

CASE REPORTS

Penetrating injury to the soft palate causing retropharyngeal air collection

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Pharyngeal injuries caused by trauma are common and have been reported previously in the medical literature. In some cases of a penetrating injury there is a collection of air in the retropharyngeal space that can be shown on lateral soft tissue radiography of the neck. If this condition is not diagnosed or adequately treated the patient may develop severe complications such as mediastinitis. A case is reported of a patient with penetrating injury caused by a curtain rail and the subsequent treatment is described.

Pharyngeal injuries are seen commonly in medical practice, in particular in an emergency medical setting. The typical history is of a sharp object held in the patient's mouth causing an injury after a sudden movement. It is most often seen in paediatric patients. A significant injury however should be suspected even when there is only minor evidence of injury present to the oropharynx. Patients can present with minimal signs and symptoms. There are potential fatal complications such as vascular injuries and infection. We report a case of a penetrating injury causing collection of air in the retropharyngeal space.

CASE HISTORY

A 28 year old female patient presented to the department of otolaryngology after referral by the accident and emergency unit. She described an accidental penetrating injury to her soft palate. She had been holding a metallic curtain railing in her mouth when she bent down. The metal object penetrated her soft palate on the right side. She complained of pain in her mouth and along her right neck. She also admitted to odynophagia but denied any dysphagia. She described a "popping" sound when she swallowed. Mild bleeding from the site of injury was present. The patient was otherwise well with no significant medical problems.

On examination of her oropharynx, there was a small laceration measuring 0.5 cm present in the soft palate on the right side. There was no evidence of bleeding from the wound or surgical emphysema in the neck. She was afebrile with a normal blood pressure and pulse rate.

Lateral soft tissue radiography of her neck showed retropharyngeal air (fig 1).

Treatment was started with intravenous fluids and antibiotics (cefuroxime and metronidazole). She was made nil by mouth. The following day repeat soft tissue radiography showed mild reduction of the retropharyngeal air. A fine bore nasogastric feeding tube was inserted and enteral feeding started.

After three days repeat lateral soft tissue films showed complete resolution of the retropharyngeal air. The patient was restarted with oral feeding and discharged soon afterwards. She was completely asymptomatic when reviewed two weeks later in clinic. No further follow up was arranged.

DISCUSSION

Injuries to the palate are commonly reported and are not normally harmful. The cause is usually trauma to the oropharynx by objects held in the mouth. Sharp objects may however perforate the soft palate and cause collection of retropharyngeal air if sufficient force is delivered. Physical evidence of injury may only be mild. Foreign bodies may become trapped and can require surgical exploration for extraction.² Blunt external trauma if of sufficient force may also cause pharyngeal tears.³ Other causes of pharyngeal perforation include instrumentation and endotracheal intubation.¹ There are also reports of retropharyngeal air collecting after dental procedures.^{4,5} This has been attributed to extraction of teeth and the use of compressed air in dental drills and syringes. Retropharyngeal air accumulation can also be spontaneous, it has been reported in patients suffering with asthma.⁶ Occult perforation may occur in the absence of any obvious clinical signs. Lateral soft tissue radiographs are invaluable in diagnosing retropharyngeal air accumulation and soft tissue swelling. Such radiographs should be performed routinely in all clinical cases as the perforation may be otherwise undetectable by physical examination alone.¹

There are potential serious complications such as vascular injury to the carotid arteries or infection such as mediastinitis and abscess formation. Clinical features include chest pain, rigors, shortness of breath, systemic upset, dysphagia, and pleural effusion. Pus can accumulate in the chest cavity. Mediastinitis when established has a recognised high mortality rate. Air can also tract inferiorly to cause a pneumomediastinum.

The treatment of retropharyngeal air with no other complications is conservative with administration of



Figure 1 Lateral soft tissue radiography of the neck showing retropharyngeal air.

intravenous prophylactic broad spectrum antibiotics that reduces the risk of sepsis. Patients usually respond well to these measures. We used nasogastric feeding to preserve nutritional status. This is beneficial in patients where oral feeding is contraindicated for longer than a few days because of persistence of retropharyngeal air or other associated factors. Surgical intervention such as drainage of abscesses or air may sometimes be required in patients.

In conclusion, early and accurate diagnosis of penetrating soft tissue injuries is very important to avoid potential complications. As such emergency cases present at the accident and emergency department, close liaison between the accident and emergency staff and ENT surgeons is vital. A high index of suspicion should be adopted in cases of oropharyngeal trauma even when the clinical features appear mild. Conservative management that entails prophylactic antibiotics is usually sufficient to effect a complete resolution of the retropharyngeal air.

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Accessory ossicle or intraepiphyseal fracture of lateral malleolus: are we familiar with these?

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A case of intraepiphyseal injury (type 7) to the lateral malleolus in a 11 year old child is described. This rare injury cannot be classified by commonly used Salter Harris classification for epiphyseal injury. Although less common, accessory ossicle of the malleoli is an important differential diagnosis for such injury. Details of type 7 intraepiphyseal injuries and accessory ossicle are described.

An 11 year old child presented to an accident and emergency department after a fall from a bicycle, sustaining injury to the ankle. There was no history of previous injury to this ankle joint. The patient had pain over the lateral aspect of the ankle and clinical examination revealed swelling and tenderness over the lateral malleolus without any abnormality over the medial side of the joint. The lateral ligament complex, medial malleolus, ankle joint, and rest of the foot were normal on clinical and radiological examination. Radiographs showed intraepiphyseal injury of the distal fibular epiphysis (fig 1). The ankle was splinted in a below knee cast and the patient was permitted partial weight bearing. Three months after the injury there was no tenderness over the lateral malleolus and radiographs showed union across the fracture site (fig 2).

DISCUSSION

The most widely used classification for epiphyseal injuries is the one proposed by Salter and Harris in 1963. Although it is comparatively concise and of clinical importance, certain types of epiphyseal and physeal injuries cannot be readily classified with this system. John Ogden devised a more inclusive classification scheme in 1981, where he described

up to nine different types of injury to the growth mechanism of the immature skeleton.¹

Type 7 epiphyseal injury

Distal fibular epiphyseal injury is commonly either Salter-Harris type I or type II injury. Type 7 (intraepiphyseal) injuries are less common, occurring as a result of supination inversion injury to the ankle joint.¹ Although this injury is described in the literature, no major series of epiphyseal injuries specifically mention it.

Type 7 epiphyseal injuries, as described by Ogden,¹ are intraepiphyseal injuries and represent propagation of the fracture from the articular surface through the epiphyseal cartilage into the secondary ossification centre. Unlike other types of epiphyseal injuries they do not involve primary



Figure 1 Radiograph showing intraepiphyseal injury of the distal fibular epiphysis.



Figure 2 Radiograph three months after the injury.

physis at all. These types of injuries are common at malleoli; and within the distal humerus or distal femur as an osteochondral fracture.

Type 7 injury at the ankle is usually treated by closed reduction as weight bearing main articular surface is not usually interrupted. A transversely oriented fracture is usually stable but a more obliquely oriented fracture requires stabilisation. As this injury is rare, there are no long term studies available but theoretically there should not be major growth disturbance or angular deformity in the long term as the physis is not injured.

Accessory ossicle

An accessory epiphyseal ossification centre or accessory ossicle may develop in either malleolus.² Whether these accessory ossification centres represent a variation of ossification or a response to repetitive occult microtrauma is conjectural. They are not anatomically separate entities from the main ossification centre, even though they appear to be radiologically. Accessory ossicles of the malleoli are common in skeletally immature individuals; the lateral ossicle has been termed the “os subfibulare” and the medial “os subtibiale”. They usually appear between the ages of 7 and 10 years and eventually fuse with the secondary ossification centre of the malleolus at skeletal maturation. In one study of normal children aged between 6 and 12 years, accessory centre of ossification was found in the medial malleolus in 20% and in the lateral malleolus in 1%.³ John Ogden in his study of 103 patients with malleolar injury and ossification variation found that bilateral involvement (involvement of either medial or lateral malleoli on both the ankle) and involvement of both lateral and medial malleoli on one side can occur.² As the patient group in his study was obviously a highly selective population, statistics of incidence of the different variation cannot be derived. Such accessory centres of ossification rarely persist beyond skeletal maturation. A centre remaining unfused in adult life could cause confusion if found in an ankle that had recently been injured.

They usually are asymptomatic. However, they may be injured. As these accessory centres are most often found in the evaluation of acute trauma, they may be mistaken for fractures. Smooth appearance of the accessory centre should help in differentiation.

An acutely symptomatic patient with an accessory ossification centre should be considered and treated as having a fracture. The most probable explanation of symptoms is that the accessory ossification centre is disrupted through the cartilaginous continuity, resulting in a fracture or pseudoar-

throsis. The diagnosis of such injury by conventional radiography is difficult and a bone scan may help to diagnose such injury.

If an accessory centre is associated with ankle injury, it is appropriate to immobilise the ankle for three to four weeks and then to encourage mobilisation and weight bearing. Excision of the fragment should be reserved for the very few patients with recurrent symptoms over a prolonged period and with tenderness at the site of the accessory centre.

In our case it was a fracture and not accessory ossicle because (1) after the traumatic event, there was pain and tenderness over the distal fibular epiphysis of the previously asymptomatic ankle joint; (2) there was sharp fracture line in contrast with a smooth appearance seen in case of accessory ossicle; (3) on follow up, there were signs of fracture healing both clinically and radiologically.

Although it is easy to use the Salter-Harris classification for common epiphyseal injury, we need to be aware of more detailed classification as described by Ogden, to identify and understand less common epiphyseal injury.

JOHN OGDEN CLASSIFICATION OF INJURY TO GROWTH MECHANISM

Classification of injury to the growth mechanism as proposed by John Ogden is described.

The classification proposed by John Ogden is one concerned with injury to the growth mechanism rather than only physeal or epiphyseal injury. It is based partially on the Salter-Harris system. It is a more detailed scheme that permits further understanding of the injury to the growth mechanism as a whole.

Type 1

The epiphysis and most of the cellular regions of the physis separate from the metaphysis. The plane of cleavage is essentially through the zones of hypertrophic and degenerating cartilage cell columns, leaving more important resting and dividing cell layers of the germinal region undamaged and contiguous with the epiphysis.

Type 1 injuries are more common in neonates and infants with limited development of the secondary ossification centre. It may occur as abnormal fractures complicating underlying diseases such as rickets and osteomyelitis.

Because of the thick periosteum, displacement of the fracture is minimal making radiological diagnosis difficult. Slight widening of the physis may be the only sign. Complete displacement of the epiphysis although rare, can occur.

Type 1B

This occurs in children with systemic disorders affecting endochondral ossification patterns in metaphysis.

In contrast with type 1A where the fracture is primarily through the zone of hypertrophic cartilage, type 1B fractures may occur in the zone of degenerating cartilage and primary spongiosa. The initial fractures tend to be microscopic trabecular injuries.

Generally subsequent growth is normal in types 1A and 1B, because the essential germinal elements, the resting and dividing cellular layers of the growth plate and blood supply, are usually undisturbed.

Type 1C

It is a comparatively infrequent injury where there is an associated injury to a germinal portion of the physis. Localised area of growth region of the physis is subjected to a crushing injury. Birth or early infancy seems to be the most likely time of such injuries. Eventually an osseous bridge forms but only after the secondary ossification centre has developed and expanded to reach the damaged region.

Type 1 injuries are less likely to occur in children beyond the first two years, when the failure pattern is more likely to create a metaphyseal fragment (type 2 injury).

Type 2

It is the commonest of all the growth plate injuries. It frequently occurs in young children (from 3 to 7 years). The fracture line propagates through the hypertrophic zone of physis and then into the metaphysis. The metaphyseal fragment, which is generally triangular and may be extremely small, is diagnostic of this type of injury.

The periosteum is usually intact on the compression side with the metaphyseal fragment, whereas the opposite tension side is associated with disruptive periosteal damage as it is stripped and torn from the metaphysis. The intact periosteum on the compression side may be used as a hinge to assist in stable reduction. The periosteum may invaginate into the gap between epiphysis and metaphysis. Displacement is quite variable and may be extremely pronounced.

Type 2B

It entails further propagation of the fracture forces on the tensile side to create a free metaphyseal fragment. This free metaphyseal fragment makes reduction much more difficult and may necessitate open reduction to stabilise the comminuted fragments.

Type 2C

There is a thin layer of metaphysis along with, or instead of, the larger triangular fragment. This osseous layer traverses most of the metaphysis. It is more common in slower growing regions, such as phalanges, which normally have increased transverse trabeculation in the juxtaphyseal metaphysis.

Type 2D

In this type of injury, the area of the metaphysis is driven into a segment of the growth plate, causing compression damage to a localised area. Because of localised damage to the growth plate, subsequent growth may be eccentric and lead to angular deformation.

Type 3A

This is an intra-articular fracture primarily involving the epiphysis, with the fracture extending from the articular surface through the epiphysis and then turning about 90° to extend along the growth plate toward the peripheral margins.

Type 3B

When there is a thin layer of metaphyseal bone with the epiphyseal fragment, it is known as type 3B injury. This injury is common when the growth plate is undergoing the final phases of physiological epiphyseodesis. This is commonly seen in lateral humeral condyle and distal tibial epiphysis (Tillaux fracture).

Open reduction is frequently indicated to obtain accurate anatomical restoration. Prognosis is good provided there is no impairment of the circulation to the separated fragment. As these fractures occur as skeletal maturity is approaching, the chance for eventual growth disturbance is minimised by the lesser amount of anticipated longitudinal growth.

Type 3C

It includes injuries involving epiphyses that have developed major contour changes, such as the ischial tuberosity, in which epiphyseal fracture propagation may not necessarily involve a joint surface.

Type 4

It extends from the articular surface, across the epiphysis and physis, and subsequently through a significant segment of the metaphysis. It is important to achieve anatomical reduction to restore both a smooth articular surface as well as normal cytoarchitectural relations of the growth plate to minimise the potential risk of subsequent osseous bridging and localised premature growth arrest.

Despite this, growth damage and premature epiphyseodesis may still occur, most probably because of microscopic, compression type injury to regions of the growth plate.

Type 4B

There is additional propagation of the fracture line through remaining portions of the physis to create an additional free epiphyseal fragment. This tendency of fragmentation is more common when the patient is approaching skeletal maturity.

Type 4C

As in type 3C, type 4C fracture involves cartilaginous regions through which the fracture may propagate. Type C injury involves metaphysis, physis, and epiphysis.

Type 4D

It is a type of injury where there are multiple metaphyseal-epiphyseal fragments. There is increased risk of traumatically induced, localised epiphyseodesis.

Type 5

This is a compression injury through certain segments of the epiphysis and physis, crushing germinal regions as well as adjacent hypertrophic regions. It is a comparatively infrequent injury and is virtually unrecognisable by standard diagnostic techniques. This injury may be misdiagnosed as a sprain and the patient may later present with angular deformity. Other causes of this type of injury are electrical injury, radiation, and frostbite.

Type 6

This type of injury involves the peripheral region of the growth plate, the zone of Ranvier. More commonly it results from a localised contusion or avulsion of that specific portion of the growth mechanism. Peripheral osseous bridge formation commonly occurs, leading to peripherally localised epiphyseodesis and subsequent angular deformity. This type of injury is thought to be the possible underlying cause of solitary osteochondroma.

Type 7

These injuries are completely intraepiphyseal and represent propagation of the fracture from the articular surface through the epiphyseal cartilage into the secondary ossification centre. They do not involve physis. These types of injuries are common at malleoli, and within the distal humerus or distal femur as an osteochondral fracture.

Type 7A

The fracture passes through both the epiphyseal cartilage and bone of the secondary ossification centre.

Type 7B

It represents propagation of the fracture through the cartilaginous portions, with involvement of some of the preossifying regions of the expanding secondary ossification centre. These types of fractures may also involve non-articular regions such as the tibial tuberosity, the greater trochanter, and the fifth metatarsal at its proximal base.

Type 8

These are injuries to the metaphyseal growth and remodelling mechanisms and represent transient phenomena primarily related to vascularity. With these injuries, the metaphyseal circulation involved in primary spongiosa formation from the cartilage cell columns is temporarily disrupted, leading to failure of normal osseous remodelling and subsequent, transiently increased osseous density as the area is revascularised.

Type 9

This is a selective injury to the diaphyseal growth mechanism. Any direct injury to the periosteum may affect the ability of the bone to remodel and increase cortical volume circumferentially.

A highly osteogenic periosteal sleeve is one of the important mechanisms for longitudinal as well as apposi-

tional bone growth. Any type of insult with this mechanism may result in impaired growth of the bone.

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Severe interscapular pain and increased creatine kinase activity: the answer was in the ankles

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Severe thoracic back pain with increased creatine kinase activity is a clinical presentation that suggests a variety of life threatening conditions. If initial examination is unrevealing, multiple diagnostic tests are usually performed attempting to identify the origin of the problem, sometimes neglecting apparently unrelated subtle physical findings. A patient is described in whom this was the initial presentation of a sensory demyelinating neuropathy, resulting in a diagnostic challenge. This case expands the differential diagnosis of severe thoracic back pain and increased creatine kinase activity, and illustrates the importance of physical examination in reaching a final diagnosis.

Acute or subacute thoracic back pain is a symptom that raises a differential diagnosis including multiple potential life threatening conditions. In the presence of raised creatine kinase (CK) activity, myocardial injury will be an immediate concern. I encountered a patient who presented with severe thoracic back pain and raised CK that resulted in a diagnostic challenge. This case illustrates how subtle physical findings, even when apparently unrelated to the presenting problem, could represent an important clue to reach a final diagnosis.

CASE REPORT

A 59 year old man gradually developed severe sharp interscapular pain with occasional descending radiation, prompting a visit to the emergency room. Initial evaluation included a diminished muscle stretch reflex at the left ankle and an absent reflex on the right with otherwise unremarkable general and neurological examinations. Haemogram, electrolytes, liver and pancreatic enzymes were normal, but

CK was 615 IU/l (normal 40–200 IU/l), with a normal cardiac (MB) fraction. Because of appropriate concerns about life threatening conditions such as myocardial infarction, aortic dissection, acute pancreatitis, or spinal cord compression, a range of emergent diagnostic investigations was obtained. An electrocardiogram showed sinus tachycardia. Chest radiograph, chest computed tomography with contrast, and abdominal ultrasound were normal. Magnetic resonance imaging of the cervical and thoracic spinal cord was unrevealing. He was treated with meperidine resulting in short lasting pain relief. Three days later he developed distal lower extremity hypesthesia and was transferred to our institution. On admission, his vital signs were remarkable only for a sinus tachycardia. Neurological examination showed mild distal hypesthesia in a stocking and glove distribution, slightly impaired vibratory sense in the feet, diffuse areflexia, and mild gait ataxia. He was treated with gabapentin, amitriptyline, and diazepam, achieving pain relief within 24 hours. Brain and thoracic spine magnetic resonance imaging, serial CK determinations, and troponin were normal. Nerve conduction velocities were generally slow with no changes in compound motor unit amplitude, supporting the diagnosis of a demyelinating peripheral neuropathy. The cerebrospinal fluid analysis showed 0 cells/mm and a protein of 790 mg/l (normal 150–450). His clinical presentation and diagnostic studies met the proposed diagnostic criteria for sensory Guillain-Barre syndrome (GBS).¹ One month later he suffered a relapse with worsening ataxia, mild distal lower extremity weakness, diffuse areflexia and more prominent sensory loss, and ataxia. Repeated nerve conduction studies showed diffuse slowing with increased latency and temporal dispersion, increased F wave latency and conduction block, further supporting the diagnosis of an acquired demyelinating

Abbreviations: CK, creatine kinase; GBS, Guillain-Barre syndrome

Table 1 Diagnostic criteria for GBS

Sensory GBS ¹	Typical GBS ^{2*}
Acute onset of symmetric sensory loss	Progressive weakness in both arms and legs†
Peak deficit achieved within four weeks	Areflexia†
Diminished or absent reflexes	Progression of symptoms over days to four weeks
Normal motor strength	Relative symmetry of symptoms
Electrodiagnostic evidence of demyelination in at least two nerves	Mild sensory symptoms or signs
Monophasic course	Cranial nerve involvement
No other known cause for neuropathy	Recovering two to four weeks after progression ceases
No family history of neuropathy	Autonomic dysfunction
Albuminocytological dissociation in CSF	Absence of fever at onset
	Albuminocytological dissociation in CSF
	Typical electrodiagnostic features

*Excluding diagnosis: diagnosis of botulism, myasthenia, poliomyelitis or toxic neuropathy; abnormal porphyrin metabolism; recent diphtheria; purely sensory symptoms, without weakness. †Features required for diagnosis.

process. He was treated with a course of intravenous immunoglobulin G with remarkable improvement. In the following three years, there were no additional relapses and his neurological examination reverted to normal.

DISCUSSION

GBS is an immune based, acquired inflammatory demyelinating polyradiculoneuropathy. The classic presentation of this syndrome consists of a monophasic acute ascending paralysis associated with areflexia, but many clinical variants have been described.² Sensory GBS is an unusual presentation characterised by symmetric sensory loss, diminished or absent reflexes and normal motor strength, associated with conduction abnormalities in motor nerves shown by electrodiagnostic studies.¹ The main differential factor between both forms of GBS is the presence of progressive weakness in more than one extremity as a required diagnostic criteria for typical GBS, a clinical finding that rules out the diagnosis of sensory GBS. The rest of the diagnostic criteria are very similar, with minor differences, as shown in table 1.

Although the presence of pain is a common feature of GBS, occurring in up to 50% of patients, it is typically severe low back or leg pain, and often is the presenting symptom.³⁻⁴ Raised CK can be occasionally seen in GBS but its origin in this syndrome is still unclear.⁴⁻⁵ The combination of severe interscapular pain and increased CK at presentation in our patient is unusual, and appropriately raised the concern of various other life threatening conditions such as aortic dissection, myocardial infarction, pulmonary embolism, or acute pancreatitis, prompting an extensive emergent work up. Weakness, sensory loss, and nerve conduction abnormalities may be subtle or absent at the onset of GBS. The

subsequent development of distal hypesthesia, hyporeflexia, and ataxia unmasked the acquired acute demyelinating neuropathic process that was confirmed by the unequivocal findings of the nerve conduction studies during the acute and relapse phase of the illness. This case suggests that GBS should be included in the differential diagnosis of severe interscapular pain associated with raised CK activity. A careful evaluation for subtle clinical signs of early GBS, such as decreased or absent ankle muscle stretch reflexes, may help redirect the diagnostic evaluation, avoid unnecessary tests, and prompt appropriate treatment of this disabling and occasionally fatal illness.

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Life threatening hyperkalaemia with diarrhoea during ACE inhibition

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A 67 year old woman developed acute renal failure with serum potassium 9.4 mmol/l requiring emergency dialysis after seven days of diarrhoea while taking an ACE inhibitor for vascular disease. Review of the literature, the *British National Formulary*, and the patient information leaflets for each of the 11 ACE inhibitors currently marketed in the UK suggests that this potentially life threatening complication of ACE inhibition is not yet widely recognised.

ACE inhibitors are prescribed for diabetes, hypertension, and cardiovascular disease on account of their cardiac and reno-protective properties. Gastroenteritis is one of the commonest minor illnesses affecting an estimated million people in the UK each year. It is usually viral in origin, self limiting, and only rarely life threatening. Inevitably, some patients will develop gastroenteritis while they are taking ACE inhibitors and when this happens the resulting volume depletion may precipitate acute renal failure as a result of the known actions of ACE inhibitors on the renal vasculature.¹ We have recently conducted a survey of emergency medical admissions and shown that a hospital with a catchment population of 150 000 might expect to see one case of acute renal failure occurring as a result of diarrhoea during ACE inhibition each month.² All cases of acute renal failure in this series resolved without the need for dialysis, after temporary withdrawal of the ACE inhibitor and large volumes of intravenous fluid. We now wish to report another complication of diarrhoea during ACE inhibition namely that of life threatening hyperkalaemia.

CASE REPORT

A 67 year old woman was admitted as an emergency unresponsive with Glasgow coma scale 6/15. Her presenting complaint was seven days of diarrhoea on a background of widespread vascular disease for which she was taking enalapril, bisoprolol, bumetanide, and isosorbide mononitrate. She was profoundly bradycardic at 20 beats per minute with systolic blood pressure 40 mm Hg and an undetectable diastolic pressure. Electrocardiography showed a broad QRS complex with absent P waves. She was paced as an emergency. Biochemistry then showed serum potassium 9.4 mmol/l, serum bicarbonate 9 mmol/l, blood urea 42.6 mmol/l, and serum creatinine 598 µmol/l. The high concentration of serum potassium was confirmed on a second specimen. Three weeks previously serum potassium had been 5.1 mmol/l with urea 15.3 mmol/l and creatinine 135 µmol/l. After treatment with calcium chloride, insulin/dextrose, and emergency dialysis, heart rate and blood pressure were restored to normal values and serum potassium decreased to 4.4 mmol/l. Rehydration with large volumes of intravenous fluid led to return of urine output. No further dialysis was required. Stool culture was sterile and her diarrhoea

settled spontaneously. This patient's ACE inhibitor and other medications were re-introduced before discharge from hospital. Serum potassium at clinic review two months later was 4.3 mmol/l with urea 9.7 mmol/l and creatinine 81 µmol/l.

DISCUSSION

Both hyperkalaemia and acute renal failure are recognised complications of ACE inhibition.³ Hyperkalaemia occurs partly because ACE inhibitors lower plasma aldosterone values and partly as a result of acute renal failure (ARF). Hyperkalaemia is more common in elderly patients, those with pre-existing renal impairment, and in patients who are also taking potassium supplements, potassium sparing diuretics, non-steroidal anti-inflammatory drugs, or β blockers.^{4,5} ARF is a consequence of the failure of renal auto-regulation because of loss of angiotensin II mediated efferent arteriolar vasoconstriction, leading to a reduction in glomerular capillary pressure when renal perfusion pressure falls: commonly because of volume depletion, hypotension, co-prescription of a non-steroidal anti-inflammatory drug or bilateral renovascular disease.¹

Our patient presented with life threatening hyperkalaemia and acute renal failure when she developed diarrhoea while taking enalapril on a background of widespread vascular disease. Her loop diuretic will probably have protected against hyperkalaemia while increasing the risk of uraemia, whereas her β blocker may have had the opposite effects.¹ No other causes of hyperkalaemia or ARF were apparent. Her ACE inhibitor was successfully reintroduced after renal function returned to normal after dialysis and resolution of her diarrhoea.

We² and others^{6,7} have previously reported cases of ARF attributable to diarrhoea during ACE inhibition and have argued that affected patients may present in this way in part because vagal stimulation by the ACE inhibitor blocks the tachycardia response to volume depletion that would normally alert a patient to the fact that they were unwell.^{8,9} There were of course other explanations for the profound bradycardia in our patient's case, namely her severe hyperkalaemia and her β blocker. We suspect that these complications of ACE inhibitor treatment will be recognised by most hospital doctors but perhaps less so by general practitioners.¹⁰ Neither the *British National Formulary* nor the patient information leaflets for each of the 11 ACE inhibitors currently marketed in the UK make any reference to the threat posed by diarrhoea during ACE inhibition, persuading us that these potentially life threatening complications of ACE inhibition are not as well publicised as they deserve to be.

What advice should be given to the patient who develops acute diarrhoea while taking an ACE inhibitor? Temporary withdrawal of diuretic or ACE inhibitor, or both, while maintaining a high oral fluid intake, is likely to be successful in mild cases. For those who develop ARF, admission to hospital for intravenous fluid will be required. Temporary

Learning point

Patients with diarrhoea or vomiting, or both, while taking an ACE inhibitor may develop acute renal failure with life threatening hyperkalaemia.

dialysis will be needed occasionally and perhaps particularly if the ARF is complicated by life threatening hyperkalaemia as in the case reported here. Similar advice is likely to be appropriate for any patient taking an ACE inhibitor, angiotensin receptor blocker, or non-steroidal anti-inflammatory drug, whose circulation is compromised for any other reason during an acute illness, for example, by septic or cardiogenic shock. Failure of renal function to recover after dialysis and restoration of extra cellular fluid volume is rare.¹¹ In the longer term there is evidence that angiotensin receptor blockers are less likely to cause hyperkalaemia than ACE inhibitors, which may make these the drugs of preference when re-instituting cardioprotective treatment.¹²

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