Case Report

Intra-abdominal hypertension is an under-appreciated cause of acute renal failure

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Keywords: abdominal compartment syndrome; acute renal failure; intra-abdominal hypertension; intra-abdominal pressure; oliguria

Introduction

Increased intra-abdominal pressure (IAP), known as intra-abdominal hypertension (IAH) is increasingly being recognized as an important predictor of adverse outcomes in critically ill patients. The prevalence of IAH has recently been estimated at between 32 and 65% in both medical and surgical intensive care units [1,2]. High grade IAH may result in abdominal compartment syndrome (ACS), where increased pressure in a closed anatomic space threatens the viability of surrounding tissues and organs [1,2]. Renal dysfunction is one of the earliest and most common presentations in ACS. As IAP increases, glomerular filtration rate (GFR) decreases progressively and anuria may ensue [3,4]. Cases of acute renal failure (ARF) quickly reversed by abdominal decompression (DC) have been well-documented in the surgical literature [3], but surprisingly, only a handful of cases were reported in the nephrology literature [4]. Even standard nephrology textbooks such as Brenner & Rector’s do not list ACS or IAH as a cause of ARF [5]. ACS is usually not considered by nephrologists as a likely cause of ARF as there are other more classic causes such as acute tubular necrosis or volume depletion, especially in non-surgical settings. We report three cases of acute oliguric renal failure which met the definition of ACS, and two cases in which the diagnosis of ACS was very likely based on the clinical course. We then review the mechanisms of renal failure in ACS, and propose some clinical criteria that could aid clinicians suspect and pursue the diagnosis of this potentially reversible cause of ARF.

Patients and methods

This case series was approved by the Institutional Review Board of the Oregon Health & Science University. The standardized technique of measurement of IAP, grading of IAH and definition of ACS were consistent with the guidelines published by the World Society of Abdominal Compartment Syndrome (WSACS: http://www.wsacs.org) [1,2]. IAP was measured by transduction of urinary bladder pressure at the bedside. With the patient in the supine position, 50–100 ml of sterile saline was injected via the aspiration port of a Foley catheter into the urinary bladder, while the drainage tube was clamped. A pressure transducer or a water manometer zeroed at the level of the symphysis pubis was then connected to the aspiration port to measure the bladder pressure. This method is easy to perform, highly accurate in estimating the IAP and has been validated in several studies [6,7].

IAH grading was performed according to the WSACS criteria [1,2]; grade I: IAP between 12 and 15 mmHg; grade II: 16–20 mmHg; grade III: 21–25 mmHg; grade IV: >25 mmHg.

ACS was defined as an IAP ≥20 mmHg and the presence of single or multiple organ failure.

Among the patients with ARF for whom our nephrology service was consulted between 2001 and 2003, we found five patients with suspected ARF due to IAH: three patients who met the definition of ACS and two patients in whom ACS was highly suspected as a cause of ARF. Only one case is described in detail, and we summarize the salient features of the other cases in Table 1.

Case series

Case 1

A 56-year-old Caucasian male with a history of end-stage liver disease and ascites secondary to primary biliary cirrhosis underwent successful liver transplantation. However, on the first post-operative day,
he made only 30 cc of urine in 12 h despite the administration of 4.9 l of intravenous (IV) fluids. At the time of the nephrology consult, the patient was mechanically ventilated and his blood pressure was 160/60 mmHg, heart rate 79/min, temperature 37.8°C and respiration rate 14/min. There was no S3 gallop and the lungs were clear to auscultation. The abdomen was markedly distended and no bowel sounds were heard. Urinary bladder pressure was 23.5 mmHg. Since serum creatinine had risen from a baseline of 2.0 to 4.0 mg/dl despite IV fluid administration ACS was suspected. Exploratory laparotomy revealed a bleeding diaphragmatic vessel. The vessel was ligated and a substantial intra-abdominal partially thrombosed haematoma was evacuated. Shortly thereafter, while still in the operating room, the patient began to make urine. Bladder pressure post-operatively fell to 14.7 mmHg and eventually normalized. He continued to make approximately 100 ml/h of urine and his serum creatinine fell to 2.2 mg/dl over the next 48 h.

Cases 2–5 are summarized in Table 1.

### Discussion

In this report, we present three cases of confirmed ACS (patients 1–3) and two highly suspected cases of ACS (patients 4 and 5). Unfortunately, IAP was not measured in patients 4 and 5, but their clinical course essentially ruled out other causes of renal failure and made ACS the most likely diagnosis. They had no evidence of intrinsic renal disease or urinary tract obstruction, had signs of IAH and did not respond to pressors, IV fluids (IVFs) or diuretics. They both had a brisk diuresis immediately following abdominal DC, that is unlikely to occur in cases of volume depletion or intrinsic renal disease. Table 1 summarizes the clinical features of all five patients before and after abdominal DC.

The causes of primary ACS include intra-abdominal haemorrhage or visceral oedema following trauma or intra-abdominal operations such as liver transplantation, as in patient 1 [1,2]. Massive ascites, as in patients 2, 4 and 5 and rectus sheath haematoma, as in patient 3, have also been reported [8,9]. Intra-abdominal space-occupying lesions, pancreatitis, complicated pregnancy and delivery are other causes of ACS. Interestingly, excessive IV fluid administration and large volume resuscitation alone have also been identified as causes for ACS (secondary ACS), even in the absence of abdominal trauma or surgery [10,11]. The mechanism of increased IAP with IV fluid administration may be related to visceral oedema and third spacing [10]. Patients 1, 3 and 5 in our series were administered a large volume of IVFs prior to their developing ACS and probably had a combination of primary and secondary ACS (Table 1).
Abdominal compartment syndrome as a cause of oliguric acute renal failure

ACS is associated with a high mortality rate, ranging from 42 to 100% if not promptly corrected, probably as a result of haemodynamic instability and multi-system failure. High IAP raises systemic vascular resistance and inferior vena cava pressure, decreases venous return and cardiac output, causes respiratory failure, increases intracranial pressure, reduces abdominal perfusion and results in renal failure and ischaemic bowel necrosis [1,2].

The normal IAP is around 5 mmHg [1,2]. Although not extensively studied, detrusor or urinary bladder dysfunction is unlikely to affect the accuracy or the interpretation of IAP measurement [12]. Values >12 mmHg are considered elevated by WSACS criteria [1,2]. Sugrue et al. [13,14] found that the odds ratio of developing renal failure was 12.4 if the IAP is >20 mmHg, and that the incidence of renal impairment was 33% with an IAP of 18 mmHg or higher compared with only 14% for an IAP <18 mmHg. Bradley and Bradley [15] were one of the first investigators to study the relationship between external abdominal compression and renal function in 17 normal human subjects. They observed a significant decrease in GFR and renal plasma flow during compression and complete reversal by DC. They ruled out ureteral obstruction as a cause of oliguria.

The pathophysiology of acute renal failure in ACS has been partly elucidated by animal experiments. Harman et al. [16] showed that artificially increasing the IAP in a canine model led to a significant decrease in renal blood flow and GFR. This reduction was reversed only minimally by fluid resuscitation but almost completely by abdominal DC. Kirsch et al. [17] studied the effects of IAH by examining a rat pneumoperitoneum model. They observed oliguria accompanied by a significant reduction of inferior vena cava (IVC) flow compared with aortic flow and suspected central venous compression as a cause of renal dysfunction. Doty et al. [18,19] showed in a swine model, that artificially raising renal vein pressure, but not renal parenchymal compression caused progressive reduction in GFR. Plasma renin, aldosterone and antidiuretic hormone levels are elevated in ACS, resulting in sodium and water retention, reduced cardiac output, increased urine osmolality [3]. These data suggest that renal hyperfiltration is the main cause of renal failure in ACS. Increased renal vein pressure and possibly IVC pressure due to increased IAP are likely the main causes of diminished renal perfusion in ACS. In addition, diminished cardiac output as a result of cardiac and respiratory haemodynamics is a contributing factor. Whether renal vasoconstriction due to elevated levels of angiotensin II plays an additional role is unclear.

Clinical clues to the diagnosis of ARF due to ACS include acute oliguric renal failure in the setting of severe abdominal distension especially after abdominal surgery or trauma, in patients with known ascites or after excessive IV fluid administration [11]. We observed that the absence of pulmonary oedema and/or signs of congestive heart failure such as an S3 gallop despite an elevated central venous pressure (CVP), and an elevated diaphragm on chest radiographs are other clues (Table 1). The elevated CVP is spurious and reflects increased pleural pressure caused by an elevated diaphragm and IAH rather than increased intravascular blood volume. Measurement of the urinary bladder pressure as a surrogate of IAP is the gold standard in diagnosing ACS and should be performed whenever ACS is suspected. Although not previously tested in randomized trials, abdominal DC if feasible and if performed early enough may reverse renal dysfunction. Delayed DC, however, can result in multi-organ failure and death (patient 3).

In conclusion, we believe that ACS or even more subtle degrees of IAP elevation are under-appreciated causes of acute oliguria, and should be added to the list of causes of acute renal failure in nephrology textbooks. Renal dysfunction in ACS appears to be related to diminished renal perfusion, partly due to the raised renal vein pressure and partly to the low cardiac output and high renal vascular resistance. A high index of suspicion is essential for the diagnosis, which may circumvent the need for dialysis and may prevent irreversible renal damage in a patient who needs DC.

Conflict of interest statement. None declared.

References


Received for publication: 13.7.06
Accepted in revised form: 24.7.06