On clinical examination of volar PIPJ injuries, the joint appears flexed with the distal and the middle phalanges deviated to one side. A true lateral radiograph is diagnostic demonstrating an oblique orientation of the middle phalanx and lateral view of the proximal phalanx. Resistance to passive flexion and extension may exist and therefore, careful evaluation of the joint is important to spot the difference between active and passive range of movements, which, in a reduced finger, might indicate loss of central slip function. It is important to determine if the lesion is acute or chronic since the age of the injury will guide the management.

Acute injuries do well if recognised and treated early (fig 4). Late reconstruction of these injuries is technically difficult and often unrewarding. The treatment results are often poor and are particularly related to failure in recognising the rotatory component of the injury and subsequently estimating the degree of instability and the extent of the soft tissue damage.

The complication of these injuries is loss of movements, a chronic laxity, pain, swelling, deformity, and a fixed flexion contracture is common when aggressive attempts to achieve full extension are not undertaken.12

Most cases in the literature are isolated reports.14 15 This itself speaks of the rarity of this condition. We report three cases of similar injuries.

All the reported cases were sent home from the A&E. They returned within a week to the hand clinic with a deformed tender swollen finger, two of them returned, unable to bear the pain. As these are extremely rare injuries a good clinical history and examination is necessary. It may not always be possible to acquire the detailed mechanism of injury especially in a busy A&E department. Clinical examination in an acute painful situation needs a lot of patience and perseverance to locate the exact cause and nature of a closed finger injury. Severe apprehension and lack of cooperation from the patient in an acute setting might also be responsible for these injuries to be missed initially. All these cases required open reduction. Early referral, within the first week, to the hand clinic is essential in a suspected case. If diagnosed late and therefore treated late the final outcome and function is compromised.

We believe that it is extremely important to be aware of this injury. Any passive painful flexion or extension of the finger PIPJ should raise a suspicion. High index of suspicion, awareness and early referral to a hand clinic, subsequent repair and rehabilitation gives a good functional outcome.

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Low cost, high risk: accidental nutmeg intoxication

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Nutmeg poisoning is rare but probably underreported and should be considered in recreational substance users with acute psychotic symptoms as well as central nervous system neuromodulatory signs that may mimic in part an anticholinergic hyperstimulation.

We report an accidental intoxication during voluntary ingestion of nutmeg. This occurred with a newly reported method for preparation of the mixture during an attempt for a low cost alternative to recreational drug use. Because of a recent second fatality with such misuse, nearly a century after the first one, and because of the suspected underreporting of the incidence of such episodes, the symptomatology and aetiology of myristicin intoxication is reviewed.

CASE REPORT

A previously well 18 year old student presented with complaints of palpitations, drowsiness, nausea, dizziness, thirst, and dry mouth. She was very anxious, restless, and agitated and described being “in a trance state”. She specifically felt “like Jack in the box wanting to get out” but did not have hallucinations. She did not complain of urinary or abdominal discomfort and gave no history of
seizures or migraine. She had an unremarkable medical and psychiatric history and denied any suicidal ideation. The patient refused to give any information regarding recreational drug use.

On examination the patient was agitated but alert, flushed but apyrexial, with a respiratory rate of 20/minute and saturation of 96% on room air. She was tachycardic at 102/minute with a blood pressure of 105/68. Cardiopulmonary examination was unremarkable. The abdomen was soft and non-tender. Cranial nerves were normal, while peripheral nervous system examination showed brisk, symmetrical deep tendon reflexes. There was no neck stiffness. Pupils were dilated to size 4 mm and were symmetrically brisk to light and accommodation.

A 12 lead ECG showed a fast sinus arrhythmia (rate 95–110/minute) with no ischaemic or hypertrophic changes. Serum urea, electrolytes, liver transaminases, full blood count, and urgent catecholamines were normal. Serum and urine toxicology screens were negative.

In view of the complexity of her condition she was admitted and five hours later admitted to having taken a large dose of nutmeg while trying to “get high”. Some 50 g of commercially available grated nutmeg were blended into a milkshake, the patient drinking three quarters of the amount. A feeling of elation was experienced by at least two people, but in our patient this was followed by her presenting symptoms 30 minutes after ingestion.

The patient was kept for observation, offered reassurance, and rehydraion. After symptoms had resolved she was allowed to return home 10 hours after presentation, 16 hours after ingestion.

DISCUSSION

Nutmeg intoxication has scantily been reported in the literature. Such reports span the period between 1908 and present day, although some claim that the first reported evidence of its effects was reported by Lobelius in 1576. Most cases concern attempts to achieve an euphoric and hallucinogenic state at low cost. Previous reports include ingestion of grated or whole nutmegs, mixing of the spice with coffee, vodka or other alcohol, and concurrent use with cannabis. This time the method reported of blending within a milkshake, the patient drinking three quarters of the amount.

Although misuse is voluntary, intoxication is invariably accidental. There have been two fatal “overdoses” in the medical literature. The first was reported in the beginning of the 20th century involving about 14 g in an 8 year old. The second was reported in the beginning of the 21st century and involved a 55 year old. Such a risk a century on is a timely reminder of the importance of this differential diagnosis when others have been excluded and when the patient could fit into an exposure category. Our patient was a higher education student who visited London for an entertainment weekend. As it was admitted, a group of people had tried and recommended a “nutmeg high”. As such, the incidence of its misuse, especially in thrill seeking but low cost living students, is underreported and the possibility of intoxication must always be entertained.

How does nutmeg exert its effects? The active substance is myristicin, the volatile oil of the spice comprising a mixture of allylbenzene derivatives and terpines. Myristicin has a weak monoamine oxidase inhibitor action and with elemicin may be metabolised to an amphetamine-like compound with hallucinogenic effects similar to lysergic acid diethylamide. Other components of myristicin (linalool, safrol, isoeugenol, and eugenol) are structurally similar to serotonin agonists that may explain the cardiovascular response. The anxiogenic effect encountered in the feeling of impending doom has been linked to the serotonergic and GABAergic activity found in trimyristin, an extract of the Myristica fragrans seeds.

Symptoms predominantly involve the central nervous and cardiovascular systems. In the former one may experience anxiety, fear, and a feeling of impending doom. Acute psychotic episodes, detachment from reality, like with “Jack in the box”, may occur as can visual hallucinations, taking the form of time, colour, or space distortions. Patients may be hostile, combative, and agitated. Chronic psychosis with prolonged use has been reported. Cardiovascular manifestations include tachycardia, which may be the only finding on examination, palpitations, hypertension, and rarely hypotension and shock.

Further symptoms are dry mouth, facial flushing, nausea, unsteadiness, epigastric pain, urinary retention, and blurred vision. Although it has been reported that a useful differentiating sign from anticholinergic intoxication such as belladonna alkaloids or atropine poisoning is that myristicin may cause miosis and not mydriasis, there is no conclusive evidence. In a review studying pupillary responses specifically, similar numbers of patients had dilated, constricted, or even unaffected pupils. While early miosis may be followed after a few hours by mydriasis, in some cases miosis was persistent 13 hours after ingestion whereas mydriasis was present from five hours. Experiments on cats failed to produce a local mydriatic action. Pupillary signs therefore are not necessarily reliable in the diagnosis of nutmeg poisoning.

Symptoms usually appear three to eight hours after ingestion and resolve within a day or two. Treatment is mainly supportive, although cases have been admitted to intensive care units, and should include cardiorespiratory monitoring for at least eight hours after ingestion (personal communication, Guy’s Hospital Medical Toxicology Unit). The patient needs regular reassurance in view of the conquering feelings of anxiety, fear, and impending calamity. Sedation with benzodiazepines can be used to calm the patient and help reverse the amphetamine-like effects. Charcoal may help decrease systemic absorption; induction of emesis is controversial.

Although the risks of nutmeg intoxication after voluntary use are not unknown to the medical community, certain groups of the population are still likely to experiment for low cost recreational drug alternatives. The presentation of acute psychotic symptoms accompanied by central nervous system neuromodulatory signs should alert the physician to this rare but probably underreported possibility especially in urban areas known to attract recreational substance users.

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Atlas: standard diagnostic tests for an unusual fracture
S Leigh-Smith, R Price, D Summers

The case is reported of an unusual atlas fracture with no reports of such an injury in the literature. The diagnosis of this injury emphasises the importance of simple clinical decision instruments, and systematic interpretation of investigations.

A 28 year old man who was 5 feet 9 inches and 16 stone was playing rugby as a number 8. He ran head first into a tackle, causing an axial compression injury to his neck. This caused immediate, dull pain over the whole of his neck. He attempted to continue playing but found that merely running exacerbated the pain considerably. He later noticed the pain localising to the whole axial area along with his head “feeling heavy and loose”. He self treated with a soft collar for two days, before presenting to the accident and emergency (A&E) department, by which time the pain was persistent in the sub-occipital area. At no stage did he have any neurological symptoms.

Examination showed painful neck movements, with pronounced reduction of range in all directions.

Cervical spine radiography showed considerable retropharyngeal soft tissue swelling in the upper cervical spine (fig 1). The AP dens view showed subtle asymmetry of the atlantoaxial joints, with minor lateral displacement of the C1 lateral mass on the right (fig 2).

His neck was immobilised, and he was transferred to the regional neurosurgical service. Review of the cervical spine radiograph showed an unusual vertical lucency projected over the dens, and computed tomography of this area was performed (fig 3). The appearances suggested a congenital midline cleft atlas, with diastasis of the anterior spondylolisthesis.

The patient’s neck was immobilised in an Aspen collar cervical orthosis. Ten days after the injury, he was brought back for flexion and extension radiographs of his neck, which showed no abnormal movement. He remained neurologically intact and external bracing was continued for eight weeks. Follow up computed tomography showed persistence of the C1 cleft but resolution of the retropharyngeal swelling. The flexion and extension views were repeated and no abnormal movement was detected. Physiotherapy was started to rebuild his range of neck movements and treat residual discomfort. Given the developmental anomaly of C1, he was advised to stop taking part in contact sports.

DISCUSSION
Screening for potentially unstable cervical spine injury starts with the use of a proven clinical decision instrument to determine who needs a cervical spine radiograph. If radiography is required the ATLS system of cervical spine radiological interpretation or similar should be used.

In this case the patient clearly had a suggestive history and abnormal examination requiring radiography. The