CASE REPORTS

Transorbital brain injuries

P Cackett, J Stebbing

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A 20 year old man attended the accident and emergency department after an assault with a short bladed knife. He had sustained a stab wound to the right orbit. There was no other significant history. On examination he was alert and orientated with a Glasgow coma scale of 15. Snellen visual acuities were no perception of light right eye and 20/20 left eye. Inspection of the right orbit revealed a right upper lid laceration and an underlying penetrating eye injury. There was no other neurological deficit. Plain orbital radiographs were obtained but did not show any defects. However, in view of the nature of the injury and the fact that the posterior extent of the orbital wound could not be visualised, a computed tomogram of the head was obtained. This showed pronounced pneumocephalus (fig 1). A fracture of the posterior wall of the frontal sinus was noted with a fracture line through the ethmoidal labyrinth extending to the medial wall of the left orbit. A fragment of bone was seen to abut the left medial rectus muscle (fig 2). The patient subsequently underwent anterior cranial fossa repair by the neurosurgeons and repair of the penetrating eye injury by the ophthalmologists. He made an uneventful recovery after surgery. There was, however, no improvement in the visual acuity of the right eye as a result of total retinal detachment with proliferative vitreoretinopathy.

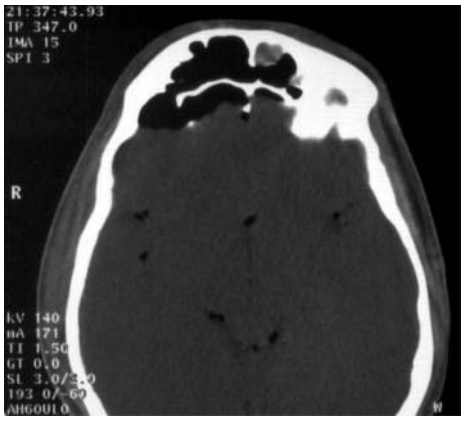

Figure 1 Computed tomogram of the head showing pneumocephalus with fracture of posterior wall of frontal sinus.

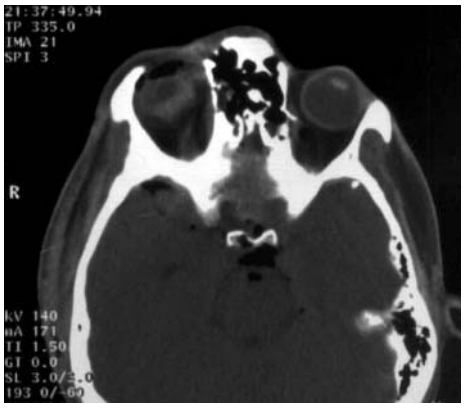

Figure 2 Computed tomogram of the head showing fracture line through ethmoidal labyrinth extending to medial wall of left orbit with fragment of bone abutting left medial rectus. The right penetrating eye injury can also be seen.

DISCUSSION

The orbit is pyramidal in shape with a quadrangular base situated at the orbital margin. The walls of the orbit are thin and may be penetrated by objects moving at some velocity directed at right angles to the wall. The roof of the orbit that is made up of the frontal bone and the lesser wing of the sphenoid is very thin and is therefore at particular risk of injury, especially in children who fall on sharp objects carried in the hand.[1 2] Penetration of the orbital walls may result in damage to the paranasal sinuses, which may give rise to emphysema of the orbit, cerebrospinal fluid fistulas, orbital cellulitis, meningitis, cerebral abscess, or pneumocephalus. The intracranial complications of transorbital stab wounds include ventricular damage, carotid-cavernous sinus fistula, pneumocephalus and subdural, subarachnoid, intraventricular, and intracerebral haemorrhage.

Orbital radiographs may be negative and are therefore unreliable in ruling out intracranial involvement, as in our case.[2 3] Furthermore, initially there may be no apparent neurological deficit on examination, and therefore a lack of neurological signs does not exclude a secondary brain injury.[3] In summary, orbital stab wounds may mask serious underlying intracranial injuries and therefore we would recommend that computed tomography is performed in cases where there is any suspicion of a secondary transorbital brain injury.

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Intermittent left bundle branch block (LBBB) as a clinical manifestation of myocardial contusion after blunt chest trauma

V R P Pizzo, I Beer, R de Cleva, B Zilberstein

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Injury to the heart in blunt chest trauma is dependent on a number of factors. Symptoms are often non-specific, and there is no gold standard test for diagnosis. Injuries to small areas of the myocardium may only be identified at autopsy. We report a 38 year old man who sustained a number of injuries in a road traffic accident, and in whom the single clinical or ECG abnormality was a left bundle branch block (LBBB); he had a myocardial injury rated as grade II. The patient was treated for his injuries and later discharged. As this is a difficult diagnosis, algorithms of blunt chest trauma may save time and money by avoiding misleading diagnosis and unnecessary monitoring and admissions.

Injury to the heart in blunt chest trauma is dependent on the magnitude of force applied to the chest, the area over which it is applied, the compliance of the chest wall, and the timing of the application of force during the cardiac cycle. Symptoms are often non-specific and physical signs frequently absent. An initial abnormal ECG may help to identify patients with blunt cardiac trauma, but normal studies lack negative predictive value. Cardiac isoenzymes alone are also not helpful. The role of troponin levels is not yet clear. Echocardiography is useful in clinically severe blunt cardiac injury.

Recently, a retrospective review has showed that the majority of patients identified as having "myocardial contusion" (blunt cardiac trauma) sustained their injuries as a result of a motor vehicle crash, and that the most frequent additional injuries were rib fractures, pulmonary contusion, and closed head injuries.[1]

Injuries to anatomically small areas of the myocardium may be identifiable only at autopsy, and often have little clinical expression or impact on contractility, but they become evident if they alter the electrical properties of the heart (conduction abnormalities and arrhythmias). A transient right bundle branch block was the most common conduction abnormality in that review, as has been noted previously, probably because of its anterior anatomical location and proximity to the sternum.

Mortality was found to be associated only with severe injuries (grade IV or greater of the Organ Injury Scale of the American Association for the Surgery of Trauma (ATLS)).[2]

We describe a myocardial contusion case where the single clinical or ECG abnormality was a left bundle branch block (LBBB), rarely reported in the literature.

CASE REPORT

A 38 year old white man was admitted to our emergency room following a motor vehicle crash. The initial evaluation, according to ATLS protocols, showed patent airways (cervical belt present), reduced left hemithorax expansibility and auscultation, and extensive subcutaneous emphysema, Heart rate was 96 beats/min and arterial pressure 120×80 mmHg. He was confused (Glasgow Coma Scale 14). As the patient was apparently drunk (alcoholic breath) he underwent an abdominal ultrasound evaluation (focused assessment with sonography for trauma (FAST) approach) that did not show free liquid pattern. The chest radiography (fig 1) showed wide subcutaneous emphysema, pneumothorax, and alveolar opacities in inferior left hemithorax associated with fractures of the second to fifth ribs at different points (flail chest).

The patient underwent closed system chest drainage without significant haemothorax and adequate lung expansion. The complementary neurological evaluation, including head and cervical CT scan, did not show abnormalities. The chest CT scan showed non-significant bilateral pleural effusion, greater on the left, with opacities on inferior lobes, multiple rib fractures, left scapula fracture, subcutaneous and soft tissue emphysema, little pneumomediastinum, patent trachea and main bronchi, and no heart or base vessel abnormalities.

The patient remained hemodynamically stable during the whole evaluation time without abnormalities on continuous cardiac monitoring. He was transferred to the surgical intensive care unit (ICU) with a diagnosis of blunt chest trauma and severe pulmonary contusion. He was awake, reporting dyspnoea (28 inspirations/min) while on supplemental oxygen delivered by face mask (5 l/min), and presenting paradoxical breathing (left hemithorax); his mean arterial pressure was 100 mmHg, heart rate was 110 beats/min and he had a normal 12 derivation ECG (fig 2). He

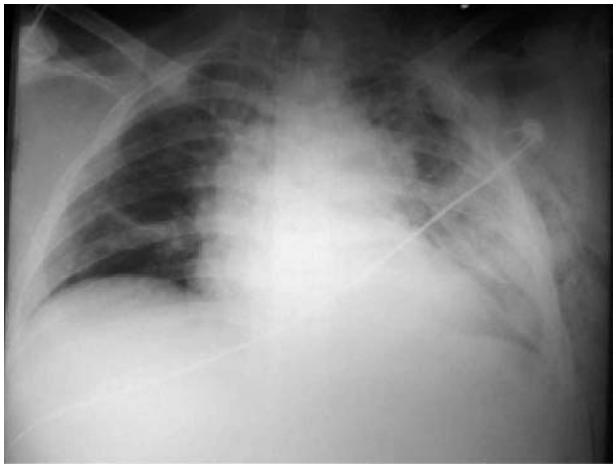

Figure 1 ICU admission chest *x* ray.

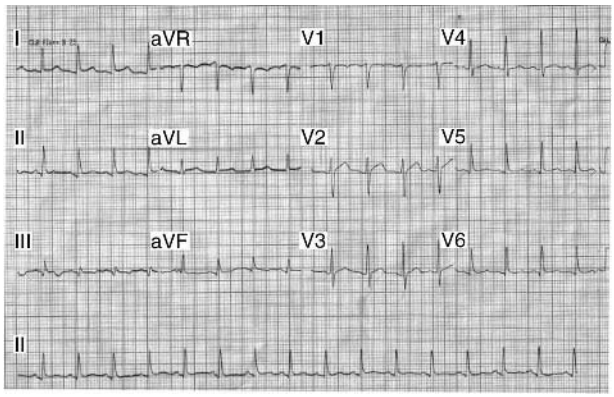

Figure 2 ICU Admission ECG.

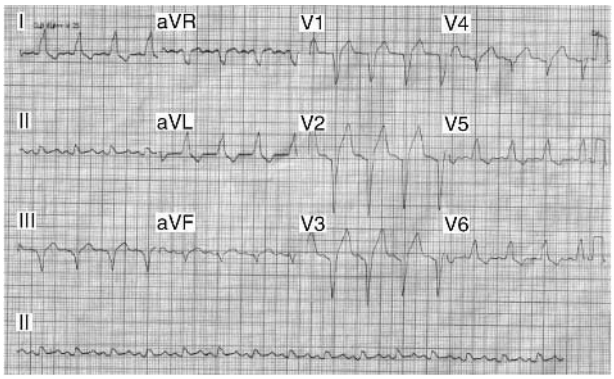

Figure 3 ECG after 18 hours showing left bundle branch block (LBBB).

received 4 litres of crystalloid fluids during the resuscitation period in the emergency room.

Once stable, the patient received analgesics and a respiratory therapy programme, including intermittent non-invasive ventilation (bi-level positive airway pressure). Eighteen hours after his ICU admission, he developed a widening of the QRS complexes on cardioscope, without symptoms, and ECG showed sinus rhythm (88 beats/min) with left bundle branch block (LBBB) pattern (fig 3) with spontaneous reversion after almost 40 minutes. Blood was taken for measurement of troponin I after this event, and gave a result of 1.8 ng/mL (normal <2.0 ng/mL). Obviously, his creatinine phosphokinase was extremely elevated from the accompanying skeletal muscle injuries. According to the Organ Injury Scale, the patient had a grade II myocardial injury.

The patient did not have a past medical history of hypertension or ischaemic heart disease, nor of smoking or dyslipidaemia. On the second and fifth in hospital days, a new LBBB pattern with variable frequencies, without hypotension or any symptoms, was observed. Transthoracic echocardiography demonstrated discinetic motion of the septal wall (without LBBB pattern on cardioscope), minor pericardial effusion, no valvular incompetence, and preserved ventricular function.

During his clinical course, the patient's respiratory status worsened, with a sudden fall in thoracic drainage output and a large pleural effusion in the left haemothorax on chest radiograph. New chest drainage was performed but it was not fully effective; a new chest CT scan showed loculated pleural effusion, and thoracoscopy was used to guide the drainage and washing of debris from the pleural space, with satisfactory radiological and clinical improvement.

Although the patient did not develop new intermittent LBBB pattern episodes, he underwent a new echocardiography that confirmed the discinetic septal motion. He was discharged of from ICU to an intermediate care facility with the chest drain, and after 1 week of respiratory therapy care and pulmonary re-expansion techniques (including incentive spirometry), he left the hospital.

DISCUSSION

Regardless of the definition of BCT, this entity becomes important only when it is associated with significant symptoms, such as arrhythmias or hypotension, or causes anatomical defects, such as valvular, septal, or free wall rupture. Electrocardiographic abnormalities have been reported to be the most sensitive screening technique for myocardial contusion. However, some studies have contradicted this, because the right ventricle most commonly sustains injury due to its anterior location beneath the sternum, while the left ventricle has large mass and generates larger voltage potentials.

In a study by Torres-Mirabal *et al*[3] in 36 patients with myocardial contusion, 58 ECG abnormalities were described. None was an LBBB. Bertinchant *et al*[4] have published ECG findings in 94 patients with suspected myocardial contusion: nine cases with right bundle branch block, but no LBBB cases reported. Cachecho *et al*[5] have reported the clinical significance of myocardial contusion in 336 victims of blunt chest trauma, with 19 bundle branch blocks but no obvious LBBB citations.

As LBBB may be also a manifestation of ischaemic heart disease, to differentiate a diagnosis of acute coronary syndrome from a myocardial contusion in a patient such as a 50 year old man, with several risk factors for coronary artery disease, a victim of BCT, could be difficult. Assessment of ventricular function, continuous cardiac monitoring, and determination of specific cardiac enzymes are potentially useful.

We report a rare myocardial contusion manifestation in a young trauma victim. As no one single test has been considered the ''gold standard'', algorithms of blunt chest trauma may save time and money by avoiding misleading diagnosis and unnecessary monitoring and admissions.

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Hepatic artery aneurysm: an unusual cause for gastrointestinal haemorrhage

H S Narula, A Kotru, A Nejim

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Aneurysms of the hepatic artery are rare. The presenting features include abdominal pain, which may be associated with a mass. More acutely, patients present with signs of hypovolaemia secondary to rupture. The patient reported here presented with gastrointestinal haemorrhage of unknown aetiology. A computed tomography scan showed an aneurysm of a visceral artery. Subsequent angiography confirmed the presence of a leak in the hepatic artery. The patient refused surgical intervention and improved with supportive treatment only.

Hepatic artery aneurysms are rare lesions presenting as rupture, a mass, or with pain. We report an unusual case of a patient with hepatic artery aneurysm who presented with gastrointestinal haemorrhage.

CASE REPORT

An 85 year old woman presented with acute gastrointestinal haemorrhage. She had a two month history of abdominal pain and a five day history of rectal bleeding, weakness, and lethargy. There was no history of prior use of non-steroidal anti-inflammatory drugs. On arrival the patient was haemodynamically stable, but she had mild epigastric tenderness and fresh blood was seen on rectal examination.

Her initial blood and radiological investigations were normal. Upper gastrointestinal endoscopy and flexible sigmoidoscopy did not reveal any significant abnormality apart from transported blood in the colon. A computed tomography (CT) scan showed a 6 cm aneurysm (fig 1) in relation to one of the visceral arteries, which had two concentric rings of calcification. The artery of origin could not be identified and an angiogram was done. This confirmed the presence of an aneurysm in the hepatic artery. The pancreas was also highly calcified.

She was treated conservatively and the bleeding stopped spontaneously. The option of surgery was discussed, but this was declined by the patient and her relatives.

DISCUSSION

Hepatic artery aneurysms are rare lesions (20% of all visceral aneurysms[1]) and difficult to diagnose clinically. They can present as a dull ache, lump, obstructive jaundice, or rarely as bleeding into the gastrointestinal tract. The common causes include surrounding inflammation, trauma, or atherosclerosis.[2] Selective angiography of the coeliac axis and superior mesenteric artery are essential, not only to confirm the diagnosis, but also to supply important information about related vascular anatomy, which is invaluable while planning the operative strategy. Other investigative tools include ultrasound, which may show a turbulent arterial waveform[3] with high peak velocity, and three dimensional CT angiography.[4]

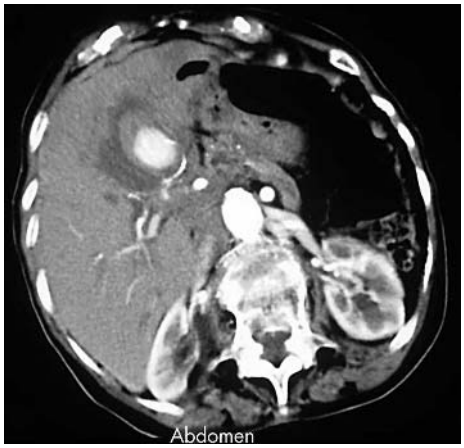

Figure 1 Computed tomography scan of an 85 year old woman showing an anuerysm in the hepatic artery (confirmed by angiography).

Management options range from reconstruction using prosthetic grafts to excision or embolisation.[5] Surgery is the treatment of choice for extrahepatic aneurysms, whereas radiological embolisation is more appropriate for intrahepatic aneurysms.[6] Some experts have suggested reserving embolisation for aneurysms which are difficult to operate upon due to poor accessibility.[7]

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An unusual case of a compound depressed skull fracture after an assault with a stiletto heel

G Stables, G Quigley, S Basu, R Pillay

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Compound depressed skull fractures occur commonly from assault with various sharp and blunt objects. The use of a stiletto heeled shoe as an offensive weapon has not been reported before as a cause of such an injury. However unusual the history of assault, a clear picture of the mechanism of the injury is vital. Patients with scalp lacerations following a direct blow to the head must be evaluated thoroughly. Early investigation and treatment are necessary to reduce the risk of complications, as illustrated by this case.

A 23 year old man presented to the accident and emergency department of a university hospital after an alleged assault. He had sustained a blow to the left side of his head with the stiletto heel of a shoe. On arrival he was fully conscious and the only apparent injury was a two cm wound in the scalp superior to the left ear. There was no history of loss of consciousness. The wound was cleaned and sutured and the patient discharged home with head injury instructions.

On returning home he became increasingly unwell with intermittent speech difficulties. He was taken to an A&E department of his local hospital 48 hours after the injury.

After initial assessment, a plain skull *x* ray revealed a minor depressed skull fracture of the left parietal bone. Computed tomography (CT) confirmed the presence of the fracture, together with underlying contusion of the parietal lobe. He was transferred to the regional neurosurgical unit for further assessment.

On arrival at the neurosurgical unit he had a mild expressive dysphasia and a temperature of 37.8℃. He was initially managed conservatively with intravenous antibiotics (certriaxone and metronidazole) which had been started in the A&E department. However, after three days his expressive dysphasia worsened. CT was repeated and now showed cerebral swelling and oedema extending down to the left temporal lobe, with areas of patchy contrast enhancement. The radiological appearances were consistent with cerebritis. At this stage, there was no obvious cerebral abscess and conservative treatment was continued, with the addition of flucloxacillin. Despite these measures, the patient deteriorated clinically and developed a left third cranial nerve palsy and a decreased level of consciousness. Further CT showed a large cerebral abscess beneath the fracture site (fig 1).

A craniectomy and debridement was performed and 20 ml of pus was aspirated. *Staphylococcus aureus* was isolated following culture of the pus. The patient improved clinically, and postoperative CT showed resolution of the abscess. He was discharged after completion of a 21 day course of intravenous antibiotics. The patient was well at follow up three months later, with a mild expressive dysphasia which had resolved at a 12 month follow up.

DISCUSSION

Open depressed skull fractures can occur in a variety of settings.[1] To our knowledge there has not been any previous report of a stiletto heel causing such an injury. This could in part reflect the relative strength of the particular heel involved in this case. We are informed that such heels are may be customised and reinforced when worn by members of the transsexual/transvestite community, of which the alleged assailant was a member.

Fractures of the skull vault are influenced by various factors, which include the thickness of the vault and the force of the impact. Rapid dynamic loading occurred in this case, with the force probably acting for a very short time (<200 ms). The size of impacting device (that is, the heel) and the force of impact are directly related to the magnitude of the dynamic load.[2] In this case, a large amount of kinetic energy made contact with the skull over a small area (~1 cm^2). The degree of local deformation in this case was enough to cause penetration and fracture of the skull.

The complications and sequelae of compound depressed fractures of the skull are minimised by early diagnosis and appropriate treatment.[3] Frequently, as in our case, there is no associated neurological injury, so depressed fractures may be missed because radiographic investigations are not done.

The Society of British Neurological Surgeons issued guidelines in 1998 for the indications for skull *x* ray following

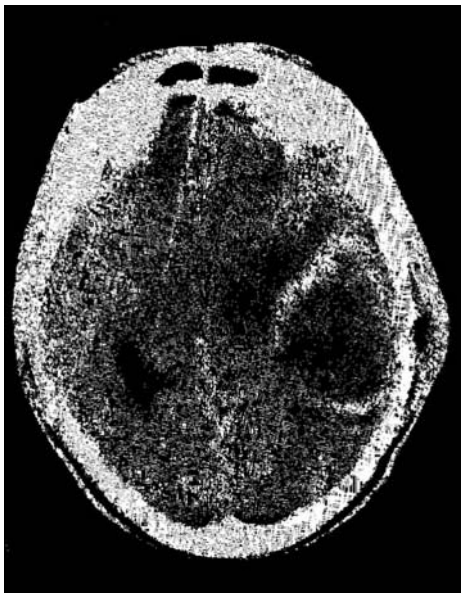

Figure 1 Cranial computed tomography showing a large cerebral abscess.

recent head trauma.[4] These include a history of loss of consciousness or amnesia, suspected penetrating injury, a scalp laceration >5 cm long, bruising or swelling, CSF leak from the nose or ear, and a violent mechanism of injury.

Even when a skull *x* ray is obtained, the depression is often not appreciated.[3] This case illustrates the need for careful examination. The scalp is relatively mobile and any area of depression may not lie directly beneath the laceration. Visual exploration of the skull through the scalp laceration may fail to reveal a fracture. Careful digital exploration of the scalp wound with a gloved finger can reveal a bone edge, a depression, or a mobile bone fragment.[5] Exploration under local anaesthesia and aseptic conditions using a finger sweep technique should be mandatory in such cases.

The management of choice in preventing infection from open depressed skull fractures is operative debridement and thorough irrigation,[6] though there is evidence that select cases can be safely managed without operation.[6 7]

The distinction between antibiotic treatment and prophylaxis is blurred, as contamination of the wound has already occurred at the time of injury.[8] The prevalence of infective complications (abscess, empyema, meningitis) after compound depressed skull fractures ranges from 4% to 10%.[9 10]

This case highlights the need for clearly determining the mechanism of injury and undertaking appropriate investigations. A high index of suspicion should be maintained if a pointed object is implicated in assaults involving the skull.

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An aortic dissection in a young weightlifter with non-Marfan fibrillinopathy

C J Hogan

Emerg Med J 2005;**22**:304–305. doi: 10.1136/emj.2003.011080

Aortic dissection (AD) is an uncommon condition that occurs mainly in the older patient population (>40 years). It is rare in younger people and is usually associated with trauma, Marfan's syndrome, or pregnancy. We report a case of a young weightlifter who died from an AD, and upon autopsy, was diagnosed as having non-Marfan's fibrillinopathy. We recommend that AD should be considered in symptomatic patients with any family history of early cardiac deaths, a history suggestive of a connective tissue disorder (that is, multiple joint surgeries) or who practise weightlifting.

Aortic dissection (AD) is an uncommon possibility in the differential diagnosis of back, chest, and abdominal pain, and of cardiac arrest, but occurs mainly in the older patient population (>40 years).[1] It is rare in younger people and is usually associated with trauma, Marfan's syndrome, or pregnancy.[2] Although Marfan's syndrome is well recognised in the emergency medicine literature, there is no mention of other fibrillinopathies or weightlifting as risk factors for AD. We report a case of AD in a young weightlifter ultimately diagnosed with non-Marfan's fibrillinopathy.

CASE REPORT

A 27 year old, previously healthy man was weightlifting at a local gymnasium when he experienced weakness, shortness of breath, and dizziness. Emergency medical services arrived within 10 minutes and found the patient unresponsive, with pulseless electrical activity as the initial cardiac rhythm. CPR was commenced as the patient was intubated, and he received epinephrine (adrenaline) and atropine during transport.

Upon arrival at the emergency department, the patient was cyanotic without pulse, blood pressure, or spontaneous respirations. Non-reactive pupils, bilateral breath sounds, and well healed incisions over both shoulders were noted, but no elongated body habitus, pigmented maculae, or subcutaneous air were seen.

Intravenous fluids, epinephrine, and vasopressin were administered, during which the cardiac rhythm degenerated

into asystole. An ultrasound of the pericardial window demonstrated no cardiac activity, pericardial effusion, or sub-diaphragmatic fluid. Despite these measures and lengthy CPR, asystole persisted and the patient was declared dead.

Relatives, once contacted, mentioned that the patient was in good health, but recalled that his mother had died at 39 years of age from an AD. The patient was aware he had an inherited connective tissue disease, having had two shoulder surgeries to repair ''loose ligaments''. He had undergone an echocardiogram without findings and no limitations had been placed on his activities. His friends, with whom he had been weightlifting, denied any knowledge of ephedrine or anabolic steroid use by the patient.

An autopsy determined that the cause of death was from a ruptured type II AD originating from the aortic root, with subsequent haemomediastinum and bilateral haemothoraces. The dissection extended to compress the left anterior descending coronary and pulmonary arteries. Pathology samples demonstrated an adventitial haematoma surrounding the aortic root with separation of the elastic lamellae between the middle and outer thirds of the media, findings consistent with a non-Marfan's fibrillinopathy.

DISCUSSION

With a mean onset age of 56.5 years, AD is a disease of older people. Patients younger than 19 years represented only 3.5% in two large series,[2] and have a high incidence of congenital cardiac anomalies (such as aortic coarctation) or connective tissue disorders (Ehlers-Danlos' and Marfan's syndromes are the most well known).

Another AD predisposition is weightlifting, which has been previously reported in both the cardiothoracic and paediatric literature.[2–5] There are no reports of AD in weightlifting patients with connective tissue disorders. The transient hypertension from increased heart rate and cardiac output have been well documented in healthy weightlifters without connective tissue disorders.[6] The highest blood pressure recorded (345/245) in prior studies was during squats (the exercise our patient was performing when he developed symptoms).

The majority of reports describe ascending ADs (the area of greatest haemodynamic stress),[3–5] which is also the most common location for dissection secondary to connective tissue disorders and congenital anomalies.[2] In these cases, the medial portion of the aorta is weakened not from hypertension induced degeneration (as is the case with the older population[1]), but instead is secondary to a congenital defect.

Perhaps the most well known connective tissue disorder is Marfan's syndrome. However, this entity represents only one end of a spectrum of conditions that stem from defective fibrillin-1 synthesis, collectively known as fibrillinopathies.[7] Fibrillin-1 is the lipoprotein that serves as the framework for elastin, the major elastic component of the aortic wall.[2 8 9] While Marfan's syndrome is a dominantly inherited condition, other fibrillinopathies vary in penetrance and expression,[7 8] and familiar non-Marfan's dissections have been described.[7 10–12] Recent work suggests that aortic involvement[13] may be related to premature termination codon mutations,[14] and to other mutations in the gene for fibrillin-1 (chromosome 15q21.1).[7 8 14]

Although no marfanoid features were noted on examination in this patient, his fibrillinopathy was a predisposition to developing AD, and the weightlifting activity was most probably a triggering event. A congenital structural anomaly is unlikely, as he had had a previous echocardiogram without finding.

The EM literature at this time does not include fibrillinopathies or weightlifting as AD risk factors. If AD is entertained as a possibility, aggressive blood pressure control, prompt imaging, and cardiothoracic consultation are essential. Common symptoms or complaints are back pain (55% of all presentations), chest pain (12%), lower extremity ischaemia (5%), and paraplegia (5%). The time elapsed between onset of a type A dissection and intervention is directly correlated with survival, with mortality increasing by 1% per hour.[15]

In conclusion, although a rare occurrence, AD should be considered in symptomatic patients with any family history of early cardiac deaths, a history suggestive of a connective tissue disorder (that is, multiple joint surgeries) or who practise weightlifting. The investigation and surveillance of fibrillinopathy patients is ill defined,[2] but prompt referral and/or admission for further investigation is merited. Cessation of weight lifting or isotonic stress activities until a definitive investigation has been obtained is prudent.

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Acute glaucoma presentations in the elderly

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Acute glaucoma classically presents with severe pain, redness, and reduced vision in the affected eye, and severe cases can also have systemic symptoms. We report three cases of acute glaucoma in elderly patients. The diagnosis of acute glaucoma in a patient who presents with sudden onset of a painful, red eye with reduced visual acuity, a hazy cornea, and a fixed, semi-dilated pupil is comparatively straightforward. However, any patient with headache, malaise, or gastrointestinal disturbance, especially with clinical signs of an acute red eye and reduced vision, should alert doctors to the possibility of acute glaucoma. This is especially important in elderly people, who may not volunteer any specific ocular symptoms.

Acute glaucoma classically presents with severe pain, redness, and reduced vision in the affected eye. Systemic symptoms such as headache, nausea, and vomiting may be associated in variable severity. The typical presentation is thus dramatic. In spite of this, especially in the elderly, there can be a delay or misdiagnosis of this condition. We report three cases to re-emphasise this and to draw attention to this condition.

CASE REPORTS

Case 1

A 77 year old woman was referred to the acute general medicine department by her general practitioner following a general deterioration in her condition. Her past medical history included Parkinson's disease, recent weight loss, and 2 days of vomiting.

On referral, her medications were sodium picosulphate 10 ml once daily, and thrice daily doses of betahistine 8 mg, bromocriptine 5 mg, domperidone 10 mg, levodopa 125 mg, and orphenadrine 50 mg. Family and personal history was inconclusive. On clinical examination, she was drowsy but oriented. All vital signs were normal. Systemic examination was within normal limits. Haematological and biochemical investigations, ECG, and chest *x* ray were normal. Malnutrition, malignancy, or chest infection was suspected as the likely causes of her condition.

Swollen lids and conjunctival injection were also noted. The systemic problem was thought to be with parkinsonism related generalised deterioration and cachexia, worsened by gastrointestinal upset and its treatment. Chloramphenicol eye ointment was started for the eyes. After 6 days, the patient was referred to the ophthalmologists with a persistent red eye.

Ophthalmic examination showed conjunctival injection and a semi-dilated, fixed pupil in both eyes. Fundus examination showed glaucomatous optic disc changes. Intraocular pressure (IOP) was 50 mm Hg in the right eye and 38 mm Hg in the left, suggesting acute angle closure glaucoma (AACG). She was given intravenous acetazolamide 500 mg immediately, sustained release acetazolamide capsules 250 mg twice daily; ocusert pilocarpine twice daily, levobunolol eyedrops twice daily, latanoprost eyedrops nightly, and prednisolone acetate ophthalmic suspension 1% (Pred Forte) eyedrops four times daily in both eyes. After 1 day, she was more comfortable, and IOP dropped to 25 mm Hg in the right eye, and 10 mm Hg in the left. YAG laser peripheral iridotomies were made in both eyes. Subsequently, the IOP was 22 mm Hg in the right eye and 25 mm Hg in the left.

Case 2

A 71 year old Indian woman was referred to the physicians by her general practitioner for sudden onset, right sided temporal headache, vomiting, and a red, sore right eye. The vision was poor from longstanding cataract and had not worsened. There had been similar "minor" episodes previously. On examination, there was scalp tenderness, and the right eye was red with advanced cataract. Erythrocyte sedimentation rate was 24 mm. A diagnosis of temporal arteritis was made. She was started on prednisolone tablets 60 mg once daily and referred to the ophthalmologists for temporal artery biopsy.

On ophthalmic examination, her vision was light perception in te right eye and 6/12 in the left. The right eye showed circum corneal congestion, corneal oedema, a shallow anterior chamber, semi-dilated fixed pupil, mature cataract, and IOP of 52 mmHg. The left eye showed a shallow anterior chamber and a pressure of 16 mmHg. A diagnosis of acute (?phacomorphic) glaucoma right eye was made. The patient was treated as for case 1. IOP dropped to 8 mm Hg in 2 hours.

Bilateral YAG laser peripheral iridotomies were performed. Subsequently she underwent cataract surgery with intra cular lens implantation separately for both eyes, and improved to 6/18 with correction in the right eye and 6/12 in the left.

Case 3

An 84 year old female with past medical history of Alzheimer's disease presented following a fall at home. She sustained a lacerated wound temporal to the left eye, which was sutured. There was no loss of consciousness or head injury.

Five days later, she developed increasing drowsiness with lucid interval, new and progressive headache and increasing confusion. Examination revealed left periocular bruising, mild subconjunctival haemorrhage and a red eye. The pupil was dilated and fixed. She was not oriented in time and space. There was no focal neurological deficit. A CT scan excluded subdural or intracranial bleed and showed fractures of the anterior and lateral walls of the left maxilla and displacement of the zygomatic arch. A fracture of the optic canal was suspected and the patient thus referred to the ophthalmologists.

On ophthalmic review, vision in the left eye was down to detection of hand movements. There was ecchymosis, chemosis, severe corneal oedema with pigment deposit, a

very shallow anterior chamber, a semi-dilated and fixed pupil, and some lenticular opacity in the left eye. Right eye vision and examination was normal except for a shallow anterior chamber. IOP was 24 mmHg in the right eye and 55 mmHg in the left. Gonioscopy revealed a narrow angle. An ophthalmic diagnosis of angle closure glaucoma in the left eye was thus made.

The patient was treated as for case 1. After 2 hours, the pressure was 35 mmHg, the headache had subsided, and the cornea had cleared. The lens was swollen. YAG peripheral iridotomies were performed the following day.

Ten days later, the patient underwent complicated cataract extraction with anterior vitrectomy and anterior chamber intraocular lens implantation. Three months later, vision was 6/60 and pressure 16 mmHg in the left eye, and the optic disc was pale and cupped.

DISCUSSION

In the UK, the incidence of AACG in people over the age of 40 years is about 1 in 1000, with a female to male ratio of 4:1.[1] In AACG, there is a build up of aqueous fluid in the anterior chamber of the eye leading to pain, reduced vision due to corneal oedema and optic nerve compromise, and redness due to circumcorneal congestion. Small, usually hypermetropic eyes that have a shallow anterior chamber and narrow iridocorneal angle are at a higher risk.[2] The initiating factor is the development of pupillary block, which is caused by contact between the iris in a semi-dilated pupil and the lens, resulting in blockage of the flow of aqueous humour from its site of production in the ciliary body, through the pupil, into the anterior chamber. This leads to a build up of aqueous humour in the posterior chamber. This in turn results in forward bowing of the iris, which then closes the already narrow iridocorneal drainage angle, producing a rise in intraocular pressure.[1 3] A persistent increase in the intraocular pressure results in a progressive and irreversible loss of vision and even blindness.

Physiological pupillary semi-dilation, such as occurs in the evening or when watching television in a darkened room, can precipitate an acute angle closure in an anatomically predisposed eye. Pharmacological dilation with 1% tropicamide, used routinely in ophthalmoscopy for diabetic retinopathy screening, rarely results in pupil block, but in diabetes the iris may spontaneously semi-dilate rather than fully dilate owing to autonomic neuropathy. This probably explains the increased risk of angle closure glaucoma in diabetic patients.[4] Various drugs, most commonly those with anticholinergic properties, such as the tricyclic antidepressants,[5] nebulised ipratropium bromide,[6 7] and nebulised atropine[8], have also been reported to precipitate angle closure glaucoma. In case 1, drugs such as levodopa and bromocriptine, which are dopaminergic, and orphenadrine, which is antimuscarinic, may have contributed to the acute angle closure.

Complete angle closure may be preceded by intermittent episodes of partial or temporary closure of the angle.[3] These are terminated by pupil constriction induced by bright light or sleep. Between episodes, however, the eye looks perfectly healthy, and the diagnosis must be based on a history of evening headaches associated with blurred vision and haloes around lights (due to the presence of corneal oedema), coupled with the presence of a shallow anterior chamber on slit lamp biomicroscopic examination. Patients with such symptoms should have a careful review of their drug treatment and be referred for an early ophthalmological opinion, as they will require laser peripheral iridotomy to prevent the development of AACG. Such a history of intermittent preceding symptoms is, however, present in only half of patients presenting with AACG.[1]

Systemic symptoms such as headache, malaise, and vomiting may be the presenting complaints apart from reduced visual acuity, pain, and redness of the affected eye in acute, prolonged closure of the iridocorneal angle. In some cases, and particularly in elderly people, the systemic disturbance may be so severe that the ocular symptoms are initially overlooked, as in our cases, and the patient may be misdiagnosed as having an acute medical or surgical condition. On examination, the visual acuity will be reduced in a red eye, which may feel hard to touch, with a cloudy cornea and a fixed, semi-dilated pupil. These patients require urgent ophthalmological referral for immediate medical treatment to lower the intraocular pressure, followed by definitive bilateral laser or surgical procedures. After iridotomy, iridectomy, or trabeculectomy, it is safe to dilate these patients' pupils and to re-start treatment with anticholinergic drugs.

CONCLUSION

The diagnosis of acute glaucoma in a patient who presents with sudden onset of a painful, red eye with reduced visual acuity, a hazy cornea, and a fixed, semi-dilated pupil is comparatively straightforward. However, any patient with headache, malaise, or gastrointestinal disturbance, especially with clinical signs of an acute red eye and reduced vision, should alert doctors to the possibility of acute glaucoma. This is especially important in elderly people, who may not volunteer any specific ocular symptoms. An awareness of the same should help in seeking early ophthalmological opinion.

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Fatal acute disseminated encephalomyelitis following treated snake bite in India

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Snake bite is an important cause of mortality and morbidity in India, with an estimated 35 000 to 50 000 fatal bites occurring annually. The neurological consequences of snake bite are predominantly the result of inhibition of neuromuscular transmission. We describe the first documented case of autopsy proven acute disseminated encephalomyelitis following treated snake bite in a young female.

A 27 year old woman was brought to the emergency department with a history of ptosis, weakness of all four limbs, and oliguria following a snake bite on the dorsum of her left foot 10 days previously. She was initially seen at a local medical college 6 hours after envenomation where 100 ml of polyvalent antivenin was administered 8 hourly, and one session of haemodialysis was given. There was an initial improvement in muscle power from grade 2/5 to 4+/5; however, there was a recurrence of weakness from day 8 after envenomation, and in view of progressive neuroparalysis and impending respiratory failure, she was referred to our institute. There was no history of blurred vision, dysphagia, dysphonia, haematuria, or dark coloured urine.

On physical examination she was conscious, oriented, and anxious, with a pulse rate of 104 beats/min, a blood pressure of 150/100 mmHg, and a respiratory rate of 26 breaths/min. There was generalised oedema, and fang marks were present on her left foot. On neurological review, bilateral external ophthalmoplegia and flaccid, hyporeflexic quadriparesis were noted. Investigations revealed normal haemogram (Hb 122 g/l (normal range 120–160 g/l), platelets 1.9×10^5/μl ($1.5–4\times10^5$), total leucocyte count 8.9×10^3/μl ($4–11\times10^3$), normocytic normochromic peripheral smear) and coagulation profile (prothrombin time 14 seconds (13–15), prothrombin time index 87%, activated partial thromboplastin time 37 seconds (35–40 seconds)), advanced renal failure (blood urea 38 g/l (2–4), serum creatinine 71 mg/l (4–12)), granular casts on urine microscopy and metabolic acidosis. Creatine kinase level was 21 U/l (normal 10–70). Plasma haemoglobin was 130 mg/l (normal <60), while urine was negative for both haemoglobin and myoglobin. Administration of antivenin and maintenance haemodialysis were continued, but the neuroparalysis persisted and a nerve conduction study revealed slowed conduction velocities, decreased amplitude of motor and sensory action potentials, and absence of F waves. The patient's sensorium gradually deteriorated over the next few days and she developed signs of raised intracranial tension (hypertension, blurring of optic discs) despite optimum haemodialysis and controlled hyperventilation. Broad spectrum antibiotics were added for ventilator associated pneumonia.

A CT scan of the brain revealed diffuse hypointensity of the white matter (fig 1) and there were 40 cells, predominantly lymphocytes, elevated protein (510 mg/l), and normal sugar on CSF examination. Oligoclonal bands were not detected. Intravenous methylprednisolone (1 g once daily for 3 days) followed by intravenous immunoglobulin (0.4 g/kg once daily for 5 days) were administered assuming a diagnosis of acute disseminated encephalomyelitis (ADEM); however there was no improvement in encephalopathy, and the patient finally died.

An autopsy was performed after informed consent from the relatives. The brain was normal on gross examination; however, there was microscopic evidence of extensive perivascular demyelination (fig 1B) and lymphocyte cuffing confrmed by solochrome cyanin stain. Lesions of similar age were distributed throughout the cerebral hemispheres, cerebellum, and brain stem. The kidneys revealed acute tubular necrosis; other organs were grossly and microscopically normal.

DISCUSSION

ADEM is an acute widespread demyelinating condition characterised by the rapid development of focal or multifocal neurological dysfunction. It usually follows 4–21 days after an infection or vaccination, and results from a transient autoimmune response against myelin or other autoantigens either by molecular mimicry or non-specific activation of an autoreactive T cell clone.[1]

More than 200 000 cases of snake bite are reported in India annually and it is estimated that between 35 000 and 50 000 of these are fatal.[2] Four clinically important types of snake are found in India: cobras (*Naja naja and Naja kaouthia*), the common krait (*Bungarus caeruleus*), Russell's viper (*Daboia russelii*), and the saw scaled viper (*Echis carinatus*).[3] The neurological consequences of snake bite are predominantly the result of inhibition of neuromuscular transmission.[4] In addition, there are reports in the literature of Guillain-Barré syndrome[5] and delayed neuropathy[6] following snake bite. To the best of our knowledge, this case represents the first report of demyelination involving both central and peripheral nervous systems following snake bite. ADEM was confirmed on autopsy, while the nerve conduction study performed antemortem was suggestive of demyelination.

The immunopathogenesis of demyelination following snake bite may be related to molecular mimicry between one of the components of snake venom and myelin and subsequent generation of pathogenetic auto-antibodies causing myelin damage. In this patient, it may also have developed as a consequence of a serum sickness-like reaction to the initial administration of antivenin, as neuroparalysis recurred a few days after definite clinical improvement. Late reactions to antivenin are immune complex diseases and present in the form of serum sickness syndrome usually 5–24 days after antivenom administration. Clinical features include fever, arthralgias, mononeuritis multiplex, and rarely,

Abbreviations: ADEM, acute disseminated encephalomyelitis

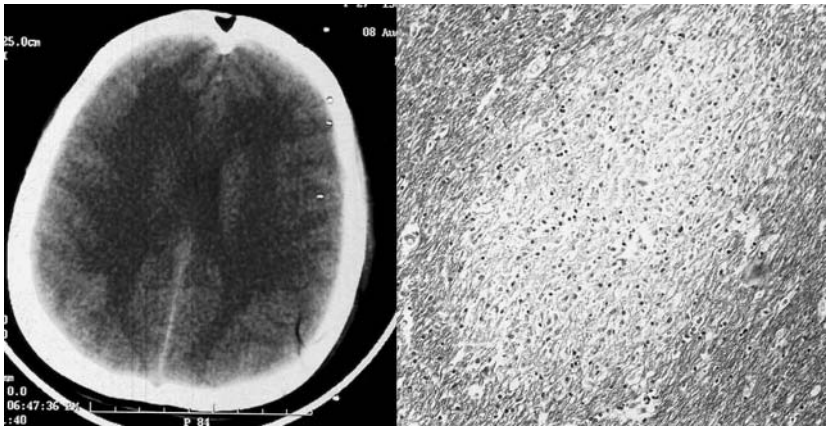

Figure 1 (A) CT scan showing diffuse white matter hypointensity; (B) solochrome cyanin stain demonstrating myelin loss and a mild lymphocytic infiltrate (original magnification × 32).

encephalopathy.[3] A reversible osmotic demyelination syndrome and demyelinating neuropathy have also been reported as a complication of dialysis in patients of advanced renal failure, and this may have been a third mechanism of demyelination in the index case; however such cases are rarely fatal.[7 8]

To conclude, we have described a previously unreported complication of treated snake bite and, if similar cases are seen by other physicians in countries where snake bite is a common problem, ADEM may be added to the list of neurological complications of snake bite. We suggest that patients with snake bite who develop unexplained encephalopathy should be screened for ADEM. Even though this is the first report of such a case, it is possible that if CT/MRI scans of the brain are ordered more often in patients with snake bite, this complication may be recognised more often and appropriate therapy instituted. Up to two thirds of patients with ADEM treated with corticoseroids benefit clinically, especially those who are treated early,[1 9] while intravenous immunoglobulin and plasmapheresis have been shown to produce dramatic improvement in steroid non-responsive cases.[10 11]

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Mydriasis due to *Datura inoxia*

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Unilateral, dilated unresponsive pupil may be secondary to a wide range of ocular and neurological disorders. "Gardener's pupil" is a pharmacological mydriasis caused by exposure to plants containing alkaloids. We report a case of mydriasis related to *Datura inoxia*, and detail a number of plants that have toxic effects. This report emphasises the importance of accurate history taking when evaluating fixed dilated pupil. We recommend that labelling of such poisonous plants should detail the local and systemic effects of accidental exposure, rather than merely the fact that they are poisonous.

Unilateral, dilated unresponsive pupil may be secondary to a wide range of ocular and neurological disorders. ''Gardener's pupil'' is a curious pharmacological mydriasis caused by exposure to plants containing alkaloids such as scopolamine, hyoscyamine, and atropine.[1 2] We report a case of mydriasis related to *Datura inoxia*.

CASE REPORT

A healthy 76 year old white male presented with a unilateral fixed dilated pupil, associated with blurred vision, noted 2 hours earlier. There was no accompanying headache, diplopia, or other neurological symptoms. Past medical history was unremarkable. Inadvertent self medication was denied.

General and neurological examination was normal. Visual acuity measured 6/5 and 6/6 in the right and left eyes respectively. The anterior segments and fundi were unremarkable, and ocular movements were full. However, the left pupil was dilated and unresponsive to both pupillary light reflex (direct, consensual) and accommodation reflex.

Topical pilocarpine 1% induced normal constriction of the right pupil but had no effect on the left, confirming the pharmacological basis of the left mydriasis.[3]

The patient subsequently recollected cleaning his indoor plants (which included *Datura inoxia*) earlier that day. Pupil size returned to normal over the next week and accommodation shortly thereafter.

DISCUSSION

A wide range of plants and plant products are potentially lethal, with local and systemic effects. Ocular side effects may be alarming, particularly when the pupil is dilated (table 1) with blurring of vision. *Datura inoxia* is not indigenous to the UK, though other members of the genus do grow in the wild. Related species (*Brugmansia*) are popularly grown in gardens in the UK for their beauty and fragrance. *Datura* is, however, poisonous, containing the alkaloids scopolamine, hyoscyamine, and atropine. These alkaloids mediate parasympatholytic effects secondary to muscarinic blockade.

Ocular toxicity occurs through inadvertent topical administration, while systemic side effects (primarily tachycardia due to a vagolytic effect[4]) occur through absorption of the alkaloids from the lacrhymal passages. Systemic effects are more pronounced upon oral ingestion. Although *Datura* is an outdoor plant, it is not fully hardy in the UK, so is moved indoors during the winter, with consequent risk, particularly to children and adolescents.[5 6] The simple measure of instillation of topical pilocarpine 1% establishes the pharmacological nature of the condition and obviates the need for

Table 1 Some plants and plant products causing mydriasis

Plant/substance producing mydriasis	Common names	Mechanism of mydriasis	Active ingredient
Amanita muscaria	Fly agaric	Sympathetic effect from action on CNS	Muscimol, ibotenic acid
Amanita pantherina	Panther cap	Parasympathetic palsy	Muscimol, ibotenic acid
Catha edulis	Kat, khat or qat leaves, also known as Abyssinian or Bushman's tea	Sympathetic stimulation	Cathione
Chenopodium oil	American wormseed oil	Not known	Not known
Lolium temulentum	Darnel, cheat, tare	Not known	Not known
Lupin seed (when ingested)		Not known	Not known
Solanum tuberosum, *Solanum nigrum*, *Solanum dulcamara*	Potato, black nightshade, woody nightshade	Parasympathetic palsy	Solanine, solanidine
Datura stramonium	Thornapple (extract is known as asthmador or stramonium)	Parasympathetic palsy	Hyoscyamine, atropine, scopolamine
Strychnos (may cause mydriasis)	Strychnine, brucine	Not known	Not known

Partly from Morton Grant W. Schuman JS. *Toxicology of the eye: effects on the eyes and visual system from chemicals, drugs, metals and minerals, plants, toxins and venoms; also systemic side effects from eye medications*, 2nd ed. 1993. By kind permission of Charles C Thomas Publisher Ltd, Springfield, IL, USA.

expensive neuro-imaging. Pilocarpine is competitively inhibited by alkaloids such as atropine, paralysing the iris sphincter.

Although accidental mydriasis is commonly due to parasympatholysis, it may also occur secondary to increased adrenergic stimulation (table 1). Care should therefore be exercised while interpreting a pilocarpine test.

This report emphasises the importance of accurate history taking when evaluating fixed dilated pupil. Retailers of such poisonous plants should detail the local and systemic effects of accidental exposure, rather than merely labelling a plant (non-specifically) as poisonous.

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