A multiple-sclerosis-like syndrome associated with glue-sniffing

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J R Soc Med 2000;**93**:313–314

To satisfy the Poser criteria[1] for the diagnosis of clinically definite multiple sclerosis (MS), conditions that could mimic MS must be excluded.

CASE HISTORY

A man aged 31 was seen with a two-week history of progressive incoordination, lower limb weakness, right hemifacial tingling and paraesthesia in the hands. A similar episode had occurred 4 years previously with good recovery. On examination he had upper limb ataxia, a mild spastic paraparesis with a sensory level to D6 and bilateral extensor plantar responses. The results of routine investigations were normal apart from a slight increase in liver enzymes. Cerebrospinal fluid (CSF) analysis was unremarkable, with absence of oligoclonal bands, but visual evoked potentials (VEPs) showed a delay in the P100 latency to pattern stimulation bilaterally (right eye 154 ms, left eye 150 ms) with amplitude preservation. Peripheral nerve conduction was normal.

The patient subsequently revealed that he had sniffed glue (Evostick, up to 5 L/week) since the age of 15 years; other recreational drugs, including alcohol, had not been used. Very high urinary hippuric acid levels (a marker of toluene exposure or intake) were consistent with inhalational abuse of toluene. Magnetic resonance imaging (MRI) of the brain showed diffuse high signal within the cerebral white matter on T2-weighted images (Figure 1). Spinal cord MRI was normal.

Solvents were withheld. After inpatient rehabilitation he became able to walk unaided with slight gait ataxia. Six months later he returned with an acute encephalopathy, increasing ataxia and distal dysaesthesia, having resumed glue-sniffing. Cognitive function, ataxia and sensory symptoms recovered over 4 days with supportive management and detoxification. He remained well for two months but subsequently experienced a deterioration in mobility with impaired visual acuity, facial numbness and urinary sphincter disturbance. Ataxia prevented him from walking. Two years after we first saw him he relapsed again, with worsening mobility, diplopia and urinary incontinence. He was admitted for one week and solvents were again withheld. A month after discharge, diplopia, sphincter function and mobility had improved. At present he is independently mobile with a stick for occasional support.

COMMENT

Most volatile glues contain toluene and n-hexane. Toluene is believed to be the main cause of central nervous system damage[2]. Long-term solvent abuse can cause permanent neurological deficits, and neuropathological studies of chronic users have revealed diffuse white matter demyelination and limited axonal degeneration. After short-term exposure to toluene-containing solvents, complete neurological recovery is usual.

If our patient had not been so candid about his recreational drug use an initial diagnosis of clinically definite MS could have been made. His features were not typical of solvent exposure. Often, chronic solvent abuse is characterized by an insidious, progressive, neurological course. In this case, the patient presented acutely despite 15 years of glue-sniffing. He made a good recovery following each 'relapse'. An acute neurological presentation of solvent abuse may therefore occur on a background of chronic exposure. Probably the relapses in this patient were associated with increased solvent abuse. The mechanism of relapse with rapid recovery is unclear. One possibility is conduction block due to an effect on membrane function and action potential transmission, with recovery as toluene and its metabolites are cleared.

Paraclinical tests such as VEPs, MRI, and CSF analysis are commonly used to support the diagnosis of MS but can

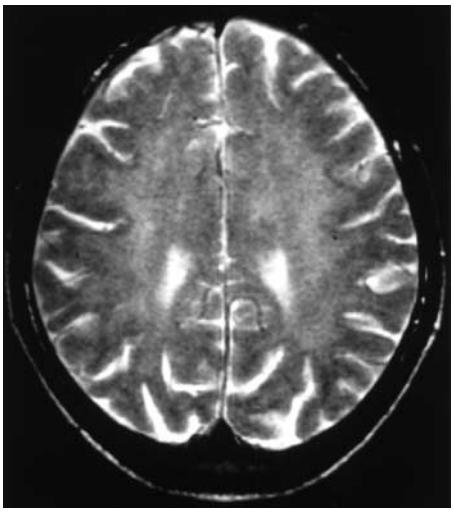

Figure 1 Abnormal diffuse high T2 signal within the white matter, extending to the arcuate fibres

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be abnormal also in solvent-induced demyelination. Delay in VEP latency, reported in up to 95% of patients with MS, has been seen in paint-thinner abusers and in symptomless workers with chronic low-level exposure to toluene[3]. Might this patient have had MS, with the glue-sniffing incidental? Diffuse white matter changes on MRI, as observed here, have previously been described in abusers of volatile glues[4]. By contrast, in MS diffuse white matter changes would be very unusual: up to 99% of patients with clinically definite MS have at least one focal white matter abnormality[5]. Absence of oligoclonal bands unique to the CSF, as noted in this case, has been reported in only 3% of patients with clinically definite MS[6].

In patients with symptoms and a clinical course suggestive of MS, glue-sniffing is a possible aetiology to be considered. Unusual clinical features such as encephalopathy during a relapse, and atypical features such as the presence of diffuse white matter lesions on MRI, will reinforce suspicion of a non-MS cause. In these cases particularly, a history of recreational and occupational exposure to toluene (dry cleaning, paint spraying, industrial cleaning) should be sought.

REFERENCES

1 Poser CM, Paty DW, Scheinberg L, *et al.* New diagnostic criteria for multiple sclerosis: guidelines for research protocols. *Ann Neurol* 1983;**13**:227–31

2 White RF, Proctor SP. Solvents and neurotoxicity. *Lancet* 1997;**349**:1239–43

3 Poblano A, Lope Huerta M, Martinez JM, Falcon HD. Pattern-visual evoked potentials in thinner abusers. *Arch Med Res* 1996;**27**:531–3

4 Yamanouchi N, Okada S, Kodama K, *et al.* White matter changes caused by chronic solvent abuse. *Ann J Neuroradiol* 1995;**16**:1643–9

5 Ormerod IEC, Miller DH, McDonald WI, *et al.* The role of NMR imaging in the assessment of multiple sclerosis and isolated neurological lesions: a quantitative study. *Brain* 1987;**110**:1579–616

6 Tourtellotte WW, Baumhefner RW, Syndulko K, *et al.* The long march of the cerebrospinal fluid profile indicative of clinical definite multiple sclerosis; and still marching. *J Neuroimmunol* 1988;**20**: 217–27

Alimentary hyperglycaemia mistaken for diabetes mellitus

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J R Soc Med 2000;**93**:314–315

When symptoms point to diabetes, the diagnosis can be confirmed by a random blood glucose concentration greater than 11 mmol/L, usually with concurrent glycosuria[1]. Sometimes the result is misleading.

CASE HISTORY

A man aged 76 attended a diabetic clinic for 4 years for control of his diabetes, which responded well to diet alone. His HbA1c levels over this period were in the range 6.2–7.5% (non-diabetic $<6.1\%$). One of us (NAM) noticed that the diagnosis was initially based on a random serum glucose level of 13 mmol/L and the patient had not experienced typical symptoms of diabetes mellitus. An oral glucose tolerance test (GTT) showed a fasting plasma glucose of 5.2 and a 120-minute value of 2.8 mmol/L.

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A prolonged GTT was arranged. The patient was advised to eat normally for at least 3 days before the test and to fast from 10 o'clock the night before the test. The glucose load was 130 mL Polycal (Nutricia) in 300 mL water, which was equivalent to 75 g glucose. The resulting glucose curve and urine sugar values are shown in Figure 1. Fasting plasma glucose was normal at 4.6 mmol/L and the value rose to a peak of 15.4 mmol/L at 45 min and was normal at 120 min. The lowest plasma glucose was

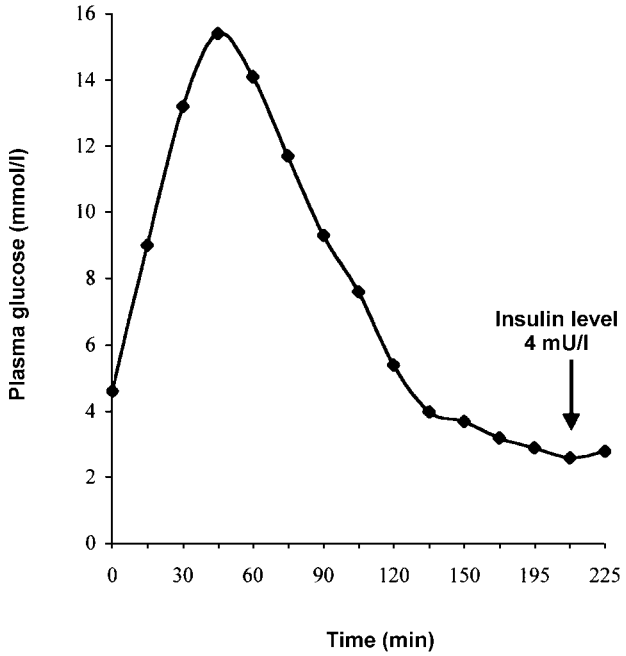

Figure 1 **Prolonged glucose tolerance test**

2.6 mmol/L at 210 min. The corresponding plasma insulin was 4 mU/L (2–15). The patient had no symptoms or signs of hypoglycaemia during the test. He stated that he had always been a healthy person. His blood pressure was 110/75 and his body mass index was 20.7. On inquiry he gave a history of a surgical procedure 48 years previously for duodenal ulcer.

COMMENT

Vagotomy and gastrectomy were once common treatments for duodenal ulcer. Elective surgery for peptic ulcer is now rare, but many individuals who underwent these operations in the past will demonstrate what is termed alimentary hyperglycaemia. In a population screening of 4667 middle-aged men, 158 (3.4%) reported a history of previous operation for gastric or duodenal ulcer[2]. Alimentary hyperglycaemia may occur in normal people and in patients with hyperthyroidism, peptic ulceration or hepatic disease[1].

Several types of vagotomy, such as truncal vagotomy, selective gastric vagotomy and selective proximal vagotomy, have been used. Characteristic changes in the oral GTT are hyperglycaemia at 30 and 60 min after glucose loading, hypoglycaemia at 120 min, blood glucose returning to normal by 180 min[3,4]. The major cause for the altered oral GTT is rapid emptying of hypertonic glucose into the proximal intestine, with brisk absorption of glucose and excessive insulin secretion. The exaggerated insulin response may be partly responsible for the later hypoglycaemia, along with other mechanisms[5].

In most cases of alimentary hyperglycaemia, the patient should simply be advised to eat smaller meals. Severe cases can be treated with α_1-glucosidase inhibitors or metformin. The permanence of alimentary hyperglycaemia will mean that these individuals are exposed to recurring cycles of hyperglycaemia and hyperinsulinaemia, and it is not known whether this increases their susceptibility to cardiovascular disease or diabetes mellitus.

Diabetes mellitus should not be diagnosed on the basis of a single random or postprandial glucose unless there are typical manifestations or complications of diabetes. A high HbA_{1c} is not confirmatory. A history of gastric or duodenal operations should be sought, a factor that may influence interpretation of random blood glucose levels and oral GTT. Conversely, previous duodenal or gastric surgery may cause the 120 min result to be normal when diabetes is present.

REFERENCES

1 Frier BM, Truswell AS, Shepherd J, De Looy A, Jung R. Diabetes mellitus, and nutritional, and metabolic disorders. In: Haslett C, Chilvers ER, Hunter JAA, Boon NA, eds. *Davidson's Principles and Practice of Medicine*. London: Churchill Livingstone, 1999:480

2 Hanson M, Trell E. Chronic effects of gastroduodenal surgery on glucose and insulin response to oral glucose tolerance test in a population study. *Acta Diabetol Lat* 1982;**21**:173–o9

3 Gulsrud PO, Taylor IL. How gastric emptying of carbohydrate affects glucose tolerance and symptoms after truncal vagotomy with pyloroplasty. *Gastroenterology* 1980;**78**:1463–71

4 Hall WH. Effect of vagotomy and pyloroplasty; the oral glucose tolerance test. *Gastroenterology* 1973;**64**:217–22

5 Akiyama H, Ohdate K, Twasaki M, *et al*. Effects of selective vagotomy with or without drainage on glucose tolerance test. *Jap J Surg* 1984;**14**:15–22

Acute myocardial infarction without raised creatine kinase activity

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J R Soc Med 2000;**93**:315–316

Measurement of serum creatine kinase (CK; EC 2.7.3.2.) is widely used in diagnosis of acute myocardial infarction[1].

CASE HISTORY

A man aged 49 years experienced chest pain, and an electrocardiogram 12 hours after onset showed a typical myocardial infarction pattern with Q waves >0.04 s and depth from the baseline more than a quarter the height of the R wave in leads I, V_I–V_6 and $_aV_L$. The laboratory profile was also typical of acute myocardial infarction—raised lactate dehydrogenase (LDH) and aspartate aminotransferase (AST) activity, leukocytosis, raised C-reactive protein (CRP)—except that total CK activity measured by the GSCC method[2] was only 30 IU/L (Table 1). On day 5 the patient died, and at necropsy he proved to have an extensive anterior myocardial infarction with left main coronary artery occlusion.

Serum was available from all phases of his clinical course, and we examined changes in the activities of various enzymes. All showed a typical pattern of acute myocardial infarction with the exception of CK activity, which remained within the normal range and showed no sharp increases. On electrophoresis CK is divided into three isozymes—MM, MB and BB[3]. Generally, isozyme MB appears in the blood within a few hours after first symptoms of myocardial infarction, peaks in about 6–18 hours and

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Table 1 **Laboratory data on day 1**

	Laboratory data	*(Normal)*
WBC	15.2×10^9/L	(3.3–9.0)
CRP	4.6 mg/dL	(<0.5)
AST	1005 IU/L	(10–40)
ALT	1200 IU/L	(5–40)
CK	30 IU/L	(60–270)
LDH	4150 IU/L	(250–420)

WBC=White blood cell count; CRP=C-reactive protein; AST=aspartate aminotransferase; ALT=alanine aminotransferase; CK=creatine kinase; LDH=lactate dehydrogenase

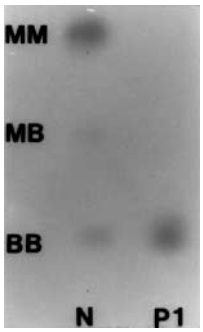

Figure 1 **Isozyme pattern of creatine kinase**
N: healthy individuals; P_1: patient

then decreases slowly, almost disappearing within 36 hours. We examined serum from the first hospital day and could find neither CK-MM nor CK-MB bands (Figure 1).

Absence of CK-MM in these circumstances suggested a possible genetic defect, so we examined the genomic DNA of the CK-M subunit. There was a point mutation, GAC (Asp)→GGC (Gly), of codon 54 on direct sequencing by the taq dye deoxy terminator cycle method[4] (Figure 2).

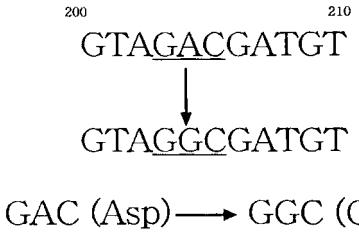

Figure 2 **Point mutation in codon 54 identified by taq dye deoxy terminator cycle method**

COMMENT

A causal relation between the missense mutation and the low CK activities is unproven. However, an effect on the substrate binding domain, the coenzyme binding domain and the subunit contact domain is plausible—as is believed to happen with silent cholinesterase and deficiency of the LDH subunit. This must be clarified—for clinical reasons and also because of the medicolegal implications of a normal CK in myocardial infarction.

REFERENCES

1 Friedberg CK. Coronary heart disease. In: Beeson PB, McDermott W, eds. *Cecil-Loeb Textbook of Medicine.* Philadelphia: W B Saunders 1967; 634–57

2 Empfehlungen der Deutschen Gesellschaft für Klinische Chemie. Standardisierung von Methoden zur Bestimmung von Enzymaktivitäten in Biologischen Flüssigkeiten. Standard-methode zur Bestimmung der Aktivität Creatin-kinase. *J Clin Chem Clin Biochem* 1977; **15**:249–54

3 Rosalki SB. Creatine phosphokinase isoenzymes. *Nature* 1965;**202**:701

4 Bailly J, Mackenzie AE, Leblond S, Korneluk RG. Assessment of creatine kinase isozyme M defect as a cause of myotonic dystrophy and the characterization of two novel CK-MM polymorphisms. *Hum Genet* 1991;**86**:457–62

Epstein–Barr virus infection mimicking extrahepatic biliary obstruction

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J R Soc Med 2000;**93**:316–318

In healthy adults, Epstein–Barr virus (EBV) infection typically causes infectious mononucleosis[1]. Liver involvement is usually of mild or moderate severity, manifested by non-specific disturbances of liver enzymes[2]. Severe cholestatic jaundice is unusual[3].

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CASE HISTORIES

Case 1

A man aged 50 was admitted with a six-day history of general malaise, non-productive cough, severe myalgia, intermittent sweats and nausea. The epidemiological and drug histories were unremarkable and his alcohol intake was not excessive. On examination his temperature was 37.6 °C; there was no jaundice, pharyngitis, lymphadenopathy or meningism. White cell count was 2.6×10^9/L with 1.02×10^9/L neutrophils; scanty atypical mononuclear cells were seen on the blood film but a Paul–Bunnell test was negative. Serial liver function tests are shown in Table 1. Arterial blood oxygen was mildly depressed at 9.2 kPa; a chest radiograph was normal; C-reactive protein was 40 g/L (normal <10); indices of coagulation

Table 1 **Serial liver enzymes**

	Days							
Case	**1**	**4**	**7**	**9**	**10**	**14**	**32**	**60**
Case 1								
Alk phos	1413	1750	2704	—	3105	1725	—	—
AST	217	97	132	—	94	48	—	—
Bilirubin	42	109	134	—	152	52	—	—
GGT	805	833	1325	—	1385	—	—	—
Case 2								
Alk phos	1201	1463	—	1154	—	—	492	364
AST	338	163	—	101	—	—	44	45
Bilirubin	143	144	—	48	—	—	21	15
GGT	285	276	—	357	—	—	160	132

Alk phos=Alkaline phosphatase; AST=aspartate aminotransferase; GGT=gamma glutamyl transferase

were within the normal range. Blood and urine specimens grew no organisms on culture and serological tests for *Legionella pneumophila*, *Mycoplasma pneumoniae*, *Chlamydia psittacci*, *Coxiella burnetti*, cytomegalovirus, Epstein–Barr virus, hepatitis A, B and C viruses, leptospirosis and toxoplasmosis were all initially negative. *Legionella* antigen was not detected in urine specimens. The differential diagnosis included biliary tract obstruction with ascending cholangitis and atypical pneumonia, so he was started on intravenous cefotaxime and oral clarithromycin.

After admission his liver function tests showed worsening cholestasis and he became jaundiced. On abdominal untrasonography the gallbladder was thick-walled but was otherwise normal; endoscopic retrograde cholangiopancreatography showed no abnormality. The bilirubin peaked on day 9, following which his liver function tests gradually improved. On outpatient review 2 and 4 weeks later the jaundice and liver dysfunction had resolved. 4 weeks after discharge convalescent serology was positive for IgM against Epstein–Barr viral capsid antigen (EBV-VCA). Four months later he was EBV-VCA IgM negative and IgG positive against Epstein–Barr nuclear antigen (EBNA).

Case 2

A 31-year-old man was referred by his general practitioner with abnormal liver function tests and a provisional diagnosis of biliary tract obstruction with infection. In the previous week he had experienced general malaise, anorexia, sweats, right upper quadrant discomfort and progressive jaundice, but no sore throat. There was no relevant epidemiological history, his alcohol intake was negligible and he was not taking any regular medication. On examination he was icteric with a temperature of 37.9 °C; he had cervical lymphadenopathy and enlarged erythematous tonsils, with 2 cm of hepato-splenomegaly. Initial investigations showed a leucocytosis of 15×10^9/L with 8.7×10^9/L lymphocytes, thrombocytopenia (117×10^9/L) and liver function tests with an obstructive pattern (see Table 1). Bacteriological examinations of urine and blood were negative and a chest radiograph was normal. Serological tests for hepatitis A, B and C viruses, cytomegalovirus and toxoplasma were negative. Abdominal ultrasonography confirmed hepato-splenomegaly and showed a thickened contracted gallbladder, but there was no evidence of biliary tract obstruction. The Paul–Bunnell test and EBV-VCA IgM were positive but EBNA IgG was negative. His symptoms and liver dysfunction gradually improved with conservative management and he subsequently became EBNA IgG positive.

COMMENT

The clinical and virological features of these two cases were ultimately consistent with EBV-induced cholestatic hepatitis as part of the syndrome of infectious mononucleosis. In healthy adults, EBV infection typically causes 'glandular fever', with general malaise, fever, sweats, sore throat, headache and fatigue; examination commonly reveals pharyngitis, generalized lymphadenopathy (particularly cervical) and splenomegaly, with atypical lymphocytes on the blood film[1] (although this can also be a feature of other acute viral infections including viral hepatitis, of serum sickness and of chronic bacterial infections[4]). A non-specific mild increase in liver enzymes is common[2] but cholestatic jaundice to the extent seen in these patients is very unusual and can lead to initial misdiagnosis of biliary tract obstruction[3].

In the cases described here, the diagnosis of EBV infection was based on the symptoms and signs, liver enzyme dysfunction and seroconversion, initially to specific anti-EBV IgM and subsequently IgG. Liver biopsy was not done in either patient because they were recovering with conservative management. It is noteworthy that both patients had gallbladder wall thickening on abdominal ultrasonography. This finding has been reported previously in association with EBV infection, and may reflect the severity of hepatitis[5].

REFERENCES

1 Epstein MA, Crawford DH. The Epstein–Barr virus. In: Weatherall DJ, Ledingham JGG, Warrell DA, eds. *Oxford Textbook of Medicine*. Chap 7.10.4, 3rd edn. Oxford: Oxford University Press, 1996:352–4

2 Horwitz CA, Burke D, Grimes P, Tombers J. Hepatic function in mononucleosis induced by Epstein–Barr virus and cytomegalovirus. *Clin Chem* 1980;**26**:243–6

3 Bernstein CN, Minuk GY. Infectious mononucleosis presenting with cholestatic liver disease. *Ann Intern Med* 1998;**128**:509

4 Gay JC, Athens JW. Variations of leukocytes in diseases. In: Lee GR, Foerster J, Lukens J, Paraskevas F, Greer JP, Rodgers GM, eds. *Wintrobes's Clinical Haematology*. Chap 72, 10th edn. Baltimore: Williams & Wilkins, 1999:1850–1

5 Sainsbury R, Smith PK, LeQuesne G, Davidson GP, Jureidini KF, Moore DJ. Gallbladder wall thickening with infectious mononucleosis hepatitis in an immunosuppressed adolescent. *J Pediatr Gastroenterol Nutr* 1994;**19**:123–5

Salmonella meningitis and a green iguana

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J R Soc Med 2000;**93**:318–319

Salmonella species are a rare cause of bacterial meningitis, affecting mainly infants and the immunocompromised. Human salmonellosis associated with exotic pets has increased[1].

CASE HISTORY

A 27-day-old baby boy was seen with an 8-hour history of irritability and poor feeding. His birth had been uncomplicated and he had been well since. On examination, he was lethargic and febrile (38.7°C) and had a diffuse erythematous rash. His fontanelle was normal. The white cell count was 12.5×10^9/L (neutrophils 66%). Sepsis was suspected and intravenous saline and cefotaxime were started. Cerebrospinal fluid showed 1500 polymorphs and 100 lymphocytes/μL, no red cells, no organisms on the Gram stain, protein 1.4 g/L, and glucose 2.7 mmol/L (serum glucose 7.4 mmol/L). Ampicillin and gentamicin were added to cover *Listeria monocytogenes*.

The next day, culture of the cerebrospinal fluid grew a Salmonella species. Treatment was continued with cefotaxime alone. The baby lived with his parents and eight-year-old brother; no one had recently had diarrhoea and stool cultures from all four family members yielded no pathogens. The Laboratory of Enteric Pathogens in Colindale, UK, identified the organism as *Salmonella poona*, a serotype found mainly in reptiles. Further questioning of the family revealed that they owned a green iguana (*Iguana iguana*), which was handled only by the father and not allowed out of its tank. The iguana was healthy but was excreting *S. poona* indistinguishable from the first isolate. Environmental swabs taken in the family home grew *S. poona* from the vacuum cleaner and the iguana tank.

The baby recovered clinically and completed a 22-day course of intravenous antibiotics. Repeat lumbar puncture after treatment was normal, and there is no evident neurological or developmental deficit. The iguana has since been moved to an animal sanctuary.

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COMMENT

Salmonella species cause 0.2% of bacterial meningitis[2]. In at least half the cases there are serious acute complications such as ventriculitis, cerebral abscesses and subdural effusions; up to 71% of patients have permanent neurological sequelae, including hydrocephalus, seizures and mental retardation[3]. Because of the severity of the infection and risk of relapse, at least three weeks' intravenous treatment with a third-generation cephalosporin (usually cefotaxime or ceftriaxone) is advised[4].

Salmonella infection is a zoonosis, and has been reported in association with numerous reptiles including turtles, iguanas and snakes[5]. Pet turtles were recognized as a major source of salmonellosis in the United States in the 1970s, and restriction on their sale was credited with preventing some 100 000 Salmonella infections a year[6]. In the past decade, reptile-associated salmonellosis has become more important with the growing popularity of exotic animals as pets. Pet-owners may be unaware that most reptiles carry and intermittently excrete Salmonella species in their faeces[5]. The contaminated environment, or other people within that environment, may also be sources of infection: this is not the first case in which the patient did not have direct contact with the reptile[1]. When encountered in human clinical specimens, reptile-associated Salmonella serotypes such as *S. java*, *S. stanley*, *S. marina, S. poona* and *S. pomona* should always raise the possibility of exposure to reptiles.

Recommendations have been published to prevent transmission of Salmonella species from reptiles to humans[1]. Pet-store owners, veterinarians and paediatricians should educate owners and potential purchasers of reptiles. Pet reptiles should be kept away from kitchens and eating areas. Owners should wash their hands thoroughly after handling reptiles. Finally, reptiles should be kept away from children under 5 years or immunosuppressed patients, who are at greatest risk.

REFERENCES

1 Anonymous. Reptile-associated salmonellosis—selected states, 1996–1998. *MMWR* 1999;**48**:1009–13

2 Schlech WF, Ward JI, Band JD, Hightower A, Fraser DW, Broome CV. Bacterial meningitis in the United States, 1978 through 1981: The National Bacterial Meningitis Surveillance Study. *JAMA* 1985;**253**: 1749–54

3 Unhanand M, Mustafa MM, McCracken GH, Nelson JD. Gram-negative enteric bacillary meningitis: a twenty-one year experience. *J Pediatr* 1993;**122**:15–21

4 Quagliarello VJ, Scheld WM. Treatment of bacterial meningitis. *N Engl J Med* 1997;**336**:708–16

5 Woodward DL, Khakhria R, Johnson WM. Human salmonellosis associated with exotic pets. *J Clin Microbiol* 1997;**35**:2786–90

6 Cohen ML, Potter M, Pollard R, Feldman RA. Turtle-associated salmonellosis in the United States: effect of public health action, 1970 to 1976. *JAMA* 1980;**243**:1247–9

Angel's trumpet and the eye

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J R Soc Med 2000;**93**:319

Acute unilateral mydriasis warrants urgent neurological and ophthalmological examination. Local absorption of alkaloid is a possible cause.

CASE HISTORY

Shortly after gardening, a woman aged 53 complained of vertigo, blurred vision and palpitations. A neighbour noted mydriasis of the right eye and she consulted an ophthalmologist. Since the reason for the mydriasis and tachycardia (120/min) was unclear, she was admitted to a department of neurology. At that time, the highly dilated pupil hardly reacted to light or convergence but the systemic symptoms had gone. The clinical findings were interpreted as sympathomimetic overactivity or parasympathetic underactivity of the pupillomotor nerve fibres. On neurological examination nothing else was found—in particular no evidence of a retrobulbar or intracerebral process. There was no relevant drug history, but detailed inquiry revealed that, while she had been cutting leaves from an Angel's trumpet (*Datura suaveolens*) in her garden, a drop of sap had entered the affected eye. The unilateral mydriasis disappeared within two days.

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COMMENT

The present case was unusual in that systemic effects of the alkaloids were the chief complaints. The typical story is of unilateral mydriasis as an isolated finding, mimicking a neuro-ophthalmological disorder and lasting up to six days[1]. Angel's trumpet or moonflower, from the nightshade family, is a popular ornamental plant in western Europe. The anticholinergic effect of its sap and other parts results from a mixture of different alkaloids, mainly hyoscine but also hyoscyamine and atropine[2]. In most cases of poisoning by such plants, alkaloid-containing parts have been taken orally. Clinical effects are especially seen in children poisoned by mistake[3] and in adolescents abusing home-made drugs for their hallucinating effects[2]. Systemic effects after topical ophthalmic applications of large doses of hyoscine have been described[4]. Absorption from the conjunctiva and, after nasolacrimal delivery, from nasal mucosa bypass the first-pass effect of the liver[4]. Other circumstances in which unilateral mydriasis has resulted from local drugs are treatment of airway disease with nebulized ipratropium bromide[5] and prevention of motion sickness with transdermal hyoscine[6].

REFERENCES

1 Voltz R, Hohlfeld R, Liebler M, Hertel H. Gardener's mydriasis. *Lancet* 1992;**339**:725

2 Smith EA, Meloan CE, Pickel JA, Oehme FW. Scopolamine poisoning from homemade 'moon flower' wine. *J Anal Toxicol* 1991;**15**:216–19

3 Rubinfeld RS, Currie JN. Accidental mydriasis from blue nightshade 'lipstick'. *J Clin Neuroophthalmol* 1987;**7**:34–7

4 Lahdes K, Huupponen R, Kaila T, Salminen L, Iisalo E. Systemic absorption of ocular scopolamine in patients. *J Ocul Pharmacol* 1990;**6**:61–6

5 Helprin GA, Clarke GM. Unilateral fixed dilated pupil associated with nebulised ipratropium bromide. *Lancet* 1986;**ii**:1469

6 Rubin MM, Sadoff RS, Cozzi GM. Unilateral mydriasis caused by transdermal scopolamine. *Oral Surg Oral Med Oral Pathol* 1990;**70**: 569–70