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Intra-abdominal Haemorrhage following a Contusion of the Abdominal Wall

BY

T. K. WHALEY, F.R.C.S. (Ed.)

AND

O. C. COLT, M.B., CH.B.
Salisbury, S. Rhodesia.

It is known that contusion of the abdominal wall may result in injury to the underlying viscera. The contusion may be due to a direct blow or the shock wave of air or underwater blast. In general, the viscera vary in their susceptibility to injury according to:

1. The thickness of their walls.
2. The degree of protection given by the pelvic or thoracic cages.
3. Their physiological state, whether flaccid or distended.
4. Their mobility.
5. Their state of health or disease.

Thus the thick walled stomach is less frequently injured than the thin walled small intestine. The normal liver and spleen, except in severe blast or crush injuries, are more frequently ruptured by blows which break the lower ribs than they are by blows on the abdomen. The distended bladder and the distended bowel are more easily ruptured than when empty. The relatively less mobile parts of the bowel—the upper part of jejunum, the lower part of ileum, the caecum and ascending colon—are more frequently injured than the remainder. In contradistinction to the healthy spleen, the diseased spleen may be injured by trivial blows or in the course of some confused incident, such as a drunken fall from a bicycle, in which case the diagnosis may not be at first apparent.

In addition to the viscera, the supporting mesentery may be damaged, leading to intra-abdominal haemorrhage, retroperitoneal haemorrhage or the deprivation of a segment of intestine of its blood supply and its subsequent gangrene.

Three abnormal constituents, therefore, may enter the peritoneal cavity: blood, urine or bowel contents.

Any injury to the abdominal wall may, if sufficiently severe, give rise to initial shock. Thereafter it is necessary to decide whether visceral damage has been done.

If the bladder has been ruptured the history of a full bladder prior to the injury, the painful and fruitless attempts to pass urine, the onset of peritonitis or, in the case of extra-peritoneal rupture, the presence of urine above the pubis are characteristic.

Both the diagnosis of and the distinction between rupture of the gut and intraperitoneal haemorrhage may be difficult. The following are some points that may be borne in mind.

When the gut is ruptured it is usually paralysed, so that it may be some time before peristalsis returns and its contents are expelled into the peritoneum.

The signs and symptoms of intra-abdominal haemorrhage will not be evident until about one pint of blood has escaped into the peritoneal cavity.

Should the shock of the initial injury not be ameliorated by adequate treatment after two hours, or should marked abdominal pain persist after four to six hours, it should be presumed that there is some internal injury.

A rising pulse rate with signs of anaemia indicates severe haemorrhage.

A rising pulse with continued vomiting and a rapid deterioration of condition without signs of anaemia is more suggestive of peritonitis. A valuable sign of this latter condition is the presence of free gas in the peritoneal cavity as revealed by loss of liver dullness.

Pain, muscular resistance, dullness in the flanks, a moderate fever and absence of peristalsis may occur in both conditions.

That the diagnosis of this type of injury may be far from obvious is illustrated by a case reported recently in which a man was seen in an out-patient department following a kick from a horse. He was sent home with a diagnosis of contusion and it was not until several days later that a bruised and gangrenous portion of gut gave way leading to a fatal peritonitis.

The following case is not unique, but is thought worth reporting:

A well-built muscular European male of 16 years was constrained, by the exuberance natural to his youth, to run as fast as he could carrying a stout piece of wood in his hand, much as one might brandish a sword. He was running close to a wall and the point of the piece of wood caught on a projecting portion of masonry, driving the butt violently into his abdomen slightly above and to the right of the
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umbilicus. The boy was “winded”; and when he had picked himself up he felt sufficiently unwell to retire to his bed. This took place at about four in the afternoon.

He vomited once during the evening and complained of pain in his abdomen, but passed an uneventful night. In the morning he had no appetite and still complained of pain. His bowels moved and he passed water normally.

Shortly after 1 p.m. on the day following his injury he was seen by one of us (O.C.C.). On examination, his colour was good, he was alert and co-operative and, though in considerable discomfort, he was not in acute pain. There was nothing to be observed on his abdominal wall but a small superficial abrasion about the size of a sixpence. His abdomen moved with respiration, but on palpation there was some muscular resistance and tenderness, particularly in the region of the blow and also in the right hypochondrium. His pulse rate was 100 and his temperature 99.5° F. There was no diminution of liver dullness and no dullness in the flanks. Active peristalsis was heard. His blood pressure was 120/80.

The possibility of an intra-abdominal injury was considered, but because the onset of a coincidental febrile illness could not be excluded and because of his good general condition, it was thought justifiable to see him later in the expectation that the true state of affairs would be more apparent.

It did not prove possible to see him again until 7 o'clock in the evening, at which time his pulse, temperature and blood pressure were virtually unchanged. His colour could not clearly be distinguished owing to poor lighting. He had severe abdominal pain, which he said had begun at about 3 p.m. His abdomen was markedly resistant and somewhat tumid. He had pain referred to both shoulders. Peristalsis was ominously absent. Immediate arrangements were made to have him admitted to hospital and for a surgeon (T.K.W.) to see him.

On admission to hospital his condition was as follows: he was obviously in great distress, complaining of severe upper abdominal pain which was referred chiefly to the left shoulder. His respirations were grunting. His abdomen was rigid, particularly in the upper left quadrant, with generalised rebound tenderness and with fixed dullness in the left flank and splenic region. His conjunctivae were pale, but his pulse rate was 90 per minute, the volume and tension being normal. His respirations were 20 per minute and his temperature 98° F. Although no peristalsis was heard, he stated he had passed flatus approximately one hour prior to admission.

An interesting feature was the almost total lack of external evidence of injury. There was a small abrasion and an associated bruise—hardly evident in the poor light in which the initial examination in hospital was conducted—an inch above and to the right of the umbilicus.

A tentative diagnosis of ruptured spleen was made and a laparotomy was done without delay.

Through a left upper paramedian incision the first feature of interest was the extensive laceration of the right rectus sheath and muscle deep to the trivial external injury. The rectus sheath was torn almost completely across anteriorly, the laceration extending round the medial border of the muscle into the posterior layer for half its transverse axis. The right rectus muscle itself was also damaged, being torn more than halfway across through its lowest tendinous intersection. A strand of the posterior sheath and perietal peritoneum left intact across the laceration had allowed a loop of small bowel to protrude between the rectus muscle and its posterior sheath, as occurs in interstitial hernia. The bowel loop showed no evidence of strangulation.

The peritoneal cavity was full of blood and a large quantity of clotted blood was found in the left paracolic gutter and in the neighbourhood of the spleen. The spleen was therefore delivered into the wound after the separation of small adhesions. Apart from small deficiencies in the parietal peritoneum—doubtless where the adhesions had been separated—no damage to the spleen or the splenic vessels could be found. The spleen was replaced and a further search for bleeding points was made. An examination of the entire length of the gastro-intestinal tract, the liver pancreas, kidneys and pelvic organs revealed a most extensive retroperitoneal haema-
toma, which also surrounded the colon at the hepatic flexure and extended into the mesentery of the small bowel. In addition, a tear, surrounded by haematoma, of the anterior layers of the great omentum at a point in its attachment to the greater curvature of the stomach, was found penetrating into the lesser sac. No overt bleeding point was located. At this stage a further inspection of the splenic area showed that no fresh bleeding had occurred from that organ. The rent in the omentum was repaired by resuturing it to the stomach, the appendix was removed as it was readily accessible and the patient's condition was good, and a careful reconstitution of the abdominal wall was done. The wound was closed without drainage.

Post-operatively, the patient complained of a mild distension for 48 hours, but thereafter his convalescence was uninterrupted and he left hospital on the tenth post-operative day.

Discussion

An interesting feature of this case was the fact that, despite the large amount of fresh blood in addition to the clotted blood within the peritoneal cavity at the time of operation, no active bleeding point was located. We must therefore conclude that the haemorrhage was the cumulative effect of bleeding from the grossly lacerated abdominal wall, the torn omentum and the retroperitoneal haemorrhage.

From a diagnostic point of view, it may be noted that there was an interval of approximately 24 hours between the original injury and the onset of unequivocal signs and symptoms of intra-abdominal damage.
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