CASE REPORTS

Fracture of the occipital condyle: the forgotten part of the neck

Andrew Kelly, Richard Parrish

Abstract
A case of occipital condylar fracture in a multiply injured and unconscious motorcyclist is reported. This injury was clinically unsuspected but found on the lowest cuts of head computed tomography. It is shown that this site is often inadequately imaged when scanning the head and neck in victims of trauma. The Anderson and Montesano classification of occipital condylar fracture is described. It is noted that types 1 and 2 are stable injuries but type 3 is potentially unstable. A retrospective analysis of 30 head computed tomography scans in trauma cases revealed that in only 16 were the occipital condyles adequately imaged. It is emphasised that vigilance is required to detect fractures of the occipital condyle and that it should be standard practice to include this area when performing computed tomography of the head in trauma victims.
(*J Accid Emerg Med* 2000;**17**:220–221)

Case report

A 16 year old male road traffic accident victim was found lying beside his motorcycle with the helmet some distance away. The exact speed and circumstances of the incident were unclear. Paramedic roadside opinion was that the victim had suffered a tumbling fall with consequent potential for twisting head injury. In the prehospital phase a semirigid cervical collar was applied and fluid resuscitation given.

On arrival at hospital the patient was haemodynamically stable with a Glasgow Coma Scale score of 9 and other than bleeding from an occipital scalp wound no external injury was seen. He was electively intubated and ventilated. Cervical spine control was maintained at all times. Fluid resuscitation was continued and initial radiographs revealed bilateral pulmonary contusions. Cervical spine plain films were normal.

Immediate computed tomography of the head chest and abdomen was performed and a frontal lobe haematoma with mild cerebral oedema was seen. The lowermost head cuts revealed a displaced fracture of the right occipital condyle (fig 1). Ventilation was continued and ongoing care given by intensiv-

Figure 1   Computed tomography showing fractured right occipital condyle.

ists and the neurosurgical team. Intracranial pressure was monitored and remained normal. Halo traction was applied before his extubation on day four. He required three months rigid fixation in a halo vest. Follow up computed tomography showed healing at the fracture site without evidence of subluxation. Residual cognitive impairment at two weeks necessitated referral to the head injury rehabilitation unit and at the time of writing cognitive function and speech are now normal and a residual swallowing problem is improving constantly.

Discussion

Occipital condylar fracture was originally described by Charles Bell in 1817.[1] This was a postmortem diagnosis of a hospital patient who sustained a fall at the time of discharge! His demise was attributed to medullary compression by the condylar fragment. Before the wide availability of computed tomography diagnosis in life was rare but by 1996 61 cases had been reported in the literature. In addition other occipital condylar fractures found at postmortem examination have been described.

Often patients with this diagnosis have loss of consciousness because of associated head injury and indeed most early neurological deficits in patients with occipital condylar fracture are attributable to such related injury. In those patients who are conscious on presentation neck pain is usual and indeed may be the only

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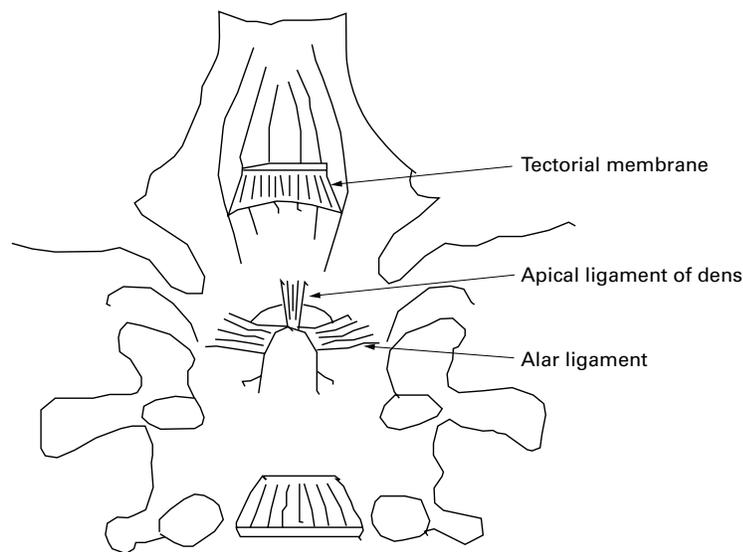
Figure 2   The atlantooccipital joint viewed from behind.

Labels in figure: Tectorial membrane; Apical ligament of dens; Alar ligament

symptom although neurological deficit including hemiparesis (attributable to contusion of ipsilateral adjacent spinal cord) is recognised. Lower cranial nerve deficits (that is, IX–XII) are a "classic" sign. However they occur in only a third of cases and within this group a third of such deficits present late.[2]

Cervical spine radiographs are usually normal[2] although prevertebral soft tissue swelling and asymmetry of the odontoid peg as seen on the anteroposterior view are recognised signs. In addition computed tomography of the head often neglects fully to image the craniocervical junction and the recognition of this injury therefore presents a diagnostic challenge. Because neurological deficit can be late in onset it is however necessary to make the diagnosis at the time of presentation. It is therefore important to retain the same index of suspicion for skull base injury as is widespread for the cervicodorsal junction.

It is best to consider the paired occipital condyles and the first two cervical vertebrae as a functional unit. The stability of the atlantooccipital junction within this unit is maintained by several structures.[3] Firstly, the tectorial membrane is a continuation of the posterior longitudinal ligament and attaches to the foramen magnum. This limits flexion and extension. Secondly, the paired alar ligaments run directly from the odontoid peg to each occipital condyle; their function is to check lateral flexion and rotation. It is these above structures that primarily stabilise this part of the neck. Finally, the anterior and posterior atlanto-occipital membranes interconnect the margins of the foramen magnum and the ring of the atlas while in addition the apical ligament extends from the odontoid peg to the anterior margin of the foramen magnum.

Anderson and Montesano described a commonly used classification system based on injury mechanism and computed tomography fracture morphology.[4] Class I fractures are impacted condylar fractures consequent upon axial loading injuries. Type II fractures are extensions of a basilar skull fracture. Both these types are functionally stable consequent upon integrity of the tectorial membrane and contralateral alar ligament. Type III fractures, sustained during extreme rotation and/or lateral bending are avulsion fractures of the condyle by the alar ligament. This type of fracture is potentially unstable because of loading of the tectorial membrane and contralateral alar ligament. Tuli *et al*[2] recommend an alternative classification. They propose that instability is determined more by ligamentous injury as detected by computed tomography alignment criteria and magnetic resonance imaging findings than on fracture morphology.

Optimal treatments are not fully agreed upon but most sources propose semirigid collar provision for stable injury and rigid immobilisation of varying design for the potentially unstable injury.

To monitor the adequacy of our computed tomography head scans in imaging the craniocervical junction we retrospectively assessed a series of 30 computed tomography scans of head injured patients examined before the case under discussion. The hard copies were examined for inclusion of the occipital condyles. In only 16 of 30 patients was there satisfactory demonstration of these structures. It is already our policy to print "bone window" images in addition to standard "brain windows" in acutely head injured patients and we now ensure that computed tomography in patients with head injury starts at the C1 ring.

In conclusion, vigilance is required to diagnose occipital condylar fracture and in particular we advise that all unconscious trauma victims undergoing computed tomography head imaging need careful examination of the craniocervical base. This should include 3 or 5 mm axial sections to cover the occipital condyles, with inspection of the resulting images on "bone windows" in addition to the conventional "brain" settings.

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Spontaneous pneumothorax: Is it under tension?

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Abstract
A diagnosis of tension pneumothorax is usually only considered within the context of trauma, incorrect chest drain insertion or positive pressure ventilation. Four patients are presented who developed spontaneous tension pneumothorax with no precipitating factors. In three of these instances, the diagnosis was only made radiologically and in every case the treating physician was unaware that a spontaneous tension pneumothorax could occur. Previously, emphasis has been placed on tracheal deviation in a tension pneumothorax. However, this is an inconsistent finding as one of the cases highlights. Patients may appear surprisingly clinically well until they decompensate. These cases are highlighted to raise awareness of this potentially life threatening condition.
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Keywords: spontaneous pneumothorax

Case reports

CASE 1
A 22 year old woman presented with sudden onset of right sided chest pain and dyspnoea. Her only past medical history was mild asthma. On examination, she was speaking full sentences, respiratory rate 24/min, haemodynamically normal and had an oxygen saturation of 97% on high flow oxygen. Of note, her trachea was central. There was clinical evidence of a right sided pneumothorax and no wheeze. A diagnosis of spontaneous pneumothorax was made but a portable chest radiograph showed this to be under tension. Re-assessment confirmed a centrally placed trachea, but apex beat was noted to be displaced to the anterior axillary line on the left and percussion resonance noted over the sternum. Needle thoracocentesis resulted in immediate relief of her dyspnoea. A chest drain was inserted, but she developed a persistent air leak with failure to re-inflate the lung. The patient proceeded to thoracotomy where she was found to have a small apical bulla, which required oversew and pleurectomy.

CASE 2
A 19 year old man developed sudden onset of left sided pleuritic chest pain and dyspnoea while walking uphill. He had no previous medical history and was physically fit. On arrival to hospital his breathlessness had resolved. On examination he was clinically well, respiratory rate was normal, pulse 60, blood pressure 130/70 and oxygen saturation 98% in air. There was no air entry on the left and decreased chest expansion on that side. A diagnosis of simple pneumothorax was made

and a portable chest radiograph ordered. The radiograph showed a left tension pneumothorax with marked mediastinal shift. On re-examination the apex beat was found to be heard maximally over the sternum and on percussion, the mediastinum was shown to be displaced to the right. He remained well until he lay flat for a chest drain insertion, upon which he became breathless and tachycardic. His symptoms settled when he sat up and the chest drain was inserted without difficulty. He was admitted to hospital for three days and made a full recovery.

CASE 3
A 20 year old woman presented to the accident and emergency department in extremis, complaining of right sided pleuritic chest pain and increasing shortness of breath for the past five hours. On examination she was tachycardic, hypotensive and dyspnoeic. Oxygen saturation was 91% on high flow oxygen. Her trachea was deviated to the left and she had decreased air entry and resonant percussion note on the right. A needle thoracocentesis resulted in dramatic clinical improvement. A chest drain was inserted; air and 700 ml of blood drained over the next two hours. Her right lung failed to re-inflate satisfactorily. At thoracoscopy she was found to have longstanding apical adhesions, which had bled as the lung collapsed causing an air leak.

CASE 4
A 45 year old man presented with a three day history of left sided chest pain, cough and dyspnoea. His dyspnoea had significantly increased that morning. On examination he was unwell, tachypnoeic 36/min, pulse 130, temperature 37.9°C and normotensive. His trachea was deviated to the right, he had decreased air entry and resonant percussion note on the left. The patient was sent to radiology by the doctor attending him as he did not believe his own clinical diagnosis of tension pneumothorax, thinking that it was impossible without precipitating trauma. Chest radiography confirmed a tension pneumothorax and on re-examination his apex beat was heard maximally over the sternum. He underwent needle thoracocentesis, which partially relieved his dyspnoea, and chest drain insertion. He made a full recovery.

Discussion
Figures from the USA state an incidence of 8600 cases per year of spontaneous simple pneumothorax,[1][2] approximately 1–2% of these will be under tension.[2] Advanced Trauma Life Support (ATLS) teaching has emphasised the importance of tension pneumothorax[3] so much

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

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

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

Conclusion

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

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Aorto caval fistula—the "bursting heart syndrome"

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

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Case report

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

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

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

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

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

Discussion

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

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

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

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

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

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

(2) Abdominal bruit and thrill

(3) Palpable abdominal aneurysm

(4) Oliguria

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

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

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

Conclusion

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

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

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Screwdriver assaults and intracranial injuries

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Abstract

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

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

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

Case reports

CASE 1

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

CASE 2

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

CASE 3

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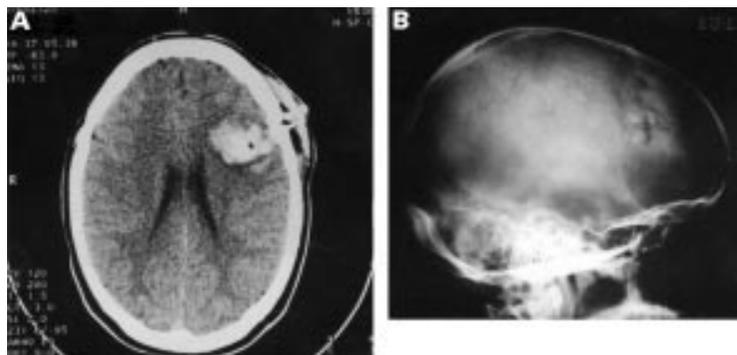

Figure 1 (A) Computed tomography showing left frontal intracerebral haematoma and skull vault fracture with (B) corresponding plain skull radiograph showing a depressed skull fracture.

subdural haemorrhage and a small contusion within the brainstem. He underwent craniotomy to evacuate the subdural haemorrhage but despite aggressive management died two days after surgery.

CASE 4
A 17 year old man was assaulted with a screwdriver and brought to A&E with the screwdriver still embedded in the left parietal region of his head. On arrival his GCS was 3/15, he was maintaining an adequate circulation but required mechanical ventilation. His pupils were fixed and dilated with no "dolls eyes" movement. Skull radiography and compted tomography showed the tip of the screwdriver to be within the brain stem. He subsequently underwent formal brain stem testing and was pronounced dead.

Discussion

The few penetrating intracranial injuries seen in the UK are predominately gun shot wounds. Occasionally more unusual weapons have been used to penetrate the cranium including nails, wooden objects, and drills.[4–6] The commonly used weapons such as knives and broken bottles rarely penetrate the skull, and are more likely to glance off the cranial surface or break. However screwdrivers, because of their rigid structure and narrow tip, may forcefully penetrate the cranial bone.

If the screwdriver is not still embedded then the external injury may appear insignificant. Unlike cranial stab wounds when the damage is usually limited to a focal area along the tract,[7] being a tool with a narrow diameter the intracranial injury can be extensive from pivoting of the tip of the screwdriver within the brain substance.

These cases are a reminder of the importance of cleaning blood from the scalp in patients with head injuries to assess any underlying wounds. The possibility of a penetrating injury can then be considered. The first two cases are also a reminder of the need for a high index of suspicion in intoxicated head injured patients in whom the diagnosis of intracranial injuries is often delayed.

In patients where there is a possibility of a penetrating intracranial injury, skull radiographs and subsequent computed tomography are indicated. As compound wounds these injuries require appropriate antibiotic cover and tetanus prophylaxis. Further neurosurgical assessment will determine whether formal debridement and removal of haematoma and devitalised tissue or depressed bony fragments is indicated.[8]

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Volar metacarpophalangeal joint dislocation

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Abstract
Volar dislocation of the metacarpophalangeal joint is a very rare clinical finding. A volar dislocation of the metacarpophalangeal joint of the left index finger in a 44 year old man is reported. Closed reduction proved unsuccessful requiring subsequent open reduction and internal fixation via a combined dorsal and volar approach. The presentation, mechanism of injury and treatment of this case and other previously reported cases are discussed.
(*J Accid Emerg Med* 2000;**17**:226–228)

Keywords: vovar dislocation; metacarpophalangeal joint

Volar dislocation of any metacarpophalangeal joint (MCPJ) is rare in comparison with its dorsal equivalent, which was described by Kaplan in 1957 and subsequently by other groups.[1–3] Only nine cases of volar dislocation of the MCPJ have been described in the English medical literature.[4–10]

The condition is classified as simple if closed reduction is successful, or complex, if open reduction is necessary to overcome soft tissue interposition.[7] We present a further case of volar MCPJ dislocation of the index finger and its management following a hyperflexion injury that has only been reported as a cause once before.[5]

Figure 1   Lateral view radiograph of the left hand demonstrating volar dislocation of the ring finger metacarpophalangeal joint (indicated by arrow).

Case report

A 44 year old right handed labourer presented to the accident and emergency department having fallen onto his outstretched left hand sustaining a hyperflexion injury. He complained of swelling and tenderness over the base of the left ring finger plus paraesthesiae and decreased movement in the digit. Examination revealed loss of bony prominence of the left ring knuckle and no movement at the MCPJ but normal movement in the joints distal to this. Radiological examination revealed volar dislocation of the left ring finger MCPJ associated with a minimally displaced fracture of the base of the proximal phalanx (fig 1). All other hand and wrist examinations were normal.

An immediate successful closed reduction was performed using inhalational anaesthesia being confirmed on radiological examination. The left ring finger was neighbour-strapped and the patient returned the following day when reduction was maintained. A week later, the patient complained of pain and stiffness around the left ring finger MCPJ. Examination revealed a maintained reduction and physiotherapy started. At three weeks after injury, once again there was loss of prominence of the left ring knuckle and a protuberance on the volar surface. Check radiography revealed a recurrent volar MCPJ dislocation and slight displacement of the fracture at the base of the proximal phalanx.

It was decided to correct the deformity by open reduction and internal fixation. Under general anaesthesia, a dorsal incision revealed the presence of scarring around the capsule and collateral ligaments. There was callus formation surrounding the base of the proximal phalanx and incongruity of the articular joint surface. Reduction of the MCPJ proved impossible by this approach alone so that a volar incision was made. This revealed an interposed volar plate within the MCPJ. After partial resection of the volar plate, reduction of the MCPJ was possible. The joint and fracture were fixed using an intra-articular k-wire. Three weeks later, the k-wire was removed and two months later there was no recurrence of deformity with 10–90 degrees of flexion at the MCPJ and full movement of the proximal and distal interphalangeal joints.

Discussion

The reports of volar dislocation of any MCPJ are rare. To date, nine cases have been reported.[4–10]

The mechanism of injury has been reported as hyperflexion in five patients, hyperextension in three and unreported in one. The management has been by a dorsal approach in five, combined dorsal and volar approach in two, closed reduction in one and unreported in the other.[4–10]

Normally, dislocation of the MCPJ is prevented by both the shape and supporting structures surrounding the joint. The MCPJ is a condyloid joint whereby the metacarpal head is narrowed dorsally and flares in a volar direction giving increasing contact with the base of the proximal phalanx as the joint is flexed. The capsule of the MCPJ extends from the neck of the metacarpal to the base of the proximal phalanx but is strengthened on all sides. Dorsally, there is a loose insertion of the common extensor tendon. The volar surface of the joint capsule is supported by the volar plate, which has a thick fibrocartilaginous distal portion and a thin membranous proximal portion. The volar plates are linked laterally by the intervolar plate ligaments. The lateral margins are also reinforced by collateral ligaments, which are more taut in flexion than extension. The saggital bands of the palmar fascia and tendons of the intrinsic musculature of the hand provide further support. The MCPJ joint is thus most stable laterally in full flexion but allows abduction and adduction in extension.

Two theories have been proposed as to the mechanism of volar dislocation of the MCPJ. Renshaw and Louis (1973) proposed that hyperextension of the MCPJ produced interposition of the volar plate, between the metacarpal head and base of the proximal phalanx, after rupture of its proximal membranous portion thereby preventing closed reduction.[11] However, Wood and Dobyns (1981) proposed that hyperflexion of the MCPJ with simultaneous proximal displacement of the proximal phalanx occurred resulting in interposition of the dorsal capsule into the MCPJ preventing closed reduction.[5] Furthermore, they performed cadaveric studies in which they applied hyperflexion and hyperextension forces to the proximal phalanx. In 5 of 10 hyperflexion injuries they reproduced interposition of the dorsal capsule but no cases after a hyperextension injury out of 16 investigated digits.[5] It is probable that the actual mechanism is a combination of the two processes, as in this case the dorsal capsule was attenuated and scarred plus interposition of the volar plate requiring partial resection.

In this case, an initial closed reduction was performed with temporary success. In only one previous case has initial closed reduction proved successful without internal fixation eventually.[8] Open reduction is necessary because of soft tissue interposition, which in this case was attributable to the volar plate.

Previous reports recommend an initial dorsal approach to the MCPJ as soft tissue interposition usually occurs on the dorsal

aspect.[7][10] Occasionally, interposition of the volar plate occurs and cannot always be remedied via the dorsal approach. A subsequent volar approach is then necessary to achieve reduction.

From our experience and that of others, it is probable that simple closed reduction in the accident and emergency setting may prove difficult or impossible because of soft tissue obstruction. If reduction is unsuccessful, referral to the hand surgery service is required to release soft tissue interposition. A dorsal approach, based on the previous cadaveric studies, should be attempted first, before a volar approach if suspected that the volar plate is involved. Furthermore, even if reduction is successful, referral to the hand surgery service is recommended, because of the likelihood of recurrent dislocation resulting from stretching of the joint capsule and surrounding ligaments. In this scenario, internal fixation of the MCPJ will be required.

Contributors
Wee Leon Lam identified the rare nature of the case, initiated the case report and contributed to the writing of the case report. Aidan Fitzgerald took charge of researching and writing the case report. Geoffrey Hooper was the surgeon who operated on this case and continued the patient's management as an outpatient as well as editing the case report.

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