CASE REPORTS

# Intrathoracic migration of a Kirschner wire

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When Kirschner wires are used in fixation of a dislocated sternoclavicular joint, disappearance of a wire beneath the skin demands urgent attention.

## CASE HISTORY

A motorcyclist aged 36 was involved in a road traffic accident and sustained a grade III anterior dislocation of the right clavicle at the sternoclavicular joint. He underwent open reduction and internal fixation with two straight Kirschner wires (K-wires). For three weeks after fixation, serial chest radiographs confirmed satisfactory reduction with the K-wires in a good position. In the fourth week, he sought advice from the accident and emergency department because he could see only one of the K-wires over his clavicle. He was symptom-free but chest X-rays and CT scans indicated that one of the K-wires had migrated to the anterior mediastinum (Figures 1 and 2). A median sternotomy was performed and the K-wire was easily removed. It had passed anterior to the brachiocephalic vein and headed inferiorly, with its tip coming to lie adjacent to and abutting the aortic arch. There was no active bleeding, but a small tear in the adventia of the aorta was repaired with 4/0 prolene. The patient recovered without incident.

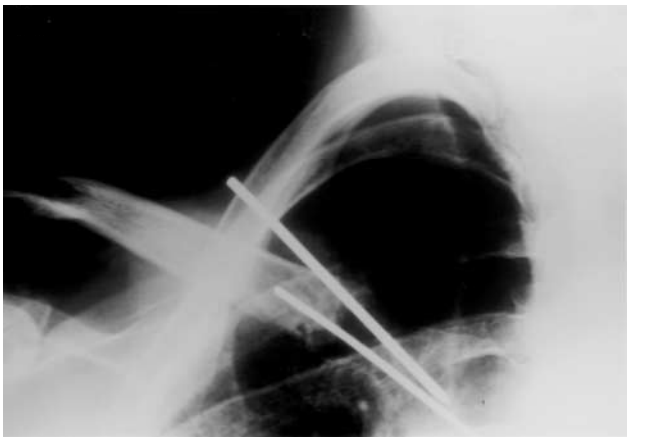

*Figure 1* **Postero-anterior chest radiograph showing intrathoracic migration of one of the K-wires now lying in the anterior mediastinum**

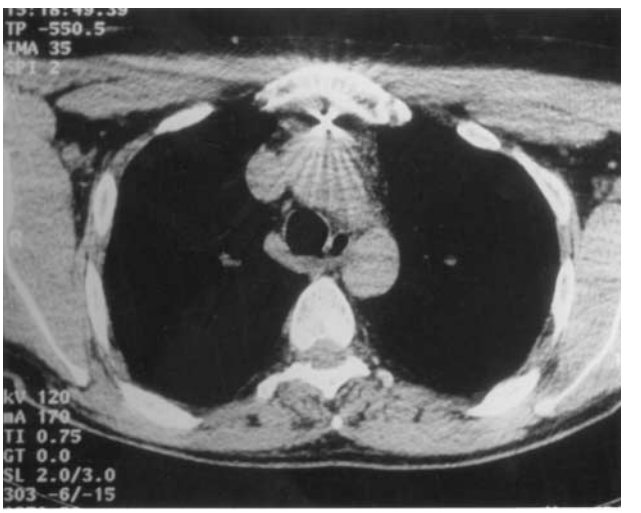


*Figure 2* **CT scan of chest showing K-wire in anterior mediastinum adjacent to aortic arch**

## COMMENT

Metallic orthopaedic fixation devices (pins and wires) have been used since the 1930s, but the incidence of migration is unknown. Reviewing all reported cases from 1943 to 1981 Lyons and Rockwood[1] found 47 instances of wire migration, with the wire migrating to a major vascular structure in 17. It is noteworthy that, in 21 of the 47, the wire had been used for internal fixation of a dislocated sternoclavicular joint.

Why should K-wires, used in this way, migrate into the thorax? Theories include muscle action, the great freedom of movement of the shoulder, negative intrathoracic pressures associated with respiration, regional resorption of bone, gravitational force and even capillary action[1,2]. Migration of K-wires has been reported as early as the day after fixation and as late as 21 years. Usually the process causes no symptoms, as here. Intrathoracic migration of wires or pins to heart[3], lung[4], aorta[1] and almost every other intrathoracic blood vessel has been reported. They can also migrate elsewhere—for example, to the spleen (which a K-wire reached from the right shoulder within 12 hours)[5] and the spinal canal[6]. The type of wire, smooth, threaded or bent, seems not to be a factor in incidence.

The most important step in preventing this potentially lethal complication is to bend the exposed part of the wire/pin after fixation. However, this does not guarantee safety since wires sometimes migrate after breakage[2]. Close clinical follow-up with frequent postoperative radiographs has been recommended, but in the case here routine radiographs showed no sign of migration in the three weeks after fixation—i.e. it can happen quickly. In this case the exposed part of the wire had not been bent.

Once intrathoracic migration of a K-wire is recognized, urgent removal is mandatory. Fatalities from wire migration

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have all been related to catastrophic cardiovascular events, and one patient died while awaiting elective thoracotomy. A CT scan of the chest will help determine the surgical approach. Sternotomy is preferable to thoracotomy in patients with suspected cardiac or intrapericardial vascular injury[3]. Video-assisted thoracoscopy may also have a role, though experience has been mixed and an open procedure has often proved necessary[7].

Since there is an increasing trend towards internal fixation of dislocated sternoclavicular joints, surgeons and others need to be aware of this potentially lethal complication.

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# Renal cell carcinoma in pregnancy


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Although rare in women of childbearing age, renal cell carcinoma is the commonest renal neoplasm occurring in pregnancy[1] and is potentially curable with prompt diagnosis and treatment. However, the symptoms can mimic more common disorders such as urinary tract infection.


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## CASE HISTORIES

### Case 1

A woman aged 28, gravida 2, para 1, was referred for evaluation of a right renal mass at fourteen weeks' gestation. She had no previous history of hypertension, renal disease, or pre-eclampsia. The mass had been detected after onset of right flank and loin pain, and hypertension of 150/90 mmHg, at twelve weeks' gestation. Urinalysis, urea, electrolytes and creatinine were normal. Urine cytology was negative. Renal ultrasonography revealed a $10 \times 6.5 \times 7.4$ cm solid mass with heterogeneous echotexture arising from the lower pole of the right kidney.

After extensive counselling and consultation with obstetric staff, it was decided that she should undergo a right radical nephrectomy, which was performed at seventeen weeks' gestation. She received standard perioperative antibiotic prophylaxis, and in addition low-molecular-weight heparin and a twenty-four-hour infusion of the tocolytic $\beta$-agonist ritodrine. Postoperatively she recovered without incident and was discharged on day seven. The remainder of the pregnancy was uneventful. She was induced at forty weeks' gestation for worsening hypertension, and gave birth to a healthy boy weighing 2.5 kg.

On histopathological examination the surgical specimen contained a grade II clear-cell renal carcinoma, with a maximum diameter of 9 cm. Pathological staging was T2 N0 Mx. The patient remains well with no evidence of recurrence at twelve months' follow-up.

### Case 2

A woman of 24, gravida 1, para 0, was referred at twenty-three weeks' gestation for assessment of episodic haematuria. She had no relevant medical history. Microscopic haematuria had been detected at her antenatal booking visit at twelve weeks' gestation. Microbiological culture was negative at that time and no further evaluation was performed until twenty-four weeks' gestation when she was referred with right loin pain and episodic frank haematuria. A full blood count and urea, creatinine and electrolytes were normal. Renal ultrasonography revealed a well-defined 5 cm solid mass arising from the medial aspect of the mid to lower part of the right kidney. An ultrasound-guided biopsy was taken but the cytopathological appearances did not allow certain differentiation between an oncocytoma and a well-differentiated renal cell carcinoma.

The urine contained atypical epithelial cells consistent with renal cell carcinoma. Further evaluation by magnetic resonance imaging confirmed the presence of a mass with an intermediate signal and slight heterogeneity. There was no evidence of surrounding soft tissue invasion or of retroperitoneal lymph node enlargement.

After further obstetric consultation, she underwent a right radical nephrectomy at twenty-five weeks' gestation. Preoperatively she received prophylactic corticosteroids to reduce the likelihood of neonatal respiratory distress syndrome, should she deliver before term. She also received indometacin as a tocolytic agent. Initial post-operative recovery was uneventful; however, she did experience symptoms suggestive of preterm labour on the sixth postoperative day and was treated with an infusion of ritodrine for twenty-four hours. Her symptoms settled and she was discharged. She subsequently underwent induction of labour at forty weeks' gestation and a 3.3 kg boy was delivered by emergency caesarean section for fetal distress.

Histopathological examination revealed a grade I renal cell carcinoma. There was no invasion of the renal capsule. There has been no evidence of recurrence at 5 years' follow-up.

## COMMENT

Although renal cell carcinoma accounts for 3% of all adult malignancies, it is rare in women of childbearing age. To date there are reports of about fifty cases diagnosed during pregnancy: it is the most common renal neoplasm reported in pregnancy, accounting for half of all primary tumours[1]. Sex and age matched data do not exist to allow comparison of the relative proportion occurring outside of pregnancy, although in general terms there is no evidence of an increased incidence of malignant neoplasms in pregnancy[2].

Pregnancy and cancer are the only two biological conditions in which antigenic tissue is tolerated by a normally functioning immune system. However, there have been no demonstrable immunodeficiencies in pregnancy to antigens carried by tumour cells[3]. Furthermore, in most cases the biological behaviour of malignancy is not influenced by pregnancy, with the possible exception of a small subgroup of hormonally sensitive malignant melanomas[4]. With breast cancer, although it seems to present at more advanced stages in pregnancy, the prognosis of each stage is similar to that in non-pregnant women[5].

In 1986, Walker and Knight[1] reviewed the presentation of renal cell carcinoma during pregnancy and found that the commonest presenting symptoms of such tumours were a palpable mass (88%) and pain (50%). Haematuria and hypertension accounted for 47% and 18% of cases, respectively. A subsequent review has suggested that there has been a change in the presentation of renal cell carcinoma in pregnant women, with diagnosis now more frequently made incidentally during ultrasound examination performed for other reasons[6]. In addition, urinary tract symptoms are experienced by many pregnant women and are often due to non-neoplastic causes, such as calculi or urinary tract infection. Similarly, hypertension, especially during the third trimester, is often induced by pregnancy and related to pre-eclampsia. This means that renal cell carcinoma may not be considered as a potential cause for such symptoms, thus leading to delay in diagnosis and treatment.

Diagnostic evaluation of the pregnant patient with possible renal carcinoma requires special consideration of non-invasive techniques and as little radiation exposure as possible to mother and fetus. As a first step, urine should be sent for cytological analysis. In non-pregnant patients intravenous pyelography (IVP) and abdominal CT are the modalities frequently employed in the evaluation of renal tumours, but there is no proven safe threshold dose of radiation exposure to the fetus[7]. Abdominal ultrasound along with magnetic resonance imaging (MRI) can adequately identify, differentiate between, and stage solid renal masses in most cases, and with their avoidance of radiation exposure to the fetus these are the investigations of choice.

Indeed, ultrasound has a similar sensitivity (85%) to that of IVP and CT for renal masses greater than 3 cm, and is much more sensitive than IVP for renal masses between 2 and 3 cm (82% *v* 52%)[8]. Renal radionucleotide scans, being associated with less radiation exposure than routine X-ray films, have been used to determine function of the contralateral kidney and intrinsic activity. They do, however, pose additional risks to the fetus from the passage of radiopharmaceuticals and contrast agents across the placenta. As an alternative, doppler assessment of the contralateral kidney may be employed.

There are several issues to consider when treating a pregnant woman with a solid renal mass that is suspicious for malignancy. First, the clinician's primary responsibility is to the mother, though management must take into account her wishes regarding the welfare of the fetus. Secondly, such cases should be managed in a multidisciplinary setting involving urologists, obstetricians, neonatologists, radiologists, histopathologists and oncologists. Thirdly, the standard surgical treatment of most stages of renal cell carcinoma is a radical nephrectomy, involving the *en bloc* removal of the entire kidney and perinephric fat within Gerota's fascia. This has been performed via both transperitoneal and extraperitoneal approaches. The small numbers of

reported cases of renal cell carcinoma in pregnancy allow few conclusions to be drawn regarding the outcomes of each. However, avoidance of disruption of the peritoneal cavity in the extraperitoneal approach may theoretically be associated with less uterine irritation and in turn fewer obstetric complications, including preterm labour.

The timing of surgery is guided by the biological behaviour of such tumours and neonatal survival rates for different gestations. The doubling time of a renal cell carcinoma is estimated at 300 days[9]. Certain considerations should be made when embarking on surgery in pregnant women. Non-obstetric surgery is performed in approximately 1 in 500 pregnancies[10]. When surgery is performed during the first trimester there is an increased risk of preterm labour with its associated problems of fetal prematurity. However, with the improvements in neonatal care, fetal survival rates continue to increase and although actual figures vary between different neonatal units, neonatal survival rates of over 90% can be expected beyond twenty-eight weeks' gestation. Therefore, it is recommended that surgery should not be delayed in the first and third trimesters. However, if a mass is diagnosed in the second trimester then it is reasonable to wait until fetal viability before proceeding to surgery.

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# Meige's syndrome in dementia with Lewy bodies


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A complicating factor in the management of dementia with Lewy bodies (DLB) is the profound sensitivity of patients to antipsychotic treatment[1], which in turn may initiate or enhance movement disorders. Florid psychotic symptoms may, however, develop before DLB can be diagnosed.

## CASE HISTORY

A woman aged 60 came to psychiatric attention for the first time when referred for advice on depression and morbid jealousy which had existed for many months. She had also experienced hallucinations and misidentification delusions. Initially she was treated with an antipsychotic (trifluoperazine 2 mg twice daily) and an antidepressant (amitriptyline 125 mg daily). Over the next few months there were repeated episodes of falling and syncope with fluctuating confusion, and a drop in her Mini-Mental State examination score indicated rapid deterioration in her cognitive abilities. Therefore, trifluoperazine was replaced with the newer atypical antipsychotic olanzapine, 5 mg daily. The results of all investigations were normal except for a CT scan of the brain, which showed moderate cerebral atrophy. Dementia with Lewy bodies was diagnosed. Although the depressive and psychotic symptoms had been improved by treatment, this benefit was overshadowed a few months later by the development of persistent involuntary bilateral blinking resulting from repetitive and prolonged contractions of the orbicularis oculi muscles (blepharospasm). Soon afterwards she began to experience an oromandibular dystonia characterized by spasms of the lower face muscles, especially those surrounding the mouth. The choreoathetotic movements seen in tardive dyskinesia were absent, and there was no known family history of movement disorders or Huntington's chorea. Although the antipsychotic medication had been stopped after development of the


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dystonia, the symptoms gradually worsened and became very distressing. In addition to dementia with Lewy bodies, she was deemed to have Meige's syndrome.

## COMMENT

Meige's syndrome is a segmental cranial dystonia that occurs in primary or secondary forms[2]. There is some evidence for involvement of the basal ganglia along with an imbalance in the dopaminergic system[3].

Up to 25% of brains in Meige's syndrome show Lewy bodies, and some workers have proposed that the syndrome is part of the clinical spectrum of Lewy body disease[4]. Indeed, very occasionally Meige's syndrome has been reported to precede the development of Parkinson's disease[5]. Secondary Meige's syndrome, however, is not restricted to those with primary neurodegenerative disorders since it has also been diagnosed in patients receiving antipsychotic medications[6]. DLB patients, who are known to be highly sensitive to these dopamine receptor blockers, may be particularly at risk of developing secondary Meige's syndrome, since they are already prone to various parkinsonian and movement disorders. Although newer atypical antipsychotics such as olanzapine and risperidone seem less apt to cause movement disorders and better tolerated in elderly patients, DLB patients may still be vulnerable to their effects. Our patient was receiving an atypical antipsychotic immediately before developing Meige's syndrome.

Meige's syndrome has been earlier reported in patients with parkinsonism, but to our knowledge this is the first case specific to DLB. In such patients, persistent involuntary blinking of the eyes (the commonest initial presentation of Meige's syndrome) is an ominous sign that should lead to early withdrawal of the offending agent. The spectrum of Lewy body disease should also be considered in individuals with persistent involuntary blinking even if they are not receiving a dopamine receptor blocker.

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# Colonic endometriosis or adenoma?


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Colonic mucosal involvement with endometriosis can result in gastrointestinal bleeding[1]. The mucosal abnormalities associated with colonic endometriosis may be difficult or impossible to identify at colonoscopy[2].

## CASE HISTORY

A woman age 37 was referred for investigation of bright red rectal bleeding of seven months' duration and usually coinciding with menstruation. Pelvic endometriosis had been diagnosed 5 years earlier and had been treated with the oral contraceptive pill and laparoscopic laser ablation. Cleft palate and hare lip, uterus didelphus and septate vagina had all been surgically corrected. On examination, tender masses were palpable in both iliac fossae. Abdominal MRI scans suggested endometriotic cysts arising from both ovaries. Colonoscopy revealed mild mucosal inflammation at 35 cm from the anal verge, and a polypoid lesion 3 cm in diameter was seen within the ascending colon 65 cm from the anal verge. On histological examination, biopsies from the 'inflamed' area of distal colon showed 'inflammatory changes', while two separate biopsies from the polypoid lesion in the ascending colon showed fragments with mixed features of an inflammatory polyp and adenomatous glands: areas of low-grade dysplasia were surrounded by inflamed fibrotic stroma with focal surface ulceration and granulation tissue (Figure 1). The clinical diagnosis was distal colonic endometriosis and a proximal adenomatous polyp. The caecal adenomatous lesion could not be removed colonoscopically because of its size and location. At laparotomy there was a diffuse mass involving the posterior caecal wall, adjacent retroperitoneal muscles, right ovary and right fallopian tube. After joint intra-abdominal examination by colorectal and gynaecological surgeons, it was concluded that these findings were not typical of endometriosis. Typical endometriotic appearances were seen in the left ovary and on the serosa of the adjacent


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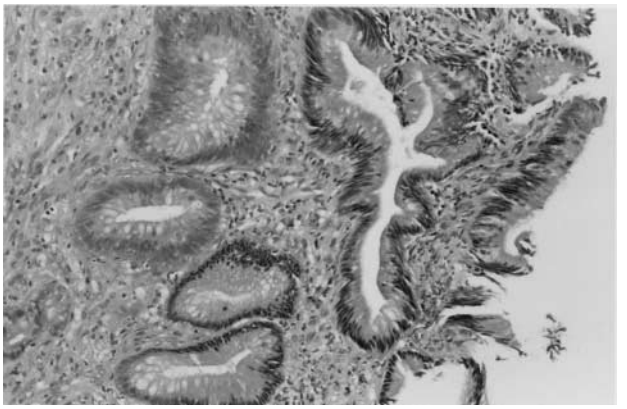

*Figure 1* **Colonoscopic biopsy showing part of the polypoid lesion.** Several glands with low grade dysplasia are surrounded by inflamed fibrotic stroma (haematoxylin and eosin × 200)

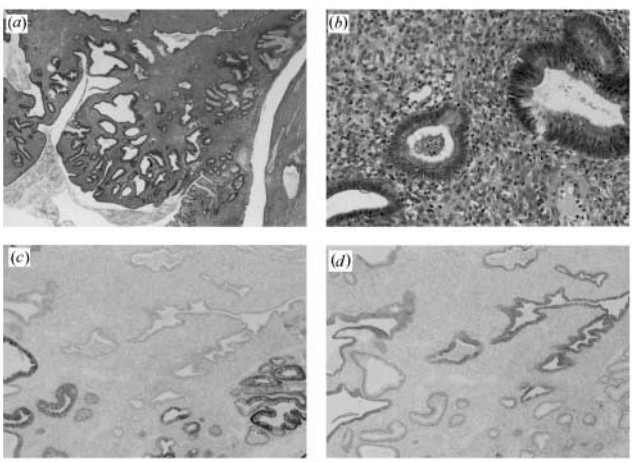


*Figure 2* **Histological findings in resection specimen.** (*a*) Haematoxylin and eosin stain, low-power view of inflammatory polypoid mass (× 20); (*b*) higher power view of colonic 'adenomatous' gland on right with adjacent endometrial gland on left (× 200); (*c*) colonic glands staining CK20 positive and CK7 negative (× 40); (*d*) same view as (*c*) but showing endometrial glands staining CK7 positive and CK20 negative (× 40)

sigmoid colon. Frozen section histological examination did not identify the nature of the mass, which was resected *en bloc* with ileocolic anastomosis. On histological examination the polypoid lesion removed from the ascending colon contained adenomatous glands with low grade dysplasia admixed with numerous foci of endometriosis resulting in marked expansion of the polypoid fronds (Figure 2 *a* and *b*). Immunohistochemical staining clearly differentiated between colonic glands (cytokeratin 20 positive, cytokeratin 7 negative) and endometrial glands (cytokeratin 7 positive, cytokeratin 20 negative, Figure 2 *b* and *c*). Sections from the retroperitoneal mass showed endometriosis. The patient has had no further colonic bleeding during 25 months' follow-up.

## COMMENT

Colonic endometriosis can mimic colorectal cancer by producing an invasive abdominal mass associated with abdominal pain, bleeding[1] or an ulcer[2,3], but 'adenomatous' change within colonic mucosa does not seem to have been reported previously. The present case suggests that colonic mucosal endometriosis can produce reactive changes in colonic glands, with cellular atypia and glandular hyperplasia mimicking dysplasia, thus making the lesions harder to discriminate from a neoplastic adenoma. It is possible that the 'adenomatous' lesion in this case resulted from invasion of a pre-existing neoplastic adenoma by endometriosis, but this is unlikely in view of the patient's young age and the absence of a history of colonic adenomas in the patient or her family. Although the colonic lesion in the present case was more fronded than a typical neoplastic adenoma, it would be difficult to use this appearance at colonoscopy to differentiate between the two conditions, since surface irregularity is also associated with malignant transformation within neoplastic adenomas.

Although endometriosis can mimic all stages of colorectal neoplasia, discrimination between colonic glands and endometrial glands is possible with cytokeratin immunostaining.

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# Subclinical type 1 diabetes

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Type 1 diabetes typically presents with acute osmotic symptoms or diabetic decompensation. It is seldom diagnosed solely as the result of a blood test.

## CASE HISTORY

A woman of 23 consulted her general practitioner because of 'flu-like symptoms, sore throat and lethargy. There was a

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*Table 1* **Glucose tolerance test results**

| | *Fasting glucose (mmol/L)* | *2 hour glucose (mmol/L)* |
|---|---|---|
| Test 1 | 5.6 | 12.3 |
| Test 2 | 5.8 | 13.1 |

strong family history of type 1 diabetes (including a brother and a first cousin) and her mother had checked her blood glucose with a meter. The reading was 13.4 mmol/L 2 hours after food. On examination her temperature was 37.4 °C and she had mild tender submandibular lymphadenopathy and tonsillar erythema. She was slim, with a body mass index of 23.

She had never experienced any osmotic or other symptoms suggestive of diabetes. In the fasting state, urine was negative for glucose and ketones and plasma glucose was 5.9 mmol/L. Further blood tests showed a normal full blood count, including differential, and normal renal, liver and thyroid function. A monospot test was positive, and infectious mononucleosis was thought the likely cause of her symptoms. She was advised to rest, and to test her blood sugar occasionally, fasting and 2 hours after meals. Postprandial hyperglycaemia continued with blood sugars on her meter ranging from 9 to 14 mmol/L. Urine remained negative for ketones and glucose. An oral glucose tolerance test (OGTT) pointed to diabetes and she was referred to the hospital diabetes clinic. Blood sugars continued at 4–7 mmol/L before meals and up to 14 mmol/L postprandially. She was now symptom-free, having recovered from her bout of infectious mononucleosis. There was no evidence of diabetic complications. Glycated haemoglobin indicated good control at 6.7%. Glutamic acid decarboxylase-65 antibody (GAD65) and islet cell antibody (ICA) markers for type 1 diabetes were strongly positive. An OGTT three months after the first showed no change (Table 1). Six months after diagnosis she is taking repaglinide 0.5 mg with meals and has good postprandial glycaemic control. She has been offered low-dose preprandial insulin but prefers to use oral medication for as long as possible.

## COMMENT

The Diabetes Prevention Trial Investigators have described a group of patients who are diagnosed with type 1 diabetes on the basis of OGTT alone[1]. The study involved screening all ICA-positive relatives of a cohort of type 1 diabetic patients with OGTT to determine glycaemic status and intravenous GTT to determine first-phase insulin response. Of 585 relatives screened, 73% had normal glucose tolerance, 14.9% had impaired glucose tolerance and 10.4% had diabetes. Amongst the diabetic cohort, diabetes was diagnosed generally on the basis of postprandial hyperglycaemia rather than fasting hyperglycaemia. In addition, these patients had an impaired first-phase insulin response to a glucose load and high-risk diabetes HLA haplotypes were common in this group. All were symptom-free and the age range was 3–45 years. The fact that they are young and ICA-positive, have a first-degree relative with type 1 diabetes, and possess high-risk HLA haplotypes makes it very likely they have true type 1 diabetes and will eventually develop symptomatic disease requiring insulin.

This newly described subset gives some clue as to the natural history of type 1 diabetes before it becomes clinically apparent. The impaired first-phase response to a glucose load suggests that the earliest manifestation of beta cell failure is loss of acute insulin response to a glucose load, leading to postprandial hyperglycaemia. The viral illness in our patient may or may not have influenced her glycaemic status, but we note that glucose tolerance was unaltered after recovery. An unanswered question is whether 'resting' the beta-cells with low-dose insulin or immunomodulatory therapies such as cyclosporin will prevent or delay progression to full-blown diabetes[2].

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# A young man with parkinsonism

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In Parkinson's disease (PD) imaging of the brain is seldom cost-effective since the chance of detecting relevant structural abnormality as a cause of the symptoms is very low. However, brain imaging can provide diagnostic clues in young patients with atypical parkinsonian features.

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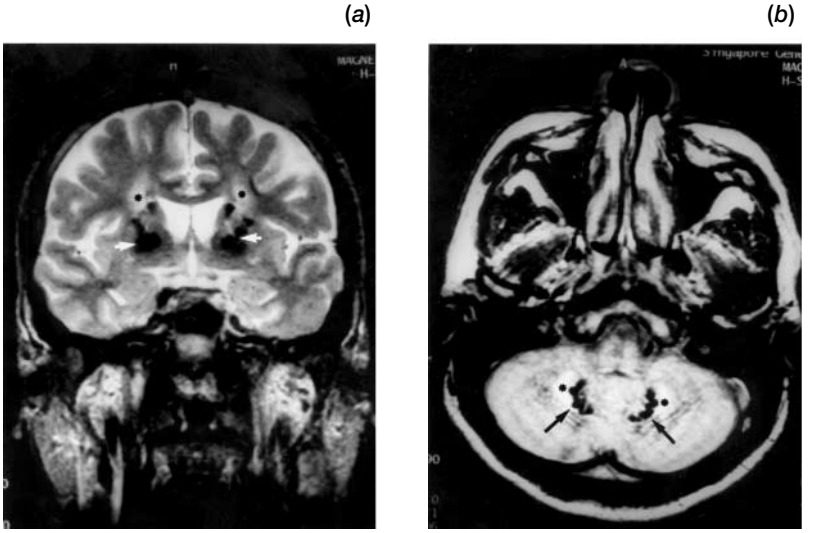


*Figure 1* **MRI scans (*a* and *b*).** T2 hyperintense areas (asterisks) in periventricular white matter and cerebellar medulla; deposition of confluent paramagnetic material (arrows) in both lentiform and dentate nuclei

## CASE HISTORY

A Chinese man aged 28 had experienced progressive slowness of movements, limb stiffness, generalized malaise and fatigue over the past eighteen months. Activities of daily living were ever more difficult. Family members noticed that his mental responses were slowing and his handwriting was becoming smaller. In addition, his speech was diminished in volume and slurred and his balance was poor, with a tendency to fall when turning or if pushed. He did not have swallowing difficulty, urinary or bowel complaints, memory loss or visual or hearing problems. There was no history of delay in his developmental milestones and no family history of parkinsonism or other neurological illness. There was also no history of any psychiatric illness, or of exposure to heavy metals and toxins.

On examination his mentation was slow and his speech hypophonic and slurred. Extraocular movements were full. There was moderately severe rigidity and bradykinesia in all four extremities with the right more affected than the left. Rest tremor was absent. In addition, he had mild bilateral dysmetria and dysdiadochokinesia. Power was full in his limbs. All his reflexes, including the jaw jerk, were brisk. There was no demonstrable clonus and his plantar responses were flexor. He had a mild stooped posture and a slow and shuffling gait, with decreased arm swing. His postural stability was poor. Investigations on blood included blood count, blood film, urea and electrolytes, liver function tests, thyroid and parathyroid function tests, vitamin E levels, calcium, phosphate, magnesium, iron, ferritin, caeruloplasmin, heavy metals and Venereal Disease Research Laboratory test. All were normal. DNA tests for trinucleotide repeat expansions for spinocerebellar ataxia 1,2,3,6 and 7 were negative. Slit lamp examination revealed no corneal Kayser–Fleischer rings or lens opacities. Somatosensory evoked and brainstem evoked potentials were normal but visual evoked potentials showed conduction abnormalities in the anterior visual pathways bilaterally. On neuropsychological testing his general intellectual abilities were of low average range; we could not determine whether there had been recent deterioration. Memory and attention abilities seemed unimpaired. MRI of the brain revealed diffuse white matter disease with extensive paramagnetic material in the basal ganglia, cerebellum, and subcortical regions (Figure 1*a* and *b*). A CT scan confirmed the gross calcifications in the dentate nuclei, basal ganglia and subcortical regions. His response to levodopa therapy was poor, and this was subsequently stopped.

The patient had four sisters and a brother. Two of the sisters were willing to have CT scans of the head, and these were normal.

## COMMENT

This case illustrates how brain imaging can provide diagnostic clues in a young patient with parkinsonism. The investigation is especially useful if the parkinsonian features are associated with other neurological features such as pyramidal tract signs, cerebellar dysfunction, and cognitive impairment[2]. MRI may demonstrate abnormal deposition of minerals such as iron, calcium, and copper or structural lesions in basal ganglia. It is a useful diagnostic tool in the differential diagnosis of parkinsonism[5]. MRI can also help distinguish some of the parkinson-plus syndromes, such as multiple system atrophy, from PD[4]. In our patient, the

imaging study was warranted by the absence of limb tremor, the poor response to levodopa, the rapid progression of his symptoms and the presence of other neurological signs.

Calcifications of the brain are found in about 0.3 to 1.5% of individuals undergoing routine CT examination, most commonly in the basal ganglia and sometimes in the cerebellar dentate nucleus[4,5]. Neurological symptoms are usually absent. Hypoparathyroidism and pseudohypoparathyroidism are common aetiologies. Idiopathic calcification of the basal ganglia is often called Fahr's disease[6] but some prefer the term bilateral striatopallidodentate calcinosis. Idiopathic calcification of the basal ganglia is usually sporadic but an autosomal dominant mode of inheritance is also seen. There is a slight preponderance in men, with presentation usually between 30 and 50 years of age. Calcifications generally precede the onset of symptoms by many years and the course is typically slow and progressive. Mental deterioration, extrapyramidal signs, and cerebellar ataxia are the three predominant manifestations, resulting in gait and speech disturbance[6]. Pyramidal signs, psychiatric symptoms, urinary incontinence, and epilepsy occur in some patients. To date, there is no effective therapy.

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