so in trauma, that its spontaneous occurrence seems to have been forgotten. This diagnosis should be borne in mind even in situations where the patient is clinically well and has signs suggesting a simple pneumothorax alone. Indeed if clinical assessment reveals hyper resonance and decreased air entry, there is likely to be a significant pneumothorax and a more thorough clinical examination to determine tracheal and mediastinal position will identify those patients in whom it is under tension. In addition to the signs of simple pneumothorax, physical examination may demonstrate a deviated trachea and/or mediastinal shift as evidenced by displaced apex beat and resonance over the sternum.

As can be seen from cases 1 and 2, young patients with good physiological reserve may actually appear to be clinically stable, however, they are at risk of sudden deterioration and possible cardiac arrest. In cases 1 and 3, the patient was subsequently found to have previously undiagnosed lung disease; this demonstrates the need for thorough investigation of any underlying cause after treatment of the acute event.

A literature search revealed very little on spontaneous tension pneumothorax in previously well patients.[4] Most cases involved pre-existing disease or were associated with surgery, intermittent positive pressure ventilation or trauma.

Conclusion

A tension pneumothorax is a clinical diagnosis that should not be overlooked, even in the absence of trauma. The trachea may be central and the patient may appear clinically well at presentation. A high index of suspicion and active confirmation of tracheal and mediastinal position will help confirm the clinical diagnosis of tension pneumothorax. Patients must not be sent for radiology. Immediate needle thoracocentesis and chest drain insertion is the emergency treatment whatever the cause.

Contributors
Holloway and Harris initiated the case report jointly following the presentation of two cases at a training meeting (case 1—Harris, case 2—Holloway). Holloway identified two further cases and conducted a literature search. The discussion and conclusion were jointly written. Harris acts as guarantor for this paper.

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Aorto caval fistula—the "bursting heart syndrome"

S Leigh-Smith, R C Smith

Abstract

Aorto caval fistula is one of the less well recognised complications of abdominal aortic aneurysm seen in accident and emergency departments. It presents in a number of different ways the commonest of which is high output congestive cardiac failure with warm peripheries. Initial diagnosis is based on the index of suspicion of the clinician. However, early diagnosis by the emergency physician and early surgery can markedly improve the patients prognosis.

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Keywords: aorta caval fistula

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Case report

A 79 year old man was seen in the accident and emergency (A&E) department five hours after a brief syncopal episode. He described a sudden onset of fast palpitations, dizziness, nausea, one episode of vomiting, sweating and a "feeling as though his heart was going to burst." These all subsided spontaneously within five minutes. He was a smoker and was soon due to undergo an elective graft replacement for a 9 cm abdominal aortic aneurysm (AAA). His main complaint on presentation was lethargy since his earlier "funny turn."

On examination he was pale but warm and well perfused. His jugular venous pressure (JVP) was increased, he had a pansystolic flow murmur, a tachycardia of 105 and BP 105/55. Abdominal examination revealed a large but non-tender pulsatile mass and a fullness in his right loin. Abnormal investigations included a mild hypoxia on air, an ischaemic ECG with left axis deviation and a mild neutrophilia.

An initial differential diagnosis of (a) arrythmia, (b) myocardial infarction, (c) leaking abdominal aneurysm was made. Blood was cross matched, a myocardial infarction screen was started, he was put on telemetry and a fluid challenge was performed.

Over the next few hours he became oliguric and shocked with no further evidence of myocardial infarction or arrythmia. He was therefore taken to theatre for repair of a suspected leaking aneurysm. An aorto caval fistula was surprisingly discovered and successfully repaired along with insertion of an aorto

bi-femoral graft. He did well postoperation after transient worsening of renal function and bilateral leg oedema that spontaneously improved.

Discussion

Spontaneous aorto caval fistula is one of the less well recognised complications of an atherosclerotic AAA and yet is more common (10% of ruptures) than aorto duodenal fistula (2% of ruptures), which may be an easier diagnostic challenge.[1] Although described as rare in most references, its quoted incidence is very variable from as low as 0.22%[2] to as much as 10%[1 3] of all AAAs. Spontaneous rupture of an atherosclerotic plaque in an existing AAA is the commonest cause (80%) with trauma (15%) and iatrogenic after lumbar disc surgery (5%) less common causes.[4]The incidence of all AAAs is increasing and therefore so will the incidence of its complications.[5]

The prognosis of this condition is very dependent on how early it is diagnosed and particularly if this is done before operation. Although survival up to two months without surgery has been reported[7] it is generally accepted that prompt surgery improves survival.[6] Diagnosis and surgery before development of shock can double the chances of survival from 25% to 50%.[8] Diagnosis before surgery is desirable as it allows preparation by the surgeon for appropriate operative techniques,[6] care by the surgeon not to dislodge debris into the inferior vena cava causing a pulmonary embolism,[5] insertion of a pulmonary artery catheter for the difficult haemodynamic control intraoperatively,[6 9] and avoidance of early fluid overload worsening the cardiac failure.[1] In one series mortality was 15% if diagnosis was made before surgery in contrast with 100% mortality if it was not.[10]

Early diagnosis is hence the key to improving patient outcome in this condition and that is dependent on the physicians awareness of it.[11] The problem is the different ways in which it can present. In fact three authors describe "classical" presentations all of which vary slightly.[1 4 5] Pain is even described as being absent,[12] or always present.[8]

Symptoms and signs may be attributable to the high venous return and arterial insufficiency to other structures caused by the fistula itself or attributable to associated intraperitoneal or retroperitoneal rupture. This sudden increase in venous return to the heart along with decreased peripheral vascular resistance can lead to cardiac arrest, but more commonly leads to an acute compensatory phase.[1]

Review of the medical literature shows the commonest symptoms and signs to be[1 4 5 8 12–15]:

(1) High output cardiac failure (dyspnoea, increased JVP, pulmonary oedema and widened pulse pressure)

(2) Abdominal bruit and thrill

(3) Palpable abdominal aneurysm

(4) Oliguria

(5) Consequences of regional venous hypertension (leg oedema with/without cyanosis, haematuria and rectal bleeding)

(6)Variable symptoms and signs (shock, abdominal pain, chest pain, low back pain, scrotal oedema, tenesmus, priapism, and poor peripheral pulses)

Once the diagnosis is suspected there are various options open to confirm it providing the patient is stable. Central venous blood may have high oxygen saturations.[6] Doppler ultrasound in A&E will show the AAA and may even demonstrate the fistula.[1 16] Angiography is considered the gold standard but only if there is no renal impairment or shock.[4] Computed tomography, magnetic resonance imaging and radioisotope studies have all been used to make the diagnosis.[4 13 16–19] Local resources and expertise are probably the most important factors in choice of diagnostic modality.[12]

Conclusion

The condition may not be as rare as expected. Considering it as a diagnosis in "arteriopaths" with acute onset of cardiac failure and listening for a bruit in all patients with a ruptured AAA[20] may increase the diagnosis rate in the A&E department.

Our case with his description of a sensation of his "heart bursting" also shows that the patient can be describing exactly what is happening to him and we should bear this in mind when using structured closed questions in our history taking!

Contributors

Dr S Leigh-Smith was responsible for the initial assessment, investigation and resuscitation of the patient and conducted the background literature review. Mr R C Smith was responsible for the subsequent assessment, operative procedure and subsequent management of the patient. Both authors contributed to the text of the article. Mr R C Smith is the guarantor.

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Screwdriver assaults and intracranial injuries

Matthew G Tutton, Bhupal Chitnavis, Ian M Stell

Abstract

Four patients with intracranial penetrating injuries from screwdrivers are presented. Two cases were fatal; the others were left with functional deficits. In two of the patients a penetrating injury was not suspected initially because the history was limited and the significance of the small entry wounds were not appreciated. Unless these wounds are carefully examined a penetrating injury is easily overlooked.
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Keywords: skull injury; brain injury; penetrating wound

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Screwdrivers are fortunately only rarely used as weapons. However, when used in an assault on the head the concentration of force into the small area at the tip of these rigid tools may enable penetration into the vault of the skull. Once through the bone the shaft of the screwdriver may then pivot around the entry point in the skull, causing an arc of intracranial injury. If the screwdriver is withdrawn, then clinical examination later may miss the small entry wound, and the seriousness of the injury may not be appreciated as intracranial injuries from screwdrivers have a high mortality rate.[1–3]

These four patients who were referred to a regional neurosurgical centre illustrate both the seriousness of this penetrating injury and how easily it can be missed.

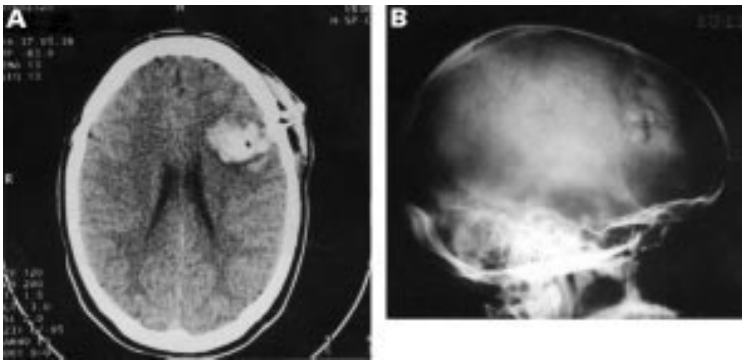

Figure 1 (A) Computed tomography showing left frontal intracerebral haematoma and skull vault fracture with (B) corresponding plain skull radiograph showing a depressed skull fracture.

Case reports

CASE 1

A 26 year old man presented to his local accident and emergency (A&E) department after an assault with a screwdriver. Initially he was thought to be intoxicated and the only apparent injury from the screwdriver was a small laceration of his left pinna. He was discharged home, but returned the next day with a headache, increasing confusion, vomiting and a dense right hemiplegia. Computed tomography showed intracranial haemorrhage within the left parietal lobe and extending into the lateral ventricles. This intracranial injury lay directly beneath the site of the laceration to the pinna. He was transferred to the regional neurosurgical unit, where he was managed conservatively. There was gradual resolution of his right hemiplegia and mild dysphasia. He was transferred to a rehabilitation unit 17 days after admission.

CASE 2

A 26 year old man was brought by ambulance to A&E after an assault in the street. Although he had blood over the left side of his head no wound was noticed. He smelled strongly of alcohol. He was mildly confused, with a Glasgow Coma Score of 14/15, and was reluctant to speak. He was initially observed to allow him to "sober up" and it was not until several hours later, when he had not improved, that closer examination revealed a 1 cm laceration and slight swelling in the left parietal region. No other injury was noted. Skull radiographs showed a depressed skull fracture and subsequent computed tomography showed a large intracerebral haematoma in the left frontal lobe with an overlying skull vault fracture (fig 1). He was admitted to the neurosurgical unit before transfer for rehabilitation.

CASE 3

A 26 year old man was reported to have been assaulted with a sharpened screwdriver. At presentation he had a GCS of 4/15, a fixed dilated left pupil and was bleeding from a point just anterior to the left ear. Computed tomography demonstrated a small depressed fracture 4 mm in diameter above the floor of the left temporal fossa with an acute left sided