Unusual presentation of more common disease/injury

Paradoxical bradycardia in a patient with haemorrhagic shock secondary to blunt abdominal trauma

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Summary

A 47-year-old woman, with no previous medical problems, presented to the Accident and Emergency department with left shoulder pain following a fall onto her left side from a horse. Physical examination was unremarkable and she was discharged with simple analgesia. However, 2 h later, she re-presented with worsening left shoulder pain, dizziness and mild epigastric discomfort. A new examination revealed blood pressure of 100/60 mm Hg, which dropped to 95/65 mm Hg on standing, a Glasgow Coma Scale score of 15 and pulse of 62 beats/min. She was resuscitated with 2 litres of Hartmann's fluid. A focused assessment with sonography for trauma (FAST) scan of her abdomen was negative. Then, 1 h later she experienced generalised abdominal pain and developed postural hypotension. However she remained bradycardic (heart rate 45–60 beats/min). Repeat examination revealed peritonitis. A further FAST scan showed free fluid in the left hypochondrium. A CT scan demonstrated a complex tear of the spleen, for which she underwent an emergency total splenectomy. Her postoperative recovery was uncomplicated.

BACKGROUND

Hypovolaemic shock can result from absolute deficiency of blood volume. This is usually compensated for by an increase in heart rate (HR). Currently there is no case report in the literature of significant shock secondary to blunt abdominal trauma associated with bradycardia.

CASE PRESENTATION

A 47-year-old woman presented to our Accident and Emergency department with left shoulder pain following a fall onto her left side from a horse. She had no previous medical problems, did not take any regular medication and was a non-smoker. On examination her vital signs were as follows: blood pressure (BP) 103/68 mm Hg with no postural drop, HR 54 beats/min, respiratory rate (RR) 14 breaths/min and a Glasgow Coma Scale score of 15. Examination of her left shoulder and abdomen was unremarkable. She was discharged with simple analgesia (cocodamol).

However, 2 h later she re-presented to the department. She had worsening left shoulder pain, dizziness and mild epigastric discomfort. Re-examination was the same as previous, without any postural drop in BP. Arterial blood gas and haematological investigations were unremarkable except for leucocytosis (white cell count (WCC) 17×10^9).

She was resuscitated with 2 litres of Hartmann's fluid. A focused assessment with sonography for trauma (FAST) scan of her abdomen was negative.

Then, 1 h later she experienced generalised abdominal pain and developed postural hypotension with lying and standing BP of 96/65 mm Hg and 80/50 mm Hg, respectively. However she remained bradycardic (HR 45–60 beats/min). A repeat examination revealed epigastric and left hypochondrium tenderness. A repeat FAST scan

showed free fluid in the left hypochondrium. An urgent CT scan was arranged, which demonstrated a complex tear of the spleen with intraperitoneal fluid estimated to be approximately 5 litres in volume (Figure 1–4). She was subsequently taken to the operating theatre where these radiological findings were confirmed and she underwent emergency total splenectomy. Her postoperative recovery was uncomplicated and she was discharged from the hospital after 5 days.

INVESTIGATIONS

1. Blood tests: normal haemoglobin (Hb), urea, creatinine and liver function tests (LFTs). WCC 17.
2. First FAST scan: normal.
3. Second FAST scan: free fluid in left hypochondrium.
4. Plain x-ray of the left shoulder: No abnormality detected (NAD).
5. CT scan of abdomen and pelvis: complex tear of spleen with intraperitoneal fluid, estimated to be about 5 litres (Figure 1–4).

DIFFERENTIAL DIAGNOSIS

Second presentation differential:

1. Low grade hypovolaemia secondary to occult trauma.
2. Mild epigastric discomfort and dizziness secondary to analgesia (cocodamol).

TREATMENT

1. As per Advanced Trauma and Life Support guidelines, focusing particularly on serial clinical examinations, appropriate investigations and fluid resuscitation.
2. Emergency splenectomy for splenic rupture.

Figure 1 CT image showing complex splenic tear 1.

Figure 2 CT image showing complex splenic tear 2.

Figure 3 CT image showing complex splenic tear 3.

Figure 4 CT image showing complex splenic tear 4.

Table 1 Hypovolaemic shock

	Stage 1, initial	Stage 2, compensatory	Stage 3, progressive	Stage 4, refractory
Blood loss	<15% (750 ml)	15% to 30% (750–1500 ml)	30% to 40% (1500–2000 ml)	≥40% (>2000 ml)
Heart rate	Normal or minimal tachycardia	Marked tachycardia (>100 beats/min)	Marked tachycardia >120 beats/min with weak pulse tachyarrhythmias	Extreme tachycardia, peripheral pulses not palpable >140 beats/min
Blood pressure	Normal	Normal	Systolic ≤100 mm Hg	Systolic ≤70 mm Hg
Pulse pressure	Normal	Decreased	Decreased	Markedly decreased
Respiratory rate	Normal	Mild tachypnoea	Marked tachypnoea ≥30 breaths/min	Pronounced tachypnoea
Capillary refill	>3 s (>10% loss)	>5 s	Much delayed	Absent
Central nervous system	Normal	Anxious/restless	Confused/agitated	Lethargic/consciousness
Urine output	Normal	20–30 ml/h	<20 ml/h	Negligible

Adapted from Graham and Parke, Irwin and Rippe, and Ledingham and Ramsay.^{1–3}

OUTCOME AND FOLLOW-UP

The patient's progress was uncomplicated. She was discharged 5 days later with appropriate vaccinations and long-term prophylactic antibiotics.

DISCUSSION

Hypovolaemic shock secondary to haemorrhage is classified into four stages according to the volume of blood loss and associated physiological responses, change in BP, HR, RR, urine output and mental status. Typically there is increasing tachycardia with progressive hypovolaemia. Often, HR is one of the first parameters to change with increasing blood loss (see table 1).^{1–3}

Between 7% and 28% of patients in hypovolaemic shock present with bradycardia.^{4–5} Common causes for bradycardia in a patient with shock are medications (such as digitalis or β -blockers) and neurogenic shock. Our patient demonstrated none of these.

Another cause of bradycardia in hypovolaemic shock that has been described in literature is severe periarrest haemorrhage.^{4–6} This was originally thought to be a poor prognostic indicator in a second phase of biphasic response to haemorrhage or volume loss. The first phase consists of tachycardia and normotension mediated by a baroreceptor mediated reflex, which causes vasoconstriction and cardiac acceleration. The second phase consist of falling BP and bradycardia, which is thought to be due to a vasodepressor and cardiac inhibitory response to a significant loss in volume, usually at least one-third of total blood volume.⁷ The biphasic pattern of response, was clearly not present in this case, as there was no evidence of the initial tachycardic response.

The occurrence of bradycardia in a patient with acute intra-abdominal haemorrhage may be explained by an increase in the parasympathetic drive. Indeed gradual pooling of blood in the abdominal cavity following trauma can result in stretching or irritation of the intraperitoneal cavity and a vagally stimulated reflex causing bradycardia. This

has been seen in patients with penetrating abdominal trauma and in those with intraperitoneal haemorrhage following abdominal surgery.^{7–11} However, to date there is no report of these findings in patients following blunt abdominal trauma. It has also been shown in animal and experimental studies where reflex bradycardic response to acute haemorrhage is abolished when the vagus nerve is cut or if the muscarine antagonist atropine is concomitantly administered.¹² From this we can understand and appreciate that bradycardia following intra-abdominal haemorrhage can be explained by this parasympathetic phenomenon, be it penetrating or blunt injury.

It may well be advantageous to have a bradycardia with significant blood loss. When venous return is reduced, bradycardia will result in a longer diastolic ventricular filling time. This could lead to maintenance or increase of cardiac output by an increase in the stroke volume. Alternatively, increased parasympathetic drive may improve tissue perfusion due to vasodilatation. This protective mechanism is seen in patients with critically reduced venous return. Here, vagally mediated cardiac depressor reflexes are activated by mechanoreceptors in the left ventricle. This has been described in patients with extra-abdominal bleeding.^{7–13}

In progressive hypovolaemia circulatory control is changed. Anatomical distribution and contribution of the components of the autonomic nervous system varies from individual to individual. It is speculated that the parasympathetic tone may have been more prominent in this case. Similarly it may be that the baroreceptors responded inappropriately causing an autonomic dysfunction with the lack of sympathetic drive.

In conclusion, patients with acute haemorrhage may not present with tachycardia. Indeed clinicians must be aware that in patients who present with hypotension and bradycardia (or lack of tachycardia) following blunt abdominal trauma may well be in hypovolaemic shock secondary to haemorrhage.

Learning points

- ▶ In hypovolaemia, vasovagal and cardioinhibitory reflexes are often ignored.
- ▶ In this case, acute haemorrhage did not present with an initial tachycardia and the assumption that progressive hypovolaemia (always) leads to an increase in heart rate (HR), may be wrong and even delay diagnosis.
- ▶ Increase in HR does occur in hypovolaemic shock, but in certain cases we may expect the measurement of HR to have little relevance in diagnosis and ongoing resuscitation manoeuvres.
- ▶ Patients who present with hypotension and bradycardia to the emergency department following blunt abdominal trauma may benefit from and increased period of close observation and clinical assessment.

Competing interests None.

Patient consent Obtained.

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