

Acute Kidney Injury Caused by Abdominal Compartment Syndrome in a Morbidly Obese Patient Resolved with Emergent Enterotomy

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Abstract

A 61-year-old morbidly obese male (184 kg, BMI 61.7) with history of heart failure with preserved ejection fraction coronary artery disease and hypertension developed fluid overload culminating in respiratory failure requiring mechanical ventilation. Difficulty in achieving adequate oxygenation led to aggressive diuresis, followed by hypotension and anuria. Physical exam revealed jugular venous distention, distant rales, a massively distended abdomen, and +3 pitting edema. Creatinine increased from 1.2 to 1.9 mg/dl. Urine sediment was unremarkable for intrinsic causes of acute kidney injury (AKI). Bladder pressure monitoring revealed increased intra-abdominal pressure. Emergent exploratory laparotomy with decompression of small bowel ileus resulted in instantaneous improvement in endorgan dysfunction. Within 48 hours postoperatively AKI resolved. Creatinine normalized with spontaneous diuresis of 6100 cc's. This case demonstrates the dire complication of abdominal compartment syndrome with AKI as an early sign. Initially AKI was contributed to overdiuresis and heart failure. Especially in morbidly obese patients ACS can easily be missed due to body habitus. Therefore awareness of the condition and early bladder pressure monitoring in the context of bland urine results are key to diagnosis. Enterotomy is a heroic option but restored renal function in this case.

Keywords

Acute kidney injury, Enterotomy, Morbidly obese male, Abdominal Compartment Syndrome

Introduction

Abdominal compartment syndrome (ACS) is a common condition in critically ill patients. Studies in medical and surgical intensive care units (ICUs) have shown a prevalence of intraabdominal hypertension (IAH) and ACS of up to 64 and 12%, respectively (5). IAH was significantly associated with

more severe organ failure, particularly renal and respiratory failure, and a prolonged ICU stay (1) and higher mortality (2). Intraabdominal pressure (IAP) in critically ill adults ranges from 4 to 7 mmHg. IAH is defined as sustained or repeated elevation of IAP ≥ 12 mmHg. Sustained elevation of IAP of >20 mmHg associated with new organ dysfunction defines ACS (3). AKI is defined as increase in serum creatinine 2-3 times from baseline or decrease in urine output < 0.5 mL/kg/h for more than 12 hours. It can be an early sign of increasing IAP. IAP has been reported to be increased in obese patients, but not in the IAH range (4). In morbidly obese patients diagnostic modalities are limited and AKI might mistakenly be attributed to other comorbidities. Bladder pressure monitoring is an easy and non-invasive method, if a foley catheter is already in place, to estimate IAP (5). In our case AKI was the first symptom of ACS in a morbidly obese patient with very limited diagnostic modalities and multiple comorbidities.

Case Report

A 61-year-old 184 kg heavy male (BMI 61.7) with a history of diabetes mellitus, heart failure with preserved ejection fraction (HFpEF), coronary artery disease and hypertension was treated in the medical intensive care unit for hypoxic respiratory failure requiring mechanical ventilation. The patient had stopped his diuretic voluntarily, which had resulted in massive fluid overload. With aggressive diuresis he became hypotensive with mean arterial pressure in the range of 60 mmHg. One day later anuria occurred. At this point he was also very difficult to oxygenate and required airway pressures above 35 cmH₂O. Physical exam revealed jugular venous distention, distant rales, a massively distended abdomen, and anasarca with +3 pitting edema. Laboratory data showed increased creatinine from 1.2 to 1.9 mg/dl, BUN of 65 mg/dl and rising WBC to 14/nL. Arterial blood gas on 85% FiO₂ showed a pH of 7.38, PCO₂ of 60, and PO₂ of 86 mmHg. Urine sediment was unconvincing for intrinsic causes of AKI (Image 1) and showed crystals, non dysmorphic red blood cells and some granular casts. Bronchoscopy revealed only markedly edematous airway mucosa. Central venous pressure was 16 cmH₂O. X-ray studies were limited due to body habitus but showed clear lungs and dilated small bowel loops. Intra-abdominal pressure was 30 mmHg estimated by bladder pressure. At this time surgery was consulted and the decision was made to perform exploratory laparotomy. Immediately after opening the abdomen, the anesthesiologist reported improved ventilation. Diffuse small bowel ileus with very distended bowels with paper thin wall was found. Via an enterotomy the bowel was decompressed. As there was no other inflammatory or infectious process found, the abdominal cavity was temporarily closed with a vacuum dressing (Image 2). Intraoperative trans-esophageal echocardiography showed a left ventricular ejection fraction of 55%. Within the first 48 hours after surgery AKI resolved. Creatinine

Dropped to 0.9 mg/dl and the patient diuresed 6100 cc's bright yellow urine spontaneously (table 1). His hemodynamics stabilized and airway pressures normalized with improved oxygenation. Unfortunately the patient went into acute respiratory distress syndrome during the following days, was unable to be oxygenated despite all efforts and passed away.

Discussion

This case demonstrates the fatal complication of ACS. AKI with anuria was an early symptom. However, initially AKI was contributed to overdiuresis versus underperfusion from acute HFpEF exacerbation. Few data exist about diagnosing and treating ACS in the morbidly obese population. Physical exam in patients with normal body habitus has been shown to be unreliable for diagnosing IAH/ACS with a sensitivity of 40 to 60.9% (6). In our morbidly obese patient it was inconclusive, if not misleading. Diagnostic tools were very limited, as chest Xray and surface echocardiogram provided limited images. Invasive hemodynamic monitoring indicated low cardiac output and supported the thesis of acutely exacerbated heart failure. However, elevated bladder pressure indicated ACS per definition (3). Nevertheless, it is unclear, if bladder pressure monitoring is reliable in morbidly obese patients. Lambert et al showed in a small study with 45 morbidly obese patients (mean BMI 55 +/- 2 kg/m²), that the mean IAP was 12 +/- 0.8 cmH₂O, IAP correlated to the sagittal abdominal diameter, as an index of central obesity, but not to body weight, or BMI. Therefore they concluded that elevation of IAP in morbidly obese patients is not a true ACS but represents a direct mass effect of the visceral obesity (7). Our patient had dilated small bowel loops on Xray and was taken for exploratory laparotomy. Due to the fact that endorgan dysfunction improved immediately after abdominal decompression, we assume that ACS was present. Cheatham et al showed in a study with 478 patients that the use of a continually revised AIH/ACS management algorithm, which included serial intra-abdominal pressure measurements, nonoperative pressure-reducing interventions, and early abdominal decompression, significantly increased patient survival from 50% to 72%, decreased resource utilization and increased same-admission primary fascial closure from 59% to 81% (8). Furthermore they identified prophylactic use of an open abdomen as independent predictor of survival. If this applies to morbidly obese patients as well is not clear. A study with 10 critically ill patients and IAH showed that bolus administration of cisatracurium can be used to temporarily reduce IAP, however no significant effect on urine output could be observed (9). Our patient was temporarily treated with a neuromuscular blocker intraoperatively but we doubt that this influenced recovery. Mohmand et al state in their review that in fully established ACS, decompressive laparotomy remains the treatment of choice and that early, rather than late, decompression is gaining more

popularity and is associated with better outcomes (8,10). But again, minimal data exist in the morbidly obese population.

Especially in morbidly obese patients ACS can be missed on physical exam, x-ray studies and invasive monitoring due to body habitus. Many questions remain unanswered and serious scholarly effort in this high risk population is warranted. Awareness of the condition and early bladder pressure monitoring is a key to diagnosis. Enterotomy is a heroic therapeutic option but restored renal function in this case.

Table

	pre-op	post-op	12 hrs post-op	24 hrs post-op	36 hrs post-op	48 hrs post-op
Creatinine	1.9 mg/dl	1.8 mg/dl	1.4 mg/dl	1.1 mg/dl	1.0 mg/dl	0.9 mg/dl
BUN	64 mg/dl	70 mg/dl	65 mg/dl	49 mg/dl	40 mg/dl	30 mg/dl
total urine output	0 cc	140 cc	2645 cc	4050 cc	5010 cc	6100 cc

Table 1: Pre- and posoperative urine output and laboratory values

Figures

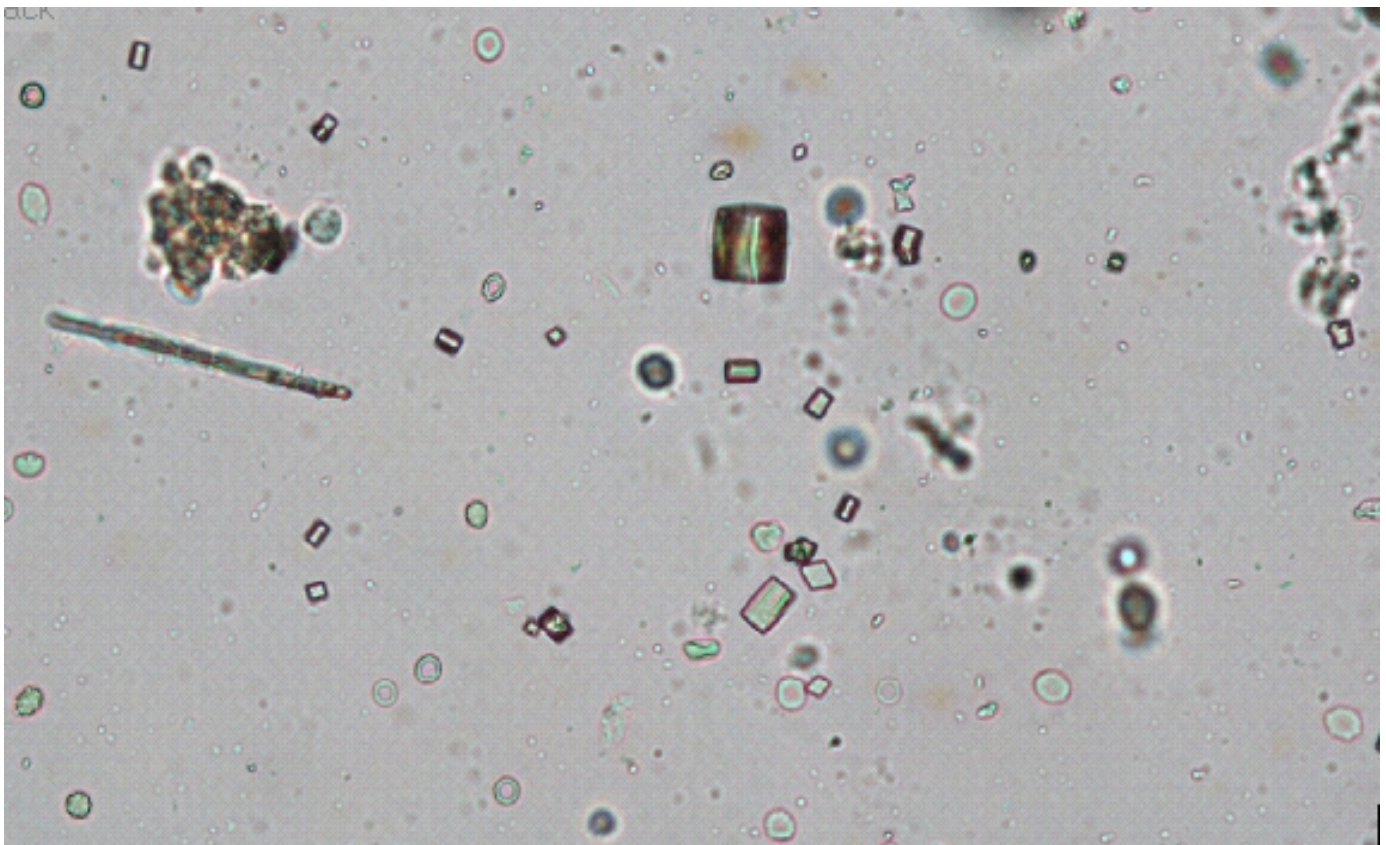


Figure 1: Urinalysis



Figure 2: Open abdomen with vacuum dressing

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