After 6 months
S-creatinine (μmol/l)	2785	1638	758	692	453	334
S-urea (mmol/l)	163.9	111.0	30.3	20.6	21.6	33.7
B-Hgb (mmol/l)	3.7	3.3	6.0	5.7	5.5	8.6
P-K (mmol/l)	7.0	3.5	3.2	3.6	4.7	4.2
HCO_3^- (mmol/l)	11.9	17.4	21.8	26.1	22.5	23.0
Clearance (ml/min)			1	3	9	22
Diuresis (ml/24 h)			300	1100	1850	3430

S: serum, B: blood, Hgb: haemoglobin, P: plasma, K: potassium, HCO_3^-: hydrogen carbonate.

This results in a relative elevation of osmolality in the cerebrospinal fluid. Water then crosses the cell membrane to achieve osmotic equilibrium, which leads to an elevated pressure in the cerebrospinal fluid and sometimes convulsions. This occurred in our patient (Figure 1). This risk should be weighed against the benefit of fast reduction in hyperkalaemia and acidosis.

Another important reason for such a high BUN, in this case, was delayed contact with the health care system. Why did the patient not consult earlier? He is unemployed, lives with his father, does not go out and has no circle of friends. He has a normal intelligence, but dropped out of university. We think that his obvious social isolation and perhaps mental illness is the reason for this failure to consult.

In conclusion, in cases of acute renal failure, prompt diagnosis and immediate treatment are essential to prevent development of severe azotaemia, which is known to worsen the prognosis and eventually leads to a fatal outcome.

Conflict of interest statement. Marija Kristina Novosel and Kjeld Erik Otte are doing research for Shire with lanthanum and are on Shire's paylist. Anne Daugaard Thuesen and Mohamad El-Faramawi have no conflict of interest. The results presented in this paper have not been published previously in whole or part.

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Received for publication: 17.11.07
Accepted in revised form: 20.11.07