

CASE REPORT

Secondary Abdominal Compartment Syndrome after Military Wounding

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Abstract

Intra-abdominal hypertension and abdominal compartment syndrome are increasingly recognised as causes of serious morbidity and mortality in critically injured patients, particularly those with significant burns. Identification of at risk patients, routine monitoring of intra-abdominal pressures and appropriate, early treatment may reduce the incidence and complication rate of abdominal compartment syndrome and so improve outcomes in critically injured personnel. We present the case of an American Marine injured in an explosion while on patrol in Afghanistan, who despite the absence of significant intra-abdominal injury, went on to develop abdominal compartment syndrome and required decompressive laparotomy

Introduction

Intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) are increasingly recognised as causes of serious morbidity and mortality in critically injured patients, particularly those with significant burns. Identification of at risk patients, routine monitoring of intra-abdominal pressures and appropriate, early treatment may reduce the incidence and complication rate of ACS and so improve outcomes in critically injured personnel. Abdominal compartment syndrome may result from injury within or without the abdomen [1] and results in a series of well recognised physiological consequences that contribute to the associated morbidity and mortality [2]. Treatment is usually surgical in the form of decompression and laparostomy.

We report a soldier, who developed ACS without any obvious primary intra-abdominal injury following polytrauma including burns whilst in Afghanistan. We believe that a large systemic inflammatory response syndrome (SIRs) response combined with aggressive fluid resuscitation was partly to blame for the ACS.

Case Report

A US soldier, injured by an Improvised Explosive Device (IED) blast whilst in the gun turret of an armoured vehicle on patrol was admitted to the Emergency Department (ED) of the UK Role 2E hospital at Camp Bastion, Afghanistan 105 minutes after injury. He was fully conscious but tachycardic (127bpm), tachypnoeic (24 breaths / minute) with a blood pressure of 166/65mmHg. He had burns of variable thickness to the face, neck, chest, both arms, both thighs and the abdomen amounting to 33% total body surface area, an open intra-articular fracture of the proximal right tibia with limb compartment syndrome and a distended abdomen. Pre-hospital treatment was not known; high flow oxygen, trauma series x-rays and a right thigh tourniquet were instigated in the ED, as well as urinary catheterisation, although intra-vesical pressures were not recorded. A Focused Abdominal Scan for Trauma (FAST) revealed no signs of significant intra-abdominal injury. Laboratory results are not available, but haemoglobin concentration remained

above 8g/dl without transfusion. He received 14mg morphine, 1.2g of benzyl penicillin and 4l crystalloid fluid intravenously and underwent rapid sequence induction of anaesthesia before his transfer to theatre within 30 minutes of his arrival. He underwent right lower limb fasciotomies and wound debridement.

Intra-operatively both his abdominal distension and cardiovascular status worsened despite ongoing resuscitation, suggesting intra-abdominal pathology. Trauma laparotomy was performed and found minimal ascites and oedematous but viable bowel. His abdominal wall burns were superficial. All cardiovascular parameters improved on opening the abdomen suggesting a diagnosis of secondary abdominal compartment syndrome. The abdominal wall was left open with a Temporary Abdominal Closure (TAC) (Figure 1). He was evacuated by American critical care assets the following day.

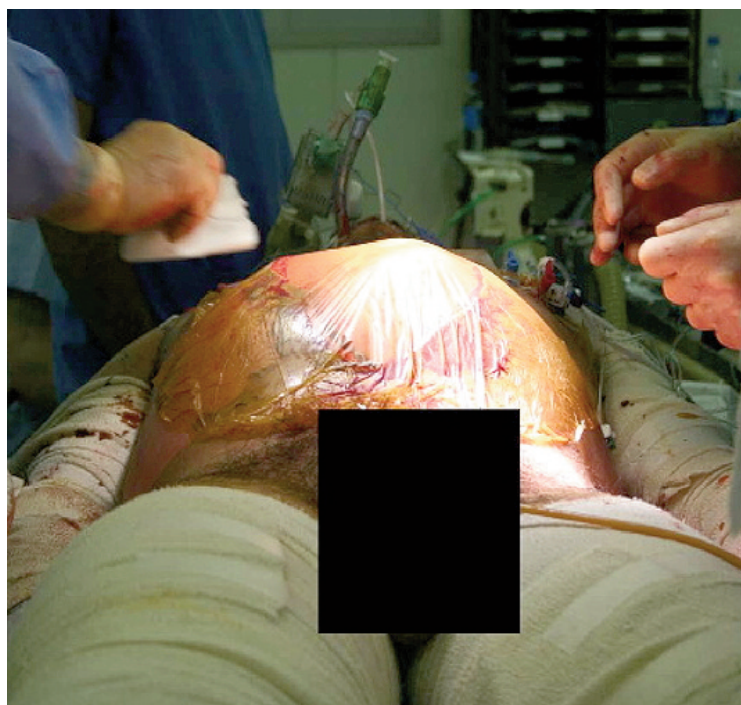


Figure 1. Temporary abdominal closure using a combination of Bogata bag and Opsite sheet

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Discussion

Abdominal compartment syndrome is the end point of a disease spectrum of intra-abdominal hypertension. IAH is defined as a pathological, sustained rise in intra-abdominal pressure >12mmHg. ACS is IAH sufficient to cause organ dysfunction. It is defined as sustained or repeated IAP >20mmHg in the presence of new single or multiple organ system failure [1]. Normal intra-abdominal pressure is 2-7mmHg. IAH is graded from I-IV depending on severity [1] (Table 1).

Grade of IAH	Intra-abdominal Pressure (mmHg)
I	12-15
II	16-20
III	21-25
IV	>25

Table 1: Grading of IAH

Once the intra-abdominal pressure reaches 10mmHg, viscera start to become compromised and if pressure rises to more than 20mmHg capillary blood flow to the viscera is severely limited. ACS itself is not graded as it is an all or nothing phenomenon, but is classified into primary, secondary or recurrent ACS (Table 2).

Primary ACS	Initial pathology is within the abdomen, pelvis or retroperitoneum.
Secondary ACS	Initial pathology is extra-abdominal.
Recurrent ACS	Redevelops after successful medical or surgical treatment of primary or secondary ACS

Table 2. Classification of ACS

The risk factors for the development of ACS are outlined in Table 3 and severely injured military casualties are at risk for several of them. Relevant to our case, burns patients are at particularly high risk of developing ACS, due to the marked SIRS response and subsequent large fluid resuscitation requirements [4].

Acidosis	pH <7.2
Hypothermia	Core temp <33°C
Massive transfusion or fluid resuscitation	>10units packed red cells or 5l colloid/crystalloid within 24hrs
Coagulopathy	APTTR >2 or INR >1.5
Haemo or pneumoperitoneum	
Sepsis or bacteraemia	Sepsis defined as SIRS + identifiable source of infection
Liver dysfunction with ascites	
Mechanical ventilation	Particularly with PEEP or auto PEEP
Pneumonia	
Abdominal surgery	Particularly with tense closure
Gastroparesis/ileus/gastric distension	

Table 3. Independent Risk Factors For Development of ACS[1,3]

Using standard burns resuscitation formulae [4], this soldier could expect to have received up to 5 litres of intravenous crystalloid in the first six hours after wounding, for his burn wound alone, as well as other fluid and blood products as required to treat associated injuries. In addition, the high risk of coagulopathy (often as a direct result of massive transfusion) and hypothermia also places such patients at high risk of IAH and ACS, even without direct abdominal injuries. One prospective study has shown a 70% incidence of IAH in patients with major burns [5].

The best treatment of ACS is prevention and is not necessarily surgical. Medical and anaesthetic measures to reduce the volume of the intra-abdominal contents (colloid rather than crystalloid resuscitation to decrease gut oedema, nasogastric drainage or bowel purgation) or increase abdominal wall compliance (optimal analgesia and sedation including complete neuromuscular blockade if necessary) can ameliorate the consequences of IAH. Continuous haemofiltration has been shown to reduce the circulating cytokine load in patients with significant SIRS and reduce both intra-abdominal hypertension and mortality [1,3].

ACS unresponsive to medical therapy produces critical ischaemia of the abdominal contents and needs to be managed as a surgical emergency [6]. The treatment is surgical decompression and management of the open abdomen. With the exception of very high intra-abdominal pressures, there are no established guidelines stating exactly when surgical intervention is indicated. However, some studies have shown improved outcomes with early decompression [7]. Temporary abdominal closure following decompression may be achieved by a variety of techniques of which the Bogata bag and Opsite™ sandwich, with or without vacuum drains are the commonest [8]. Patients who have undergone surgical decompression can still develop ACS, so IAP should still be monitored, but those that continue to deteriorate post-decompression have a poor outcome [9].

This soldier suffered multiple injuries including significant burns. He was managed according to BATLS protocols and rapidly received four litres of intravenous crystalloid. He developed a considerable SIRS response and secondary ACS. As no intra-abdominal pressure monitoring was available, this diagnosis was made retrospectively on the basis of the clinical findings of increasing abdominal distension and worsening cardiovascular status that consequently normalised with decompression. Intra-abdominal pressure monitoring is not routinely performed until arrival at Role 4 facilities, but this case highlights the potential benefits of pressure monitoring closer to the point of wounding. Monitoring could be performed at Role 2E/3 facilities cheaply and easily, given that most seriously injured patients will already have a urinary catheter in situ. Early detection and treatment of ACS may reduce mortality and morbidity in this cohort.

Conclusions

Abdominal compartment syndrome causes significant morbidity and mortality. Early identification and timely surgery can reduce this. Currently, intra-abdominal pressures are not routinely measured until Role 4. Identification of at risk patients, a high index of suspicion and routine monitoring of intra-abdominal pressure nearer the point of wounding in susceptible patients may allow earlier detection and treatment of intra-abdominal hypertension. If ACS is suspected clinically, surgical intervention should be prompt and may be life saving.

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