NDT Plus (2008) 5: 357–358
doi: 10.1093/ndtplus/sfn099
Advance Access publication 15 July 2008

NDT PLUS
Nephrology Dialysis Transplantation

Nephroquiz
(Section Editor: M. G. Zeier)

Acute abdomen and acute kidney injury: a common entity, a not so rare link?

Sofia Jorge[1], José António Lopes[1], Patrícia Lages[2], Hugo Corte-Real[3], Henrique Bento[3], Carlos França[3] and Mateus Martins Prata[1]

[1]Department of Nephrology and Renal Transplantation, [2]Department of Surgery and [3]Department of Intensive Medicine, Hospital de Santa Maria, Lisboa, Portugal

Case

A 45-year-old Caucasian male with a 16-year history of HIV-1 infection and a 5-year history of ileocecal non-Hodgkin lymphoma was admitted to the emergency room with fever, and abdominal pain and distension. In the previous month, he noticed abdominal pain. A CT scan and colonoscopy were performed and confirmed tumour relapse involving the distal ileum, caecum and right colon.

At admission, the patient was dehydrated, hypotensive, tachycardic, tachypneic and febrile. He presented ascites and a petrous mass in the right lower abdominal quadrants. Oliguria was documented. Laboratory tests (Table 1) revealed anaemia, elevation of acute-phase reagents, renal dysfunction, hypernatraemia, metabolic acidosis and hypoxaemia. Elevation of creatine kinase and lactic dehydrogenase, and hyperphosphataemia, hypocalcaemia, hyperuricaemia and hyperkalaemia were also diagnosed. Abdominal plain did not reveal air–fluid levels, and abdominal CT scan showed ascites and a voluminous mass in the ileon, caecum and right colon. A paracentesis was performed and an exsudate with high cellularity (28.160/mm^3) with numerous blasts was drained. Fluid resuscitation was started and vasoactive support was needed. The patient required ventilatory support and was admitted to the Department of Intensive Medicine. Empirical antibiotherapy (meropenem) and rasburicase (0.2 mg/kg, single dose) were administered. There was refractory oliguria, and continuous venovenous haemodiafiltration was started. The abdomen remained distended, haemoglobin decreased by 2 g/dL and 2 units of erythrocyte concentrate were administered. Cultures were negative. Despite the central venous pressure of 8–12 mmHg, mean arterial pressure of 65 mmHg, stabilization of haemoglobin (8.6 g/dL) and undetectable uricaemia had been achieved, the patient kept oliguric.

Table 1. Laboratory tests at admission

Parameter	Value	Normal range
Haemoglobin (g/dL)	8.8	13–17
Leucocytes (cells/mm^3)	18 000	4000–10 000
Polymorphonuclear cells (%)	92	40–70
C-reactive protein (mg/dL)	23	<0.5
Uraemia (mg/dL)	220	<45
Serum creatinine (mg/dL)	3.6	< 1
Potassium (mEq/L)	6.5	3.5–5.5
Sodium (mEq/L)	148	135–145
Chloride (mEq/L)	101	95–110
pH	7.1	7.35–7.45
Bicarbonates (mEq/L)	15	22–26
$PaCO_2$ (mmHg)	21	35–45
PaO_2 (mmHg)	55	60–100
Oxygen saturation (%)	82	>90
Lactates (mEq/L)	62	<18
Creatine kinase (UI/L)	1214	<145
Lactic dehydrogenase (UI/L)	851	200–450
Phosphates (mg/dL)	7.6	2.5–4.5
Ionized calcium (mEq/L)	0.7	1.12–1.32
Serum uric acid (mg/dL)	26	<7
PT (s)	10/11	
TTPa (s)	27/29	
Alanine aminotransferase (UI/L)	25	<35
Aspartate aminotransferase (UI/L)	20	<35

Question

What is your diagnosis?

Correspondence and offprint requests to: José António Lopes, Department of Nephrology and Renal Transplantation, Hospital de Santa Maria, 1649-035, Lisboa, Portugal. Tel: +351-961203912; Fax: +351-217805679; E-mail: jalopes93@hotmail.com

© The Author [2008]. Published by Oxford University Press on behalf of ERA-EDTA. All rights reserved.
For Permissions, please e-mail: journals.permissions@oxfordjournals.org

Answer to the quiz

The intra-abdominal pressure (IAP) was measured and it was elevated (86 mmHg). A catheter was placed intraperitoneally and 1600 cc of haematic liquid was drained. The IAP decreased to normal values (<20 mmHg), the patient recovered from diuresis, renal replacement therapy was suspended and there was complete renal function recovery (serum creatinine 0.8 mg/dL) within 4 days.

Discussion

In the present case, we consider that multiple factors contributed to acute kidney injury (AKI) development (dehydration, bleeding, septicaemia and tumour lysis syndrome). However, the elevation of the IAP and the temporal relationship between abdominal decompression and renal function recovery lead us to also consider that abdominal compartment syndrome (ACS) could play an important role in the pathogenesis of the AKI. ACS was most probably caused by the voluminous tumour mass and intra-peritoneal bleeding by the tumour, possibly aggravated by massive fluid resuscitation.

ACS is defined as an IAP >20 mmHg accompanied by organ failure, such as respiratory, haemodynamic and renal failure [1]. Intra-abdominal hypertension causing compression over inferior vena cava, and thus, decreasing venous return and cardiac output, is considered the main pathophysiological mechanism of AKI in the ACS [2]. Placement of a catheter via the urinary bladder to measure IAP is considered the 'gold standard' [1]. After the diagnosis of ACS, abdominal decompression achieved through laparotomy or intra-peritoneal catheter placement usually reverses oliguria [3].

In conclusion, we highlight that ACS should be considered as a potential aetiology of AKI in patients with acute abdomen, and its diagnosis and treatment should be established as soon as possible, allowing renal function recovery.

Conflict of interest statement. None declared.

References

1. Malbrain ML, Cheatham ML, Kirkpatrick A *et al*. Results from the international conference of experts on intra-abdominal hypertension and abdominal compartment syndrome: I. Definitions. *Intensive Care Med* 2006; 32: 1722–1732
2. Barroso S, Hernandez R, Ruyz B *et al*. Deterioro de la funcion renal asociado a aumento de la presion abdominal. *Nefrologia* 2007; 1: 85–86
3. An G, West MA. Abdominal compartment syndrome: a concise clinical review. *Crit Care Med* 2008; 36: 1304–1310

Received for publication: 1.5.08
Accepted in revised form: 18.6.08