A wolf in wolf's clothing — the abdominal compartment syndrome

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Four patients are described in whom massive abdominal distension after laparotomy led to increased airway and central venous pressure and severely reduced urine output. All cases were associated with massive fluid resuscitation and operative findings were a grossly oedematous bowel with free fluid under pressure in the abdomen. These findings are consistent with the diagnosis of intra-abdominal compartment syndrome. In 1 case trauma was remote from the abdomen indicating that abdominal surgery or trauma may not be a prerequisite for the development of the condition. Recognition of the features of the condition is essential as it can only be treated by decompression of the abdominal contents.

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Oliguria following raised intra-abdominal pressure has been described previously,[1,2] but the term intra-abdominal compartment syndrome appears to have been coined by Fietsam *et al.*[3] in 1989 when they described 4 cases of abdominal distension in patients undergoing repair of a ruptured aortic aneurysm. The intra-abdominal compartment syndrome usually occurs after emergency aortic surgery and direct abdominal trauma, but we describe 1 case where the condition followed elective aortic surgery and 1 case where the condition occurred following trauma remote from the abdomen.

Case reports

Case 1

A 70-year-old man underwent elective infrarenal aortic aneurysmectomy. The only complicating factor of note in the pre-operative phase was evidence of an old inferior myocardial infarct on electrocardiography. During the operation, which lasted 3½ hours, he required 5 litres of crystalloid fluid and 6 units of blood as replacement for blood lost. Urine output for the procedure was 2 litres. Otherwise the operation was uneventful and he was admitted to the intensive care unit (ICU). On admission,

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abdominal distension was noted and over the next few hours his central venous pressure (CVP) rose initially from 9 mmHg to 17 mmHg by 07h00 the following morning.

The following day cardiovascular variables were unstable. Systolic blood pressure was labile and hovered around 100 mmHg. CVP was 17 - 19 mmHg. Urine output had decreased to less than 50 ml/h. By this time inotropic support had been started with both dopamine and dobutamine. A total of 21 litres of fluid, mostly crystalloid but including 4 units of blood, had been administered as part of resuscitation. His abdomen was noted to be swollen and tense. Because his condition was deteriorating, he was taken back to the operating theatre at 19h00 (approximately 32 hours after aneurysmectomy) for a re-laparotomy. He was found to have a moderate retroperitoneal haematoma, mild ascites and an extremely oedematous bowel. The haematoma was evacuated and he was returned to the ICU with the abdomen open and mesh inserted to contain the bowel. Following decompression of the abdomen immediate improvement in his condition was noted (Fig. 1). Urine output improved dramatically, his blood pressure increased, his CVP dropped markedly and he was weaned off all inotropes within 36 hours. Peak airway pressure dropped by 10 mmHg. His postoperative course was stormy with repeated infections and an adult respiratory distress syndrome. He was in the ICU for 28 days, but was eventually discharged and is now well.

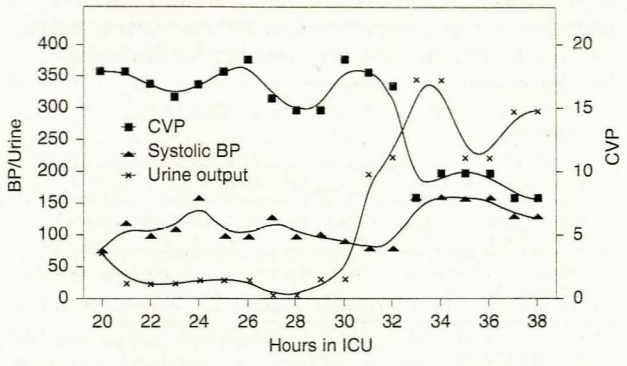

Fig. 1. Graph of CVP (cm H_2O), peak airway pressure (cm H_2O), systolic blood pressure (mmHg) and urine output (ml/kg/h) showing the fall in CVP and peak airway pressure together with a concomitant increase in urine output and systolic blood pressure following decompressive laparotomy 32 hours after aneurysmectomy.

Case 2

A young woman was shot accidentally while a gun was being cleaned. Injuries to the large bowel necessitated a hemicolectomy. Seven hours postoperatively she showed signs of a compartment syndrome with a tense abdomen, high peak airway pressure and high CVP, although urine output and fluid resuscitation were unremarkable. The pressure within the abdomen[2] was 39 mmHg. She underwent decompressive laparotomy but the clinical syndrome only resolved over several hours and she still required aggressive fluid resuscitation (Table I).

Case 3

A man was shot in the chest and abdomen, suffering injury to both the small and large intestines as well as the diaphragm. Eight hours after laparotomy his abdomen was noted to be extremely tense. Peak airway pressure on the ventilator was 51 cm H_2O, urine output was decreased and his requirement for resuscitation fluids was high. The pressure within his abdominal compartment was measured at 40 mmHg and he was taken to theatre for a decompressive laparotomy (Table I). The condition appeared to resolve, but he went on to develop an adult respiratory distress syndrome from which he died several days later.

Case 4

This patient was admitted after a motor vehicle collision wherein she sustained a severe crush injury of the lower leg which necessitated amputation. There was no indication of any abdominal pathology at the time. She was admitted to the ICU because she had required massive resuscitation for severe hypovolaemic shock. Initially she appeared to do well. She was breathing spontaneously on a 'T' piece. Blood pressure was 120/70 mmHg and urine output 2,5 ml/kg/h.

Seven hours after admission to the ICU she became restless. Her respiratory rate increased to 35/min and her $Paco_2$ decreased to 4,3 kPa. Although she remained well oxygenated her condition was obviously deteriorating and she was therefore ventilated. Peak airway pressure following intubation was noted to be 53 cm H_2O. She was also oliguric and had an increased requirement for fluids at this time. Her abdomen was tense and rigid and she was taken to theatre for laparotomy as it was thought that an abdominal injury had been missed. A simple paracentesis was undertaken by the registrar before she was taken to the operating theatre and he described the fluid within the abdomen as clear but under considerable pressure. Unfortunately the pressure was not measured.

At laparotomy she was found to have a large amount of clear fluid in the peritoneal cavity and a grossly oedematous bowel, findings which were present in all cases. There was no other injury. Once again, the fluid was under considerable pressure. After decompression, urine output increased, peak

Table I. Haemodynamic, airway and abdominal pressures and fluid balance in the 4 subjects*

Condition	CVP (cm H_2O)		Systolic blood pressure (mmHg)		Peak airway pressure (cm H_2O)		Fluid input (ml/kg/h)		Urine output (ml/kg/h)		Abdominal pressure (mmHg)
	Pre-op.	Postop.	Pre-op.	Postop.	Pre-op.	Postop.	Pre-op.	Postop.	Pre-op.	Postop.	
1. Aortic aneurysm	18	9	63	160	63	50	12,1	4,3	0,5	4,7	?
2. Gunshot abdomen	21	12	106	86	56	49	7,3	14,2	1,4	2,4	39
3. Gunshot abdomen	17	15	113	146	51	36	9,2	5,5	0,5	2,0	40
4. Crush injury	18	14	115	140	59	36	14,7	3,2	0,7	2,1	?

All values are averaged for 3 hours prior to decompressive laparotomy and the 3 hours following decompressive laparotomy.

airway pressure fell and her requirement for resuscitation fluids decreased dramatically (Table I). She recovered and was discharged from the ICU 6 days later.

Discussion

Compartment syndromes have most commonly been observed in the extremities after soft-tissue injury, vascular injury or fractures.[4] The pathophysiology within the tissue would appear to be related to increased intra- and extracellular fluid that causes increased pressure, decreased capillary perfusion and tissue hypoxia which in turn leads to more swelling and sets up a vicious circle which will eventually cause cell death and necrosis within the compartment.[5] While it usually follows direct trauma, a compartment syndrome may follow injuries to muscle from an ischaemia/reperfusion syndrome.[6]

Compartment syndromes have both local and systemic effects. Local effects involve damage to the organ therein, usually the muscle and the nerve in the case of a peripheral compartment. In the abdomen, internal organs such as kidneys, intestines[7] and the liver must be taken into account. Furthermore, the complex vascular responses caused by changes in afterload and preload also serve to confuse the picture.

The pathophysiology is not limited to the compartment and there would appear to be gross systemic effects as manifested in a 'leaky capillary syndrome', systemic hypoxaemia, massive fluid resuscitation and cardiovascular instability.[5] As a consequence of the intricate cardiovascular changes there is likely to be unrecognised hypovolaemia which will in turn cause problems.

Raised intra-abdominal pressure is well recognised as a cause of renal impairment.[1] While it was not measured in either patient 1 or patient 4, it would appear to have been a problem insofar as decompression of the abdomen in both cases resulted in immediate improvement. Should it be necessary to measure intra-abdominal pressure this may be accomplished through a nasogastric tube or via a urinary catheter.[2] Perhaps if this had been done in the above cases re-operation would have been carried out sooner. What is not so well recognised is the effect of the compartment syndrome on other organ systems which may, in turn, have systemic effects, and also the depressed cardiac output that occurs both as a direct cardiovascular[8] effect and secondarily due to hypovolaemia caused by loss of fluid into the interstitium. These patients require large amounts of fluid in an attempt to maintain cardiac and renal output. The large amounts of fluid required have been implicated as a cause of the syndrome. In virtually all cases the bowel has been found to be heavily oedematous; it is felt that this is due to the resuscitation and that this, in turn, is the root cause.[3] There is another possibility. The compartment syndrome, perhaps because of hypoxia, circulating mediators released from the white cell or bowel[9,10] or even reperfusion syndrome[11,12] following hypoxic damage to other tissues, affects Starling mechanics[12] in respect of the reflection and permeability coefficients; this takes place in such a manner that capillary permeability[13] becomes grossly abnormal and a leaky capillary syndrome results. This in turn causes hypovolaemia which, if not treated, will cause further hypoxia and a vicious circle that further compromises the microvasculature, increases the 'leakiness' and compromises the function of all tissues and organs. Whatever the reason, the increased requirement for fluids is symptomatic of this underlying problem and the effect of that problem on the body as a whole. In fact, the effects of pressure are extremely complex and probably comprise a whole series of abnormalities which finally manifest themselves as an abdominal compartment syndrome. In any event, the treatment is support and removal of the underlying cause, i.e. aggressive fluid resuscitation and abdominal decompression.

Compartment syndromes may range from mild to severe and may complicate any major abdominal procedure, although they tend to develop most often in those who have undergone major vascular operations or suffered abdominal trauma. The effects of the pressure on the bowel may be particularly important[10] and may well explain the condition's tendency to develop into a multiple organ failure syndrome or an adult respiratory distress syndrome through translocation of intestinal bacteria and endotoxins. Abdominal pressure can be measured fairly simply through a urinary catheter[2] and it would seem that measurement of intra-abdominal pressure should form part of the armamentarium of diagnosis. In particular, a high index of suspicion is required, especially in view of the possibility that an abdominal compartment syndrome may follow any form of severe trauma, even trauma that is remote from the abdomen. It is also likely, however, that some cases will continue to defy accurate diagnosis but will respond to decompressive laparotomy.

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