

CASE REPORTS

Injuries sustained by aircrew on ejecting from their aircraft

C A Read, J Pillay

Abstract

This paper describes some of the injuries sustained by the aircrew who ejected from their aircraft after a mid-air collision, and discusses the types of injury that such patients may suffer.

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Keywords: ejection seat; spinal injury

We report the cases of four patients who were involved in a military aircraft accident. The pilot and navigator of two Tornado F3 fighter jets ejected after a mid-air collision. The first aircraft lost two thirds of its right wing and part of its tail. The second plane lost part of its right wing. Both aircraft were destroyed on impact with the ground some five miles apart.¹

Case 1

THE NAVIGATOR OF THE FIRST AIRCRAFT (35 YEARS)

His Glasgow Coma Score (GCS) was 13/15 (E4M6V3) at the scene but on arrival at the accident and emergency (A&E) department, it had dropped to 10/15 (E4M3V3). The right pupil was noted to be larger than the left and both pupils reacted briskly. The pulse, blood pressure and respiratory rate were within normal limits.

Radiographs of the cervical spine showed evidence of C1-C2 subluxation with 1 cm forward displacement of C1 on C2. The patient was kept immobilised on the spinal board and intubated, paralysed and his lungs ventilated. Computed tomography (CT) of the brain showed a left frontal haematoma, with no mass effect. The patient was then airlifted to the regional neurosurgical centre for further management. He was managed conservatively in the intensive treatment unit and extubated after 24 hours.

It was then noted that the patient had left upper extremity weakness. Further investigation by CT and magnetic resonance imaging (MRI) (figs 1 and 2) showed atlantoaxial dislocation with avulsion of the transverse ligament and gross posterior soft tissue disruption.²

Six days after admission, the patient underwent C1 to C2 modified Gallie and transarticular C1/C2 fusion and fixation. His postoperative course was uneventful and on discharge

to his local hospital for rehabilitation, the patient was walking with a frame and able to converse. At six months after the operation, he had complete recovery of motor power in the right arm. Unfortunately weakness in the left



Figure 1 Lateral radiograph of the cervical spine showing widened atlantodental interval.

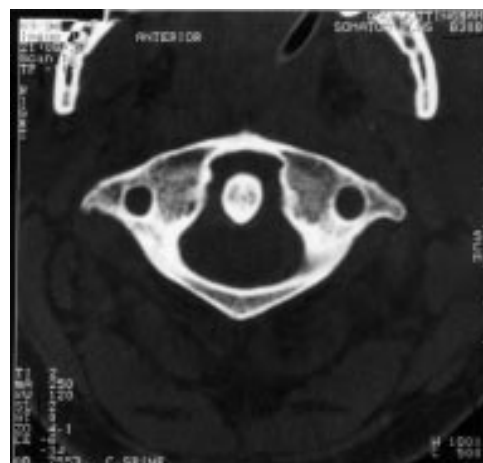


Figure 2 Axial CT at C1. The widening of the atlantodental interval is shown.

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Figure 3 T2 weighted MRI showing central disc protrusion at C5/C6, with an increase in T2 signal in the cord suggestive of a contusion.

arm has persisted. This is thought to be attributable to brachial plexus injury, based on clinical examination findings; unfortunately this has prevented his return to flying.

Case 2

THE PILOT OF THE FIRST AIRCRAFT (35 YEARS)

There was loss of consciousness from the time of ejection and the first thing he remembered was lying on the ground at the scene. He was then able to get up and walk around; however on doing so he developed paraesthesia of both upper limbs below the elbow. This was his only symptom and in particular he did not complain of any neck or chest pain.

On examination he had a GCS of 15 and was alert and orientated. Vital signs were normal. Examination of the neck was normal except that extension of the cervical spine worsened the patient's symptoms. The power in his upper and lower limbs was noted to be globally reduced and was graded at 3/5 (MRC grading)

Though the patient continued to complain of paraesthesia below both elbows, there was no objective sensory abnormality on testing. Plain radiographs of the cervical spine showed no bony abnormality but there was some soft tissue fullness at the level of C6/C7.

MRI was arranged and this showed central disc protrusion at C5/C6 with an increase in T2 signal in the cord suggestive of a contusion (fig 3).

As the patient was intent on returning to active flying, he was referred to the spinal unit where he underwent an elective C5/C6 discectomy and interbody fusion.

He made an uneventful postoperative recovery and returned to active flying within six months.

Case 3

PILOT OF THE SECOND AIRCRAFT (30 YEARS)

This man did not lose consciousness and he walked around helping with the rescue after the crash. He complained of lower back pain but there was no neck pain. On examination his vital signs were normal with GCS of 15. On examination, there was some tenderness over the lower cervical spine only with no distal neurology. Radiographs of the spine raised the suspicion of C6 compression. CT confirmed slight anterior compression of C6 but there was no derangement in mechanical alignment. He was treated conservatively and made a good recovery. He was able to return to active flying duties.

Case 4

THE NAVIGATOR OF THE SECOND AIRCRAFT (34 YEARS)

There was no history of loss of consciousness and he had recall of the ejection. On arrival at hospital, he complained of left facial pain only. There were no neck symptoms.

On examination he was alert and orientated. His vital signs were within normal limits. He had a minor facial laceration. He was mildly tender over the paraspinal muscles of the neck and lower thoracic spine. There was no distal neurology. Cervical and thoracic spine radiographs were normal but there was a suggestion of slight loss of height at the level of T9. The patient was admitted for observation. He remained stable and he was well enough to be discharged the next day, to be reviewed in the fracture clinic. He was referred for a course of physiotherapy but as he had persistent localised tenderness over the lower thoracic spine, a bone scan was arranged. It confirmed an increased uptake at the level of T9.

The patient progressed well with physiotherapy to strengthen his back muscles and was then discharged from the clinic. He returned to flying duties.

Discussion

The ejection seat has been responsible for saving the lives of thousands of pilots around the world since its introduction in the late 1940s. Typical survival rates quoted in the literature vary from 80–97%.³

On most modern seats escape is initiated by pulling a seat firing handle. This fires a cartridge and gas from this is then piped around the seat to initiate⁴:

- 1 Powered shoulder retraction
- 2 Canopy jettison or fragmentation by miniature detonating cord (MDC)
- 3 Firing of ejection gun to propel seat from aircraft
- 4 Firing of rocket pack to propel seat away from aircraft

The medical problems encountered with ejection can be classified as follows:

- 1 Injuries from the emergency that causes ejection—fire or collision.

- 2 Canopy jettison: burns from "MDC splatter" and cuts from fragmented plastic. For these reasons, aircrew are always advised to wear their visors down, to protect the face.
- 3 Firing of ejection gun: spinal injuries.
- 4 Entering airflow: wind blast may cause lung damage; seat tumbles at variable speed, which may be as high as 180 rpm. (All seats have a drogue parachute or deployable aerodynamic panels to prevent tumbling); flail injuries to extremities.
- 5 Parachute deployment: snatch injuries.
- 6 Landing: lower limb injuries.

Probably the most serious injuries are those of the head and spinal column. There are many factors that influence whether aircrew who eject sustain a spinal fracture or not. These include: the design of the seat, aircraft speed and attitude at time of ejection and age of the patient.

It is generally accepted that radiographic evidence of vertebral fracture can be found in 30%–70% of aircrew after ejection.^{5 6} To avoid excessively high acceleration rates, the ejection gun fires in three stages with a short delay between each (a few milliseconds). Individual vertebrae are able to withstand large compressive loads if applied at right angles to the plane of the intervertebral disc. However, because of engineering constraints the initial thrust is an impulsive load that is experienced at an angle to the long axis of the spine. In our case, the navigator who was severely injured was given no warning of the ejection as there was not enough time. This is called a command ejection. The ejection seat had a power retraction unit fitted, however, which would have ensured his spine was aligned in the correct position before any seat movement occurred.

The lower thoracic spine is especially prone to injury.⁷ This part of the spine is subjected to the static load of the trunk and head transmitted through the vertebrae above it. At this region of the spinal column the vertebral end plate has the highest loading per unit area.⁴ The vertebral bodies act as dampers, the blood within them being squeezed out under compression, before vertebral collapse or fracture.⁸ Spinal injuries from ejection do occur at other sites, as demonstrated in the cases that have been described here. The mechanism of the injuries we have described is complex and

includes several factors such as aerodynamic helmet lift, head flail, impact of the head on the ejection seat head box.

After the accident referred to in this report, the Royal Air Force now sends all of its aircrew who have ejected from aircraft for a complete spinal MR scan (personal communication).

Conclusions

A&E physicians may be called to see aircrew who have ejected from their aeroplanes. The RAF aircraft accident rate amounts to approximately 7–9 per year, not all of which involve ejection. The ejection rate for 1998 was 0.17 per 10 000 flying hours (personal communication). It is the fact that ejectees are seen so infrequently that leads to relative ignorance by receiving hospitals of the forces involved and the possible injury patterns. After initial resuscitation it is essential that particular attention be paid to examination of the spine and peripheral nervous system. To avoid missing potential injury further imaging of the spinal column with either CT, or ideally MRI should be considered mandatory in all patients who have ejected from aircraft.

Contributors

Mr Colin Read wrote the initial draft of the paper and did the follow up on the patients mentioned in the paper. This included obtaining written consent for publication from three of the four airmen concerned. It was not possible to contact the fourth airman as he had left the airforce. The written permission of Wing Commander Broadbridge, RAF Innsworth was also obtained and he has authority to consent from the point of view of the RAF and the MOD. Mr Jai Pillay was the consultant responsible for the patients at the time of initial injury and was responsible for reviewing the initial draft of the paper and overall supervision. Mr Jai Pillay is guarantor for this article.

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Conflicts of interest: none.

- 1 Ministry of Defence. *Military Aircraft Accident Summary of a Royal Air Force Board of Enquiry*. Coningsby, Lincolnshire: Ministry of Defence, 1997.
- 2 Boos N, Khazim R, Kerslake RW, et al. Atlanto-axial dislocation without fracture. *J Bone Joint Surg (Br)* 1997;**79-B**: 204–5.
- 3 Milanov L. Aircrew ejections in the Republic of Bulgaria, 1953–93. *Aviat Space Environ Med* 1996;**67**:364–8.
- 4 Anton DJ. *Escape from aircraft*. In: Ernsting J, King P. *Aviation medicine*. 2nd ed. Guildford: Butterworth Heinemann, 1988:200–15.
- 5 Newman DG. The ejection experience of the Royal Australasian Air Force:1951–92. *Aviat Space Environ Med* 1995;**66**:45–9.
- 6 Sandstedt P. Experiences of rocket seat ejections in the Swedish Air force:1967–87. *Aviat Space Environ Med* 1989;**60**:367–73.
- 7 Macalister A. *A textbook of human anatomy*. London: Griffin, 1889.
- 8 Roaf R. A study of the mechanics of spinal injuries. *J Bone Joint Surg (Br)* 1960;**42-B**:810–23.

Adult spinal cord injury without radiological abnormality

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Abstract

Spinal cord injury without radiological abnormality is rare in adults. A case is described of a 61 year old man who fell 15 feet from a ladder striking his head on a wall who presented with neck pain and with motor and sensory neurological abnormalities in his limbs. Plain radiographs of the neck revealed no fractures or dislocations. Further imaging with computed tomography and magnetic resonance imaging revealed an osteophyte fracture with associated cord contusion at

the C5 level. Careful neurological examination is essential in all cases of potential spinal injury.

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Keywords: spinal cord injury

Case report

Spinal cord injury without radiological abnormality (SCIWORA) is well recognised in paediatric trauma.¹ The laxity and flexibility of the cervical spine in children, allows for excessive movement without bony injury and can result in cord damage without radiological abnormality. This is rare in the adult population.¹ We present a case of adult SCIWORA to highlight the importance of thorough neurological examination and early diagnosis of cord injury in trauma.

A 61 year old man fell 15 feet off a ladder, hitting his head on a brick wall. He was not rendered unconscious and was helped up to his feet by his family. The ambulance crew found him walking around in no apparent distress, but complaining of some neck pain. He was immobilised with a collar, head blocks and tape, and was transported on a spinal board to the accident and emergency department.

On arrival, he was found to be fully conscious with no evidence of significant head injury. He had a pulse of 60, his blood pressure was 150/80. He complained of pain in his neck, tingling in his arms and was unable to extend his left elbow.

He was tender over the mid-cervical spine. Initial neurological examination revealed bilateral reduction in sensation to light touch over the C6–C8 distribution and reduced power in wrist flexion bilaterally. In the lower limbs there was symmetrical reduction in sensation extending up to the T12 level. Power at the left ankle and left hallux was reduced in both flexion and extension. The tone was normal in all four limbs.

Radiographs of the cervical, thoracic and lumbar spines showed degenerative changes but no soft tissue swelling, loss of alignment or fractures (fig 1).

Subsequent computed tomography of the cervical spine demonstrated a fracture of an osteophyte on the anteroinferior border of C3, and a small osteophytic fragment from the posterior superior aspect of C5, which had entered the spinal canal (fig 2).

After discussion with the orthopaedic consultant, the patient was given methylprednisolone and was nursed at 45 degrees in a hard collar.

Magnetic resonance imaging was performed on the following day. It demonstrated a well defined area of increased signal intensity on T2



Figure 1 Plain cervical spine radiographs.

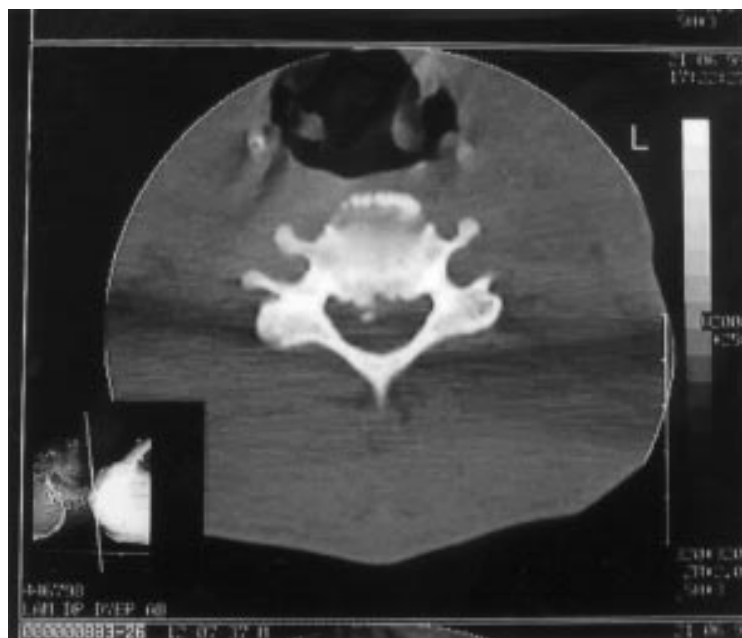


Figure 2 Computed tomography demonstrating an osteophyte fracture with fragment lying in the spinal canal at the C5 level.



Figure 3 Magnetic resonance imaging demonstrating spinal cord contusion at the C5 level.

weighting and FLAIR sequences within the cord at the level of C5. T1 weighting showed no increase in the signal pattern. These findings were considered to be consistent with localised contusion and oedema of the spinal cord at the C5 level (fig 3).

Forty eight hours after admission, neurological examination revealed spasticity in the left lower limb and reduced tone in both upper limbs. Reflexes were present and brisk in both lower limbs. Power was reduced in both upper limbs with the loss being greatest in the left upper limb and in the left lower limb.

Table 1 Motor power at 48 hours

	Upper limbs			Lower limbs	
	Right	Left		Right	Left
Shoulder abduction	5	5	Hip flexion	5	3
Elbow flexion	5	4+	Hip extension	5	3
Elbow extension	4	2	Knee flexion	5	3
Wrist flexion	3	1	Knee extension	5	3
Wrist extension	3	1	Ankle plantar flexion	5	5
Finger abduction	2	0	Ankle dorsiflexion	5	4

Joint position sense was reduced in both upper limbs but normal in the lower limbs. Sensation to pin prick was reduced on the left in the distribution of C6 to S4. Table 1 summarises the neurological findings.

A diagnosis of central and left anterior spinal cord syndrome at C6 level was made. A neurosurgical opinion was sought; flexion/extension views were requested. These revealed no instability of the cervical spine. Conservative treatment was advised. The patient's neurological signs did not improve.

Discussion

SCIWORA in paediatric trauma is well documented and hence clinical suspicions of spinal cord injury are not allayed by normal views of the cervical spine on plain radiographs. This case of adult SCIWORA demonstrates the importance of relying on clinical skills to identify spinal cord injury even in the non-paediatric population. Normal plain radiographs of the adult cervical spine do not exclude neurological damage in the presence of an abnormal neurological examination.

Early diagnosis of spinal cord injury is important to optimise outcome. Administration of a bolus dose of methylprednisolone (30 mg/kg) within three hours of injury followed by a methylprednisolone infusion of 5.4 mg/kg per hour for 24 hours or administration of a bolus dose of methylprednisolone within eight hours of injury followed by an infusion for 48 hours results in a significantly better neurological outcome.² In this case of adult SCIWORA methylprednisolone was administered based on the clinical findings and results of computed tomography performed on the day of admission, before definitive radiological imaging by magnetic resonance imaging.

Funding: none.

Conflicts of interest: none.

1 Advanced Life Support Group. *Advanced Paediatric Life Support*. 2nd ed. London: BMJ Publishing Group, 1997:174.

2 Bracken MB, Shepard MJ, Holford TR, *et al*. Administration of methylprednisolone for 24 or 48 hours or tirilazad mesylate for 48 hours in the treatment of acute spinal cord injury. Results of the Third National Acute Spinal Cord Injury Randomised Control Trial. National Acute Spinal Cord Injury Study. *JAMA* 1997;277:1597-604.

Hyperventilation: cause or effect?

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Abstract

A young person presenting with shortness of breath is common to the accident and emergency department. Usually this hyperventilation is anxiety related or a panic attack, but sometimes it can be caused by a serious underlying condition like pulmonary embolus. Acute shortness of breath in any patient should never be dismissed lightly. It is important to realise that pulmonary embolus can present without chest pain and with shortness of breath as the major symptom. Such patients can be distinguished by close attention to history and examination, risk factors for thromboembolic disease and the use of basic investigations (electrocardiogram, chest radiography and arterial blood gas analysis). A serious cause for shortness of breath must be excluded before labelling it as "hysteria" or "panic".

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Keywords: hyperventilation

Doctors in accident and emergency (A&E) medicine are confronted with a large number of patients that have the label of a "panic attack". Typically these are young patients who are hyperventilating. Not all hyperventilation in the A&E department is "cause"; some of it is "effect". The difficulty is spotting the one when hyperventilation is effect and not cause. It is important for emergency physicians, who are often junior doctors to consider any history of shortness of breath as a major symptom. Such patients require detailed assessment to exclude a serious cause. The following case discussion highlights the factors that differentiate hyperventilation "cause" from "effect".

Case report

A 26 year old woman presented to the A&E department of a district general hospital at 1402 with a history of sudden onset "shortness of breath, fast heart beat and intermittent dizzy

spells to the point of near collapse" lasting 30 minutes. Her symptoms had almost resolved at the time of arrival in the A&E department. She had a similar attack in the past and was admitted to a medical ward of the same hospital eight months previously, from where she took her own discharge against medical advice within 24 hours of admission. She had undergone outpatient investigations including a 24 hour cardiac tape recording, which was reportedly normal. Past medical history revealed that she had undergone surgery for varicose veins at age 16 years. She was taking oral contraceptives and smoked 10 cigarettes per day. On examination, she had a pulse rate of 125 beats/min and a blood pressure of 140/90 mm Hg. Notably her oxygen saturation on air was recorded as 88% in the ambulance before arrival, but subsequently was normal on 10 l/min of oxygen and remained normal even after the oxygen was discontinued. Examination of the respiratory and cardiovascular system was unremarkable. An electrocardiogram (ECG) was performed, but not commented upon. It was subsequently reported as showing a right ventricular strain pattern with S1Q3T3 and was considered to be abnormal (fig 1). Arterial blood gas analysis and chest radiography were not performed. After observing the patient for 30 minutes, a reassessment of her condition revealed a pulse rate of 90 beats/min, blood pressure of 140/90 mm Hg and no hypoxia on air. She was discharged with a presumptive diagnosis of "anxiety related hyperventilation" and asked to see her general practitioner if her symptoms recurred. Two days later she was admitted to hospital after another episode of acute shortness of breath. On this occasion she was unwell, tachycardic, hypotensive and hypoxic. ECG was unchanged. Arterial blood gas analysis showed a P_{O_2} of 8.0 kPa on 40% oxygen. A Krypton ventilation perfusion lung scan revealed multiple defects on the perfusion scan (fig 2) with a high

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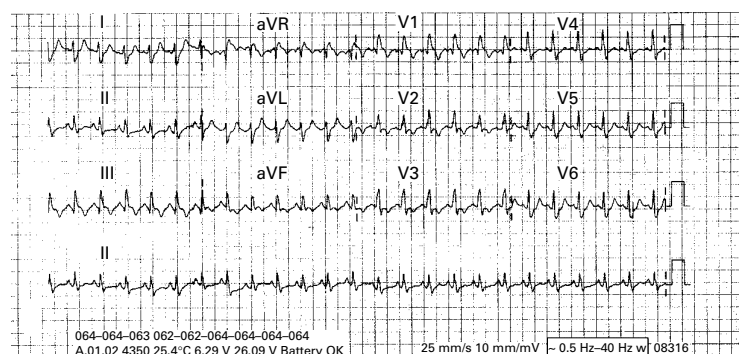


Figure 1 ECG showing right ventricular strain with S1Q3T3.

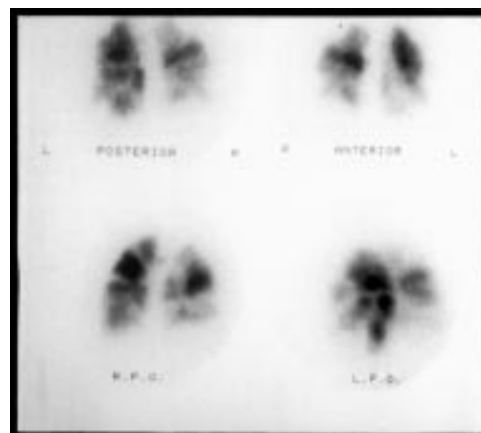


Figure 2 V/Q scan showing multiple perfusion defects.

probability of pulmonary embolus. She was given heparin and responded well. She was discharged after nine days and prescribed warfarin.

Discussion

A young woman presenting with shortness of breath, fast pulse and respiratory rate and dizziness is quite common in emergency medicine. In many cases anxiety and panic are the cause of this shortness of breath, but sometimes it may be a symptom of a more serious underlying condition like pulmonary embolus. It is important for emergency physicians to exclude such pathology before labelling shortness of breath as anxiety related. This can be done by close attention to history and examination, pre-existing risk factors and the use of some basic investigations attainable in the A&E department.

Regarding the clinical history, too little was made of her having shortness of breath to the point of near collapse. A detailed history would have highlighted this.

Examination revealed a tachycardia, tachypnoea and a documented period of hypoxia. This should have indicated the likelihood of a serious underlying cause. No reassurance should have been gained by the fact that her physiology was seen to normalise.

Risk factors for thromboembolic disease include major abdominal/pelvic surgery, immobilisation, recent lower limb trauma, pregnancy or post-partum, major medical illness and previous proven deep vein thrombosis or pulmonary embolus. Her risk factors for thromboembolism were minor, but did include smoking, oral contraceptives and previous lower limb varicose vein surgery (10 years ago).

Any abnormality in ECG and chest radiograph will suggest a serious underlying cause for "hyperventilation". Such patients should be referred for urgent investigations to exclude pulmonary embolism. Her ECG was abnormal and that in itself should have dictated a referral.

The main discriminating factors in this case were dizziness to the point of near collapse, shortness of breath, respiratory rate > 20 breaths/min, hypoxia recorded by pulse oximetry in the ambulance and an abnormal ECG.

A district general hospital with a catchment population of 200 000 may expect to diagnose 50 cases of pulmonary embolism annually.¹ As some of these only become apparent at

necropsy, the true incidence of pulmonary embolism is probably much higher at 1% of all admissions.² The incidence in A&E attendances is unknown, but is likely to be only a small proportion of young patients presenting with hyperventilation. Risk factors are important and 80–90% of patients with pulmonary embolus have predisposing factors. The clinical patterns of pulmonary embolus include sudden collapse (faintness and/or hypotension), pulmonary haemorrhage syndrome (pleuritic chest pain and/or hemoptysis) and isolated dyspnoea. This last category of patients presenting with shortness of breath without chest pain contributes up to 25% of all those diagnosed with pulmonary embolus.³ Most patients (>97%) with pulmonary embolus are short of breath with a respiratory rate > 20 breaths/min. ECG, arterial blood gas analysis and chest radiography are the basic investigations for diagnosing pulmonary embolus. D-dimer assays can be used to exclude the presence of thromboembolic disease.⁴ Newer D-dimer assays show promise as a tool for use in the A&E department. The diagnosis of pulmonary embolus can be confirmed by lung imaging using ventilation/perfusion isotope scanning, pulmonary angiography or spiral computed tomography. The guidelines for initial assessment and action are outlined by the British Thoracic Society, Standards of Care Committee Report.³ (At present the British Thoracic Society guidelines do not mention the use of D-dimer assays).

The lesson that should be learnt from our case is that pulmonary embolism can present without chest pain and consequently a symptom of shortness of breath should always be taken seriously, particularly if the features previously mentioned are present.

Contributors

Tapan Mehta contributed in data collection, literature search, illustrations and writing of the paper. John Sutherland contributed in literature search and writing of the paper. David Hodgkinson edited the paper and is also the guarantor.

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Conflicts of interest: none.

- 1 Anderson FA, Wheeler HB, Goldberg RJ, *et al.* A population-based perspective of the hospital incidence and case-fatality rates of deep vein thrombosis and pulmonary embolism. *Archives of Intensive Medicine* 1991;15:933–8.
- 2 Stein PD, Henry JW. Prevalence of acute pulmonary embolism among patients in a general hospital and at autopsy. *Chest* 1995;108:978–81.
- 3 British Thoracic Society Working Party. Suspected acute pulmonary embolism: a practical approach. *Thorax* 1997; 52:S1–24.
- 4 Miller A. A fresh look at D-dimer in suspected pulmonary embolism. *Thorax* 1998;53:821–2.

Combined brachial plexus and vascular injury in the absence of bony injury

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Abstract

Neurovascular injury to the axillary vessels is well described in association with fracture or dislocation involving the shoulder joint or the humerus. Such injury however can also occur in the absence of bony injury. A case is presented of damage to the axillary artery and brachial plexus following blunt trauma. This case demonstrates that complex neurovascular damage can occur in the absence of fracture or dislocation. The importance of a thorough clinical assessment is highlighted and priorities with regard to diagnosis and management are discussed.

(*J Accid Emerg Med* 2000;17:378–379)

Keywords: vascular injury; axilla; brachial plexus

Case report

A 75 year old woman was admitted to the accident and emergency (A&E) department after a history of a fall on the outstretched right hand. This had resulted from her being “blown over” by a strong gust of wind. On arrival to A&E she complained of pain in the right shoulder. Clinical examination revealed swelling and tenderness in the area of the right axilla and shoulder. On further examination her radial pulse was present but was noted to be somewhat weaker than that on the left side. The right arm was also felt to be colder than the left. Neurological examination revealed a complete motor and sensory deficit affecting the right arm. Radiography was urgently carried out which showed no evidence of fracture or dislocation to the shoulder or humerus.

The swelling in the axilla continued to increase and the lady became hypotensive. Despite the presence of a radial pulse a clinical diagnosis of a vascular injury to the axillary artery was made in addition to a presumed injury to the brachial plexus. After successful resuscitation by volume replacement angiography was carried out, which showed leaking of contrast material from a branch of the axillary artery (fig 1).

At formal exploration in the operating theatre a side branch of the axillary artery was found to be avulsed from the axillary artery itself. Haemostasis was readily secured. No evidence of injury to the exposed parts of the brachial plexus was apparent at operation. Postoperatively the neurological deficit persisted. Nerve conduction studies were carried out that confirmed avulsion of the roots of the brachial plexus that was not amenable to surgical treatment.

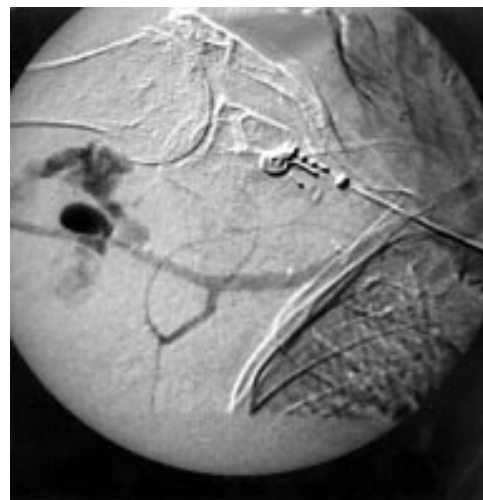


Figure 1 Angiogram showing contrast material leaking from the axillary artery.

Discussion

Damage to the axillary artery after blunt injury usually occurs as a consequence of severe trauma. Arterial injury is well described in association with fracture of the upper humerus^{1 2} or dislocation of the shoulder.^{3 4} Our case shows that arterial injury in the axilla can occur with low impact as well as high impact injuries and that severe vascular and neurological injury can occur in the absence of fracture or dislocation. This case also confirms the close association between arterial injury to the axillary artery and damage to the brachial plexus.

BRACHIAL PLEXUS INJURY

Neurological injury to the brachial plexus is usually caused by severe traction to the nerve roots that occurs at the time of injury. As in our case these injuries are most often not amenable to surgical treatment. Volume loss into the axilla as a result of associated arterial injury can be considerable in these cases and was sufficient to cause hypotension in the case described. The tamponade effect of the haematoma being contained in the axilla delays exsanguination. We have identified only one case in the medical literature where damage to the axillary artery itself was the cause of death in the absence of associated injury.⁵ The high axillary pressures caused by the tamponade effect can, however, cause a compression neuropathy to the brachial plexus. Prompt evacuation of the haematoma may significantly reduce the subsequent neurological deficit in such cases. The long term outcome of neurovascular injury to the axillary structures is not dependent on the vascular injury, which can

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usually be successfully managed, but upon the recognition, treatment, and outcome of the associated nerve injuries.⁶

Conclusion

This case highlights the importance of performing a full, detailed neurovascular examination in all patients with an upper limb injury. This case also confirms that a significant vascular injury can be present even when the distal pulse is present and that vascular injury can occur in the elderly with lesser degrees of force because of atherosclerotic rigidity of the artery. In cases where a vascular injury is suspected and the patient is stable angiography should be rapidly carried out to delineate the anatomy of the injury allowing surgical access to be planned. Although a coordinated vascular and orthopaedic approach and prompt surgical treatment will optimise post-injury upper limb function the long term results of these complex

injuries are usually determined by the presence and severity of associated nerve injuries.

Contributors

Ahmed Ismail contributed the original idea for this paper and contributed to the writing of the paper. Aidan MacNamara was responsible for reviewing the literature and writing the paper. The guarantor of the paper is Aidan MacNamara. Funding: none.

Conflicts of interest: none.

- 1 Byrd RG, Byrd RP, Roy TM. Axillary artery injuries after proximal fracture of the humerus. *Am J Emerg Med* 1998;16:154-6.
- 2 Hayes JM, VanWinkle G. Axillary artery injury with minimally displaced fracture of the neck of humerus. *J Trauma* 1983;23:431-3.
- 3 Adovasio R, Visintin E, Sgarbi G. Arterial injury of the axilla: an unusual case after blunt trauma of the shoulder. *J Trauma* 1996;41:754-6.
- 4 Gates J, Knox J.B. Axillary artery injuries secondary to anterior dislocation of the shoulder. *J Trauma* 1995;39:581-3.
- 5 Lodding P, Angeras U. Fatal axillary artery injury following anterior dislocation of the shoulder. *Ann Chir Gynaecol* 1998;77:125-7.
- 6 Shaw A, Milne A, Christie J, et al. Vascular trauma of the upper limb and associated nerve injuries. *Injury* 1995;26:515-18.

Acute cerebrovascular accident after minor trauma in a 1 year old

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Abstract

Acute cerebrovascular accident in an otherwise well child is a rare presentation. A case is described where the diagnosis was delayed because of association with minor trauma and a misleading diagnosis of soft tissue injury.

(*J Accid Emerg Med* 2000;17:379-380)

Keywords: cerebrovascular accident; minor trauma

Case report

A 1 year old girl presented to the accident and emergency (A&E) department one hour after a witnessed fall from a sofa. She landed on her left side hitting her head, and afterwards was reluctant to use her left arm and leg. She cried immediately after the fall, had sustained no loss of consciousness and had not vomited or slept since the incident. Her past medical history was unremarkable with a normal delivery and developmental milestones. There was no relevant family history.

On examination she was distressed, crying when moved. There was no obvious limb deformity. There was no spontaneous left arm movement but she could be encouraged to withdraw the limb. She was able to weight bear on both lower limbs but had minimal spontaneous movement of her left leg. She was fully alert with no cranial nerve abnormality detected. After analgesia she settled and full passive movement was achieved in both hips. Radiographs of left shoulder, elbow and wrist



Figure 1 The area of low attenuation (standard orientation) is demonstrated.

were normal. She was discharged home that night to be reviewed the following day at which time there was no change in her condition. On second review her mother described her to be continuously irritable, she had deteriorated in that she was now unable to weight bear, had decreased tone on the left side, flaccid reflexes, an upgoing left plantar and facial asymmetry on crying.

Computed tomography of the head was performed under sedation and showed a low attenuation area in the posterior aspect of the

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internal capsule (see fig 1). A diagnosis of probable cerebral infarct was made.

She was transferred to Great Ormond Street Hospital for follow up. No cause was found after extensive investigation. She was given aspirin 5 mg/kg/day.

Discussion

Cerebral infarction is uncommon in children (incidence 0.63/100 000/y,¹ 0.58/100 000/y²). The underlying condition remains unknown in as many as half of cases.³ The cause in a sick child may be obvious, for example, severe dehydration in a diarrhoeal illness, diabetic ketoacidosis, severe sepsis or sickle cell disease. Systemic hypertension does not play a major part in children. Higgins² demonstrated aetiology as 21% infection, 18% vascular disorders, 15% haematological disorders, 13% cardiac disorders (a large majority being cyanotic heart disease in children less than 2 years), trauma 8%. Clotting disorders have been further investigated and Baca⁴ demonstrated 7 of 10 cases of infarction to have anticardiolipin antibodies and 2 of the 10 to have a temporary protein C deficiency. This may explain a large proportion of the previously labelled idiopathic cases. Protein S deficiency can be associated with nephrotic syndrome and leukaemia. Other aetiologies includes trauma, for example internal carotid artery damage via the roof of the mouth with a lollipop stick, vasculitides and rare disorders such as Moyamoya.

The major presenting feature (after excluding those related to premature birth, birth trauma and head injury) is acute hemiplegia, which may be associated with seizures and altered consciousness. It is important to obtain any history of trauma, recent infection (55% in a study by Eeg-Olofsson⁵), family history of thrombotic disease or sickle cell disease as it may be the first presentation in a young child.

Extensive investigation should be carried out at a specialist centre to identify any treatable cause. Survival is in the region of 80–90%.^{1–5} Lanska⁶ showed residual disability in 50%, however all were ambulatory, 19% had recurrent seizures. The risk of recurrence is dependent on the cause. Baca's study had no recurrent episodes after 15.7 months while taking aspirin.

Aspirin is used for its anti-platelet action and as in juvenile arthritis is well tolerated. Carers need to be alert for symptoms of salicylism and the serious complication of Reyes syndrome. There is no mention in the literature about the use of dipyridamole specifically for neurological situations.

Conclusion

Our case demonstrates the following points:

- A diagnosis of soft tissue injury should not have been made in a child with upper and lower limb weakness after a fall.
- A history of trauma is important to obtain but in this case may have been misleading.
- Children may need daily review and paediatric advice should be sought if there is no clear diagnosis at presentation.

Contributors

Wendy Mathews performed the literature search and cowrote the case study. Ramzi Freij checked the literature search and cowrote the case study. Kambiz Hashemi edited the paper and discussed the core ideas.

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Conflicts of interest: none.

- 1 Schoenberg BS, Mellinger JF, Schoenberg DG. Cerebrovascular disease in infants and children: A study of incidence, clinical features, and survival. *Neurology* 1978;**28**:763–8.
- 2 Higgins JJ. *Neuropediatrics* 1991;**22**:190–3.
- 3 Trescher WH. Ischaemic stroke syndromes in childhood. *Pediatr Ann* 1992;**21**:374–83.
- 4 Baca V. Cerebral infarction and antiphospholipid syndrome in children. *J Rheumatol* 1996;**23**:1428–31.
- 5 Eeg-Olofsson O. Stroke in children. Clinical characteristics and prognosis. *Acta Paediatr Scand* 1983;**72**:391–5.
- 6 Lanska MJ. Presentation, clinical course and outcome of childhood stroke. *Paediatr Neurol* 1991;**7**:333–41.

Trauma induced testicular torsion: a reminder for the unwary

Yeap Joo Seng, Kevin Moissinac

Abstract

Trauma induced testicular torsion is a well recognised entity, the incidence being 4–8% in most studies reporting on testicular torsion. The signs and symptoms of testicular torsion may easily be mistakenly attributed to preceding testicular trauma if there was such an event. A patient is described with trauma induced testicular torsion who presented on three occasions before a decision was made to perform scrotal exploration. Unfortunately, an orchidectomy was the outcome. The message that trauma can and not infrequently does precipitate torsion, needs to be reiterated. Awareness of the entity and constant vigilance is required of clinicians to avoid a delay in definitive treatment.

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Keywords: testis; torsion

The incidence of testicular torsion has been rising^{1–3} but this has been mirrored by a rise in the testicular salvage rate.^{1–3} Barker and Raper in 1964 reviewed the literature and found the immediate testicular salvage rate to be 10% and reported a salvage rate of 29% in their own series.⁴ Reports in the 1970s and 1980s, taking delayed testicular atrophy into account, reported testicular salvage rates from 42–79%.^{1–3 5–8} This improvement was attributed to an increasing awareness of the condition among the general practitioners and hospital doctors, leading to an increased and an earlier diagnosis.^{3 7} A willingness to explore immediately where there is doubt has also contributed to the improved salvage rate.⁶ In the 1990s, Daehlin *et al*⁹ reported a testicular salvage rate of 56% after a 4–10.5 year follow up.

Trauma induced testicular torsion is now a well recognised entity, the incidence being 4–8% of testicular torsion in most series^{1 3 10 11} with the highest reported incidence being 12%.¹² The reports that have commented on the testicular salvage rate in trauma induced testicular torsion are largely in the form of case reports and contain small numbers of patients. The testicular salvage rate reported in trauma induced testicular torsion has been variable (0%,^{10 13 14} 33%,¹⁵ 80%¹¹ and 100%¹⁶). The scrotal and testicular symptoms and signs of trauma induced testicular torsion may be mistakenly attributed to the trauma itself. A case of trauma induced testicular torsion, initially diagnosed as a traumatic scrotal haematoma, is

reported to serve as a reminder and to promote an awareness of the entity.

Case report

Somersaulting off a springboard, the perineum and the scrotum of a 14 year old boy were the parts of the body to first hit the water in the swimming pool. He presented for medical attention a few hours later when the pain did not resolve. At the medical examination, a tender right scrotal swelling was noted and a diagnosis of a traumatic scrotal haematoma was made and he was discharged with analgesics.

He presented for medical attention two days later and was again discharged with a similar diagnosis. He presented a third time, five days after his initial injury because of worsening of his pain and swelling. On physical examination, the scrotum was markedly swollen and indurated, with the scrotal skin taut. A diagnosis of traumatic scrotal haematoma was made by the duty surgeon, and a decision was made to explore the scrotum to evacuate the haematoma for the relief of the symptoms.

At scrotal exploration, the right testicle was found to be gangrenous secondary to torsion of the spermatic cord. A moderate amount of scrotal haematoma was also present and this was evacuated. A right orchidectomy was performed. Orchidopexy of the left testis was performed. Postoperative recovery was uneventful and he was discharged on the second postoperative day.

Discussion

The most important risk factor for testicular torsion is an anatomical predisposition, the commonest being a high or a complete investment of the testicle and the spermatic cord (bell clapper deformity) by the tunica vaginalis.^{3 4 17 18} The cremasteric muscle surrounds the spermatic cord in a spiral manner and contraction of this muscle has a rotational effect on the testicle. A strong contraction of this muscle can therefore rotate a predisposed, freely mobile testicle, which may go on to undergo torsion.^{11 19}

The combination of reports on trauma induced testicular torsion^{10 11 13–16} with this case report gives an overall testicular salvage rate of 40% (6 of 15) in trauma induced testicular torsion in the medical literature. It may be very difficult at times to distinguish clinically, torsion and an acute injury. These patients must be referred immediately to a urologist or a general surgeon where an assessment can be made and further investigations or immediate surgical exploration performed as necessary. Where there is a high clinical suspicion of

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testicular torsion, further investigations are unnecessary and immediate surgical exploration is warranted.²³ However, where physical examination is equivocal, colour Doppler ultrasonography or scintigraphy may be helpful in establishing the diagnosis.²³⁻²⁵

The immediate testicular survival rate and the subsequent testicular atrophy and impairment in testicular function are directly related to the duration and the degree of the torsion.^{1 3 6-9 18 20 21} Only 4% of testis are found to be non-viable in acute torsion of less than 12 hours duration compared with a 75% infarction rate if the history was greater than 12 hours.³ Time therefore is of the greatest essence if the testis is to be saved. Recent reports suggest that most delays to testicular exploration occur because of late presentation to the hospital.^{5 10 22} In this reported case, there was not only a delay in presentation but the patient was also then discharged on two occasions when the diagnosis of traumatic scrotal haematoma was assumed because of the history of trauma.

We feel that a clear message that trauma can precipitate torsion needs to be reiterated. A high index of suspicion is required of the examining clinician. It is all too easy to attribute the testicular pain, swelling and induration as secondary to testicular trauma. Clinicians should always attempt to think and look beyond the obvious.

Contributors

Yeap Joo Seng performed the literature review, discussed and outlined the core aspects of the case report and participated in the writing of the paper. Kevin Moissinac was the duty surgeon who performed the surgical exploration, initiated the case report, participated in the literature review and the writing of the paper. Kevin Moissinac will act as guarantor.

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- 1 Williamson RCN. Torsion of the testis and allied conditions. *Br J Surg* 1976;**63**:465-76.

- 2 Coggon D, Nelms M. Incidence of testicular abnormalities. *Lancet* 1984;ii:747.
- 3 Anderson JB, Williamson RCN. Testicular torsion in Bristol: a 25-year review. *Br J Surg* 1988;**75**:988-92.
- 4 Barker K, Raper FP. Torsion of the testis. *Br J Urol* 1964;**36**:35-41.
- 5 Chapman RH, Walton AJ. Torsion of the testis and its appendages. *BMJ* 1972;1:164-6.
- 6 Cass AS, Cass BP, Veeraraghavan K. Immediate exploration of the unilateral acute scrotum in young male subjects. *J Urol* 1980;**124**:829-32.
- 7 Cattolica EV, Karol JB, Rankin KN, et al. High testicular salvage rate in torsion of the spermatic cord. *J Urol* 1982;**128**:66-8.
- 8 Jones DJ, Macreadie D, Morgans BT. Testicular torsion in the armed services: twelve year review of 179 cases. *Br J Surg* 1986;**73**:624-6.
- 9 Daehlin L, Ulstein M, Thorsen T, et al. Follow-up after torsion of the spermatic cord. *Scand J Urol Nephrol Suppl* 1996;**179**:139-42.
- 10 King LM, Sekaran SK, Sauer D, et al. Untwisting in delayed treatment of torsion of the spermatic cord. *J Urol* 1974;**112**:217-21.
- 11 Elshahry S, Praniokoff K, Magoss IV, et al. Traumatic torsion of the testis. *J Urol* 1984;**132**:1155-6.
- 12 Leape LL. Torsion of the testis: Invitation to error. *JAMA* 1967;**200**:669.
- 13 Kursh ED. Traumatic torsion of the testicle. *Urology* 1981;**17**:441.
- 14 Manson AL. Traumatic testicular torsion: case report. *J Trauma* 1989;**29**:407-8.
- 15 Cos LR, Rabinowitz R. Trauma-induced testicular torsion in children. *J Trauma* 1982;**22**:244-6.
- 16 Rogus BJ. Polyorchidism and testicular torsion. *Urology* 1988;**31**:137.
- 17 Parker RM, Robison JR. Anatomy and diagnosis of torsion of the testicle. *J Urol* 1971;**106**:243-7.
- 18 Udeh FN. Testicular torsion: Nigerian experience. *J Urol* 1985;**134**:482-4.
- 19 Muschat M. The pathologic anatomy of testicular torsion. *Surg Gynecol Obstet* 1932;**54**:758.
- 20 Skoglund RW, McRoberts JW, Ragde H. Torsion of the spermatic cord: a review of the literature and an analysis of 70 new cases. *J Urol* 1970;**104**:604-7.
- 21 Anderson JB, Williamson RCN. The fate of the human testes following unilateral torsion of the spermatic cord. *Br J Urol* 1986;**58**:698-704.
- 22 Rampaul MS, Hosking SW. Testicular torsion: most delays occur outside hospital. *Ann R Coll Surg Engl* 1998;**80**:169-72.
- 23 Siegel MJ. The acute scrotum. *Radiol Clin North Am* 1997;**35**:959-76.
- 24 Süzer O, Özcan H, Küpeli S, et al. Color Doppler imaging in the diagnosis of the acute scrotum. *Eur Urol* 1997;**32**:457-61.
- 25 Hendriksx AJM, Linh Dang C, et al. B-mode and colour-flow duplex ultrasonography: a useful adjunct in diagnosing scrotal diseases? *Br J Urol* 1997;**79**:58-65.