Clinical

Knee dislocation in touch rugby: a case study

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We present a case history of an injury which on initial presentation sounded reasonably innocuous, but on further examination proved to have catastrophic injuries and sequelae. This case demonstrates the importance of a thorough neuro-vascular examination and is relevant to Naval medical officers who will frequently provide pitch and ship borne medical cover for sports.

Case History

A 30 year old solicitor presents to Accident and Emergency with a swollen painful left knee after sustaining a valgus and anterior-posterior force onto his straight left leg whilst playing touch rugby. He gives a history of “dislocating” his knee with it popping back into joint afterwards. He was unsure if knee went posterior or laterally.

On Initial Examinations:
Tender swollen left knee, with difficulty examining ligaments due to pain but ligaments appear intact. Intact sensation distal to knee, but unable to palpate distal pulses.

Investigation:
X-ray NAD

Initial Diagnosis:
Patella Dislocation with ?vascular injury

Initial Plan:
Referred to Orthopaedics.

Subsequent Examination:
Painful swollen knee and calf, 2cm circumference difference left to right. No palpable pulses. Pain on passive extension of toes.

Diagnosis:
Dislocated left knee with Popliteal artery disruption and acute compartment syndrome. Listed for Immediate Surgery.

Initial Surgical Findings and Treatment:
Knee, complete disruption of all four ligaments, no bony injury. Intimal tear to popliteal artery and vein with large haemotoma around popliteal artery. Sciatic Nerve explored and found to be intact. Marked compartment syndrome all four compartments. Knee stabilised with external fixator, reverse saphenous graft and vascular repair performed. Four compartment fasciotomies.

Subsequent Medical Issues:
Rhabdomyolysis requiring HDU treatment.

Subsequent Surgical Intervention:
Delayed primary closure and split skin grafts to fasciotomy wounds. Delayed Left knee reconstruction.

Outcome:
Not able to play rugby but walking with minimal pain and stable knee.

Discussion

Knee dislocation is one of the most severe orthopaedic injuries, owing to the high incidence of neurovascular damage and subsequent limb ischaemia(1). The knee is a relatively stable joint, and as a result normally requires a significant degree of force in order for the tibia to become displaced; typically, at least three of the main ligaments must rupture for dislocation to occur(2). Causes therefore involve high impact injuries such as vehicle collisions, falls, blast injuries and high-impact sports injuries(2).

The case above is an example of a low velocity dislocation. Low-velocity dislocations are typically caused by sporting injuries(3), and result in lower rates of soft tissue and neurovascular damage(3). In contrast high-velocity dislocations result in vast damage to...
the knee anatomy, and are more likely to cause neurovascular damage(1). Typical presenting signs and symptoms include severe diffuse pain surrounding the joint, immobility, and instability of the joint. Those without reduction of the joint typically present with obvious deformity(1). However, knee dislocations can often self-reduce, making the true incidence of all dislocation difficult to evaluate, and increasing the likelihood of under-diagnosis of knee dislocation. However, they occur more rarely than most other knee injuries, with an incidence rate of around 1 in 150,000 estimated(4). The ratio of male to female injury is around 3:1, with the majority of patients presenting in their third decade of life(5).

Classification

Dislocations can be classified anatomically in terms of the displacement of the tibia with respect to the femur(6), or by the level of ligamentous involvement(2). According to the anatomical definition, dislocation can occur as an anterior, posterior, medial, lateral or rotary injury. Anterior dislocations account for approximately a third of all knee dislocations and result from hyperextension of the knee(8). Some sources believe the level of hyperextension must exceed 30° in order for a full dislocation to occur(6). In the majority of anterior dislocations, the posterior capsule tears, both the anterior cruciate ligament (ACL) and posterior cruciate ligament (PCL) are torn, and one of the cruciate ligaments is injured, depending on the angle of impact(8). Alternatively, the ACL may tear alongside the posterior capsule, whilst the PCL remains intact. The common mechanism for such injuries is motor vehicle collisions, or high impact sporting injuries from behind, forcing the tibia forward in relation to the femur(1).

In posterior dislocation, there is typically disruption of both cruciate ligaments, most commonly caused by strong force exerted onto the tibia with the knee flexed, resulting in the tibia being forced posterior to the femur(8). The mechanisms behind such injury include the anterosuperior tibia striking the dashboard of a car, or a runner falling on a flexed knee(3). Posterior dislocation accounts for around 25% of dislocations, with the remaining dislocations being lateral dislocation (accounting for around 13% of all dislocations), medial dislocation (3%) and rotary dislocation (4%) 8). These
dislocations require varus, valgus and rotary impact, respectively (see Figure 1). Rotary dislocations are further subdivided into anteromedial, anterolateral, posteromedial and posterolateral injuries(9).

Treatment
Following diagnosis of knee dislocation, the limb must be reduced, and immobilized in a posterior or medial/lateral splint(3). Nervous or vascular damage may not be immediately apparent, and so immediate investigation is integral. Angiography/arteriography were previously standard procedure in all cases of dislocation(10). However, if vascular injury is irrefutably apparent and viability is at immediate risk, surgery should be commenced immediately(11). Orthopaedic and vascular consultations are also integral to maintaining limb viability. Typically orthopaedic intervention is required if the joint shows evidence of instability following reduction(12). This usually involves fasciotomy, particularly if distal fracture of injury to venous vessels is apparent, or compartment syndrome is present(1).

Current research indicates that surgical repair of the knee ligaments is also indicated. Ideally, reconstruction as opposed to repair should be carried out, as these carry a slightly better prognosis in the long term(13). Postoperative physiotherapy and observations are also vital to optimise recovery(14). In most patients, immobilisation for around 6 weeks is sufficient(14). The patient must be assessed over an extended period of time, to ensure delayed compartment syndrome, post-traumatic arthritis or any other complications do not arise after the injury. Owing to the high level of trauma typically needed for knee dislocation to occur, other injuries such as fracture of the tibial or femoral condyles are likely to be evident, further complicating the rehabilitation process(2).

Complications

Popliteal Artery Trauma
In both anterior and posterior dislocations, approximately 30% of dislocations are associated with trauma to the popliteal artery, one of the key concerns regarding knee dislocation(15). The popliteal artery is held in place by both the adductor magnus and soleus, thus making it vulnerable to rupture by hyperextension of the joint(14). Vascular damage resulting from popliteal rupture can present in a variety of ways. In the mildest cases, popliteal pulses may be present, with normal limb and distal pulses evident(5). In such examples, the possibility of vascular trauma should not be excluded, as, following reduction, pulses may return while vascular injury persists. A delay in vasculature of over 6 hours has been found to severely reduce the viability of the limb: an amputation rate of approximately 85% has been reported for limbs with no repair to vascular injury within 8 hours of trauma(16). More commonly, the limb will appear obviously deformed, with some evidence of vascular injury, be it ischaemia, pallor or other symptoms of vascular injury associated with compartment syndrome(5). Venous injury and injury to small vessels must also be excluded(5) as trauma to such vessels can equally compromise the viability of the leg. The genicular branches of the popliteal artery alone cannot provide sufficient vascular support if popliteal artery rupture occurs, further highlighting the importance of vascular viability maintenance. Deep vein thrombosis is also a noted complication of knee dislocation, and should be excluded by performing a venogram(1).

Peroneal Nerve Injury
The peroneal nerve is damaged in around 20 to 40% of dislocations(8) and occurs due to severe stretching or traction to the nerve. Medial and rotary (posterolateral) dislocations most commonly lead to such damage, and prognosis regarding nervous function is poor (5). Peroneal nerve damage typically presents with paraesthesia of the dorsum of the foot and diminished dorsiflexion strength (17). It is often difficult to distinguish between limitations of movement due to pain and actual nerve injury.

If the nerve remains in continuity, around of 80% of patients will recover dorsiflexion(18), whereas nerve grafts due to frank rupture result in less than 50% of patients recovering palsy(18). It is estimated that around 60% of patients will resume an acceptable level of
activity following knee dislocation, whilst around 15% will result in a severe limitation with regard to activities of daily living, possibly requiring later arthrodesis(18). Around 77% of patients who sustain low-velocity dislocation go on to participate in sport of some form(1). Provided vasculature remains viable, prognosis is good, whereas peroneal damage carries a poorer prognosis. Therefore, preservation of vasculature and nervous supply through rapid surgical intervention is integral to a good prognosis.

**Ligament damage**
Disruption of the knee ligaments causes instability, lower limb dysfunction and an increase in the long-term risk of osteoarthritis(13). Controversy regarding the management of ligament disruption exists. Non-operative techniques are possible, such as immobilisation with plaster of Paris to provide stabilisation, although this is usually complicated by stiffness and loss of function(19).

Most authors now advocate surgical intervention as it restores the stability necessary for rehabilitation and increases the range of joint movement, when compared to non-operative methods(19,20,21). The use of external fixators or cross pins to secure the reduction have been recommended to prevent secondary subluxation and to protect neurovascular structures following popliteal artery repair(22,23). In young active patients, early ligament reconstruction within three weeks of injury using autogenous tendons is common amongst reports in the literature. This enables improved rehabilitation participation and an earlier return to activity(13,22). During recovery the knee should be immobilised in the opposite direction to that in which dislocation occurred to prevent subluxation(23).

**Compartment Syndrome**
The muscles and associated neurovascular structures of the leg are arranged in discrete compartments, with each compartment enclosed by a fascial sheath. Compartment syndrome occurs when perfusion pressure falls, relative to that of the intracompartmental pressures, and was first described by Volkmaan in 1872(24). Correct diagnosis is necessary to prevent the potential sequela of rhabdomyolysis, renal failure, permanent sensory and motor dysfunction, amputation, or even death(25). Although a paucity in the literature appears to exist regarding the incidence of compartment syndrome following knee dislocation, the incidence in soft-tissue and popliteal artery injuries is reported as between 23 and 50% of cases and so compartment syndrome should be considered in those presenting with knee dislocation(26).

**Causes and Symptoms**
Compartment syndrome can be caused by fracture, blunt trauma, and reperfusion following arterial obstruction(27), and the cause can be placed under two subheadings; those which decrease compartment size, and those which increase intracompartmental contents. Causes of decreased compartment size include dressings, plaster casts, external compression, and thermal injuries, whilst causes of increased compartment content include haemorrhage, oedema and increased capillary permeability following trauma or reperfusion.

The classical symptoms of compartment syndrome include pain disproportionate to that expected in the situation, muscle weakness, hyperaesthesia in the relevant nerve distribution, tense compartment fasciae, and pain on passive stretching(35). Ideally, compartmental pressures should be measured to both facilitate diagnosis and to enable future comparisons to be made to these baseline measurements(26). Normal pressures are 10-15 mmHg but can rise to 60 mmHg following injury or fracture(30). A difference between diastolic blood pressure and compartment pressure of less than 30 mmHg indicates compartment syndrome(35). However, a high index of clinical suspicion(31) can enable the early recognition of symptoms and subsequent treatment, both of which are the ultimate aims in the care of compartment syndrome(32).

**Treatment**
Any dressings surrounding the limb should be removed and the leg lowered to the level of the
heart, thus ensuring any hypotensive effect is not due to limb elevation(32). The main treatment is surgical fasciotomy with all four compartments of the leg opened and decompressed, which can be achieved with either two separate incisions on the anterolateral and posteromedial sides, or one lateral incision(27). In cases where repair to damaged vascular structures is necessary, prophylactic fasciotomy is advocated to prevent compartment syndrome occurring due to oedema, secondary to the restoration of circulation(16). Complications of fasciotomy include muscle herniation, soft-tissue tethering, sensory changes and recurrent ulceration(25).

Rhabdomyolysis
Rhabdomyolysis describes the breakdown of muscle cells and the subsequent release of contents, most notably myoglobin and potassium, the causes of which include trauma, vessel occlusion, compartment syndrome, and crush injury(28). Large volumes of fluid can be sequestered from damaged muscle cells resulting in hypovolaemia, hypernatraemia, hyperkalaemia, and pre-renal failure(29). The substantial release of myoglobin causes renal tubule obstruction leading to acute renal failure (ARF)(28), and the link between traumatic crush injuries and ARF was first noted during the London bombings of World War II(33).

Diagnosis and Treatment
A five-fold increase in creatinine kinase in the absence of myocardial infarction, tea coloured urine, and a positive urine dipstick test for haem all indicate rhabdomyolysis(34). Rapid replacement of fluid within six hours is advocated to help prevent volume depletion, acidosis and ARF, and 12-14L of fluid may be required. An alkaline fluid regimen should be commenced, with 50mmol of bicarbonate added to 1L of hypotonic saline, and in crush victims fluid resuscitation should be started prior to extrication(29). Where adequate volumes of urine are passed (>20ml / hour), 50ml of 20% mannitol, an osmotic diuretic, can be added to each litre of fluid(29). This serves to increase renal blood flow, increase glomeral filtration rate to prevent blockage of renal tubules, and move fluid from the interstitial space by osmosis, thus reversing hypovolaemia(28). Temporary control of hyperkalaemia can be achieved with hypertonic glucose or bicarbonate, or indefinitely with dialysis. Dialysis is necessary in established ARF, or in cases of severe hyperkalaemia and acidosis(28).

Conclusion
This case demonstrates the importance of a thorough neuro-vascular examination in patients presenting with what initially could be interpreted as a patella dislocation. If treatment had been delayed in this case it could have resulted in amputation of the affected limb. In an acutely swollen knee, a thorough orthopaedic examination of the knee is difficult and the only indication of a severe injury may be neurovascular compromise.

Royal Naval Medical Officers providing pitch side and “ship side” cover to sportsmen should ensure that they have a high level of suspicion for severe injuries that whilst uncommon can be catastrophic if missed.

References