Rectus sheath haematoma: 'a diagnostic dilemma ?'

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Rectus Sheath Haematoma is a relatively rare presentation in the context of acute Accident and Emergency presentations. Correct diagnosis and subsequent management depend on sound clinical acumen and history taking with appropriate use of diagnostic aides in order to avoid prolonged and inappropriate management of such cases. We present an interesting case of a young fit male whose history alone suggested the diagnosis despite an initial diagnostic dilemma.

CASE REPORT

A fit 18 year old male rugby player presented to Accident and Emergency Department with a 4 hour history of acute onset progressive right lower quadrant abdominal pain. Such pain, described as sharp in nature and exacerbated by pelvic movement and coughing, was initially noticed during a rigorous exercise programme on a rowing machine. He was normotensive, afebrile with a pulse rate of 86 bpm (average).

Examination revealed quite a marked degree of abdominal guarding and rigidity particularly in the right lower quadrant. Nothing else remarkable was in evidence on further examination. Subsequent investigation revealed normal Fbc, Bioprofile, and plain radiographic studies of the abdomen. The patient remained in extremis and treated accordingly with incremental doses of intravenous opiate analgesia. A provisional diagnosis of acute appendicitis was made and admission arranged for surgical management.

Despite refractory presentation a decision was made to continue with supportive symptomatic management. The patient showed symptomatic improvement with conservative management (intravenous fluids and analgesia).

'Next-day' ultrasound abdomen revealed a large Rectus Sheath Haematoma. The patient quickly improved and was discharged the following day on oral analgesia as required.

Appropriate advice was issued regarding sports, exercise, and training.

DISCUSSION

Rectus Sheath Haematoma, as an acute abdominal presentation,[1] is relatively uncommon. Reported rates vary,[2] but current literature suggests a female:male preponderance of 2–3:1 in the 50 to 60 year old age group (however, equal distribution in the younger age groups is noted despite different aetiology). Overall incidence is not quantified owing to rarity of presentation.

Rectus Sheath Haematoma is an accumulation of blood in the anterior Rectus abdominis muscle. Primary causation is due to either epigastric vessel rupture or muscle body tear. This may occur in any abdominal quadrant but typically sub - umbilical due to Rectus Abdominis *posterior wall supportive deficit* below the level of the linea semilunaris (weak transversalis fascia and peritoneum being the essential supportive framework for Rectus Abdominis) and *relative infrastructural rigidity* (firm adherence of inferior epigastric penetration arteries to rectus abdominis). As in our case, intense, perhaps unaccustomed, muscular exertion induced labile contractile muscle lengths and *shear forces* in a plane opposite to epigastric penetration vessels resulting in immediate haematoma formation.

Predisposing factors associated with Rectus Sheath Haematoma formation are varied and include hypertension, arteriosclerosis, old age, obesity, direct abdominal trauma, anticoagulant therapy, previous abdominal surgery, pregnancy and acute intra-abdominal pressure fluctuations (straining, coughing, exertion) – the latter illustrating male predominance, as in our index case.

Interestingly, one series reported a case related to intramuscular administration of low molecular weight subcutaneous heparin.[3]

Clinical presentation is often non-specific[5] – pain and swelling/mass in any one of the four abdominal quadrants (left upper quadrant being the site of least frequency in reported series) – typically the lower quadrants being the sites of most frequent occurrence.[4] Pain, often in isolation of mass/swelling, is usually of acute onset, of moderate severity with signs suggestive of peritoneal irritation. The presence of an associated mass is often highly suggestive, particularly if right sided and is considered diagnostic if extends to midline. Some series' have quoted associated signs – notably *Fothergill's Grey Turner's*, *Cullen's,* and dysuria/urinary frequency – depending on breach of peritoneum but these are typically associated with delayed presentation beyond 48 hours.

Supportive data may occasionally include a mild leucocytosis, a low grade pyrexia or a corroborative drop in haemoglobin (clearly extent related), however, these are considered unusual.

Diagnostic conclusion is usually successful with a combination of clinical awareness and radiographic imaging – Ultrasound or Computed Tomography. Ultrasound is usually the investigation of choice due to high sensitivity rates (approaching 100% in most series), time/cost efficacy and radiation safety protocol. Classical ultrasonographic appearances range from sonolucent (early stage) to sonodense (late stage) appearance with time from initial injury.

Computed Tomography is considered more sensitive an investigation and useful in cases of inconclusive ultrasound but due to issues relating to time/resource management is less commonly employed. Magnetic Resonance Imaging has been less employed and is considered as sensitive as the aforementioned. Should the diagnosis continue to prove elusive, more invasive methods such as diagnostic needle aspiration and laparotomy have been suggested, however, the former has not received much acclaim due to reasons relating to sepsis propagation.[1]

A conservative non interventional approach to diagnosis/management has received acceptance most recently. Such approach, of course, advocates analgesia, serial clinical assessment, bedrest, haematoma compression, icepack application, and managing the predisposing cause. Clearly, haemodynamic compromise necessitates operative intervention - clot evacuation and vessel ligation via laparotomy or laparoscopic[6] approach is usual. Levy *et al*[7] reported successful interventional outcome with *Gelfoam embolization* however this method has not received much acclaim.

Rectus Sheath Haematoma may prove to be a diagnostic dilemma; differential diagnoses would have to include ovarian pathology (torsion, cyst), appendicitis, intestinal pathology (obstruction, neoplasia, perforation) and strangulated herniae.

As our case demonstrates, reasonable prolonged conservative management (in the context of clinical awareness and corroborative diagnostic exclusion) should negate the need for intervention in the case of Rectus Sheath Haematoma assuming haemodynamic stability.

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Accidental human poisoning with a veterinary tranquilliser

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Detomidine is an alpha-2 adrenoceptor agonist drug, which is used in veterinary medicine for sedation of large animals.[1–4] It is highly potent and has sedative and analgesic properties.[1 2] This paper reports a case of accidental human poisoning with this drug.

CASE REPORT

A 35 year old farmer was preparing to sedate a bull in order to pare its hooves. No vet was present. The farmer had 5 ml of Domosedan (detomidine hydrochloride 10 mg/ml) in a 5 ml syringe with a 21G×1.5″ (0.8×40 mm) needle attached. The bull moved as the farmer was about to give this medication and he injected himself in the thenar emminence of his left hand. He later said that he had been holding the syringe by the barrel rather than the plunger and he did not think he had injected a significant volume of the drug. He claimed that he could not recall how he came to possess the drug. He had no relevant medical history, was on no medication, and had last drunk alcohol 4 days previously.

Within 10 minutes of this accident, the farmer began to feel drowsy, dizzy, and relaxed. His companions noted that he was not performing his tasks normally. When questioned, he responded in a slurred and drunken manner. His gait became unsteady. He was carried to a vehicle and taken to the emergency department.

On arrival he was noted to be drowsy, with slurred speech, and a passively euphoric state, but his Glasgow Coma Score (GCS) was 15. His heart rate was 44 beats/min and his blood pressure 106/48 mmHg. A small superficial puncture wound was noted on the left thenar emminence. No other abnormality was apparent on examination. His ECG showed a sinus bradycardia with no evidence of heart block or ischaemia.

Peripheral intravenous access was obtained and an infusion of 0.9% sodium chloride was started. Two boluses of atropine 500 μg were given. Within minutes, a sustained rise in heart rate and blood pressure was noted, but the patient remained significantly drowsy. He was given 2 litres of intravenous saline over 2 hours. TOXBASE (an online poisons database; www.spib.axl.co.uk) was consulted. The patient was observed overnight with cardiac monitoring but received no further medications. Over the next 16 hours he regained full consciousness and his pulse and blood pressure remained normal. He was discharged home.

DISCUSSION

Detomidine (trade names Domosedan and Dormosedan) is used for sedating horses and other animals to facilitate examination and surgical procedures.[1 2] Detomidine causes a bradycardia with blood pressure initially raised and then normal or low, reduced cardiac output, and slowing of intracardiac conduction with partial sinoatrial and atrioventricular block. Respiratory depression and diuresis may occur.[1 2] Detomidine potentiates the actions of other sedative and analgesic drugs. It has similar effects to clonidine.[5–7]

TOXBASE (www.spib.axl.co.uk/toxbase/poisons%20information/d/detomidine.htm) mentions that a dog that chewed a vial of detomidine had salivation, sedation, collapse, hypotension, bradycardia, arrhythmias, and coma, which lasted for 18 hours. The treatment advised by TOXBASE in human poisoning is supportive, with observation for at least 4 hours, or longer if symptomatic, and monitoring of blood pressure and cardiac rhythm. Hypotension should be corrected by raising the foot of the bed and by giving intravenous fluids, and vasopressors should be considered in severe cases. Bradycardia may require treatment with atropine and rarely with cardiac pacing. Severe and persistent hypertension may be treated with phentolamine.

The only previous report of detomidine poisoning in humans is a single case of deliberate injection of detomidine 50 mg and butorphanol 100 mg in a 36 year old man.[8] Butorphanol is an opioid drug for which the antidote is naloxone.[9] The patient presented with a GCS of 6 and required a continuous infusion of naloxone, but his heart rate never fell below 70 beats/min and his blood pressure never below 110/80 mmHg. He made a full recovery.