aplastic anaemia is characterised by anaemia, leucopenia, and thrombocytopenia. The defect is cellular with an increased tendency to infection and bleeding.[4] Although prejudicial to the success of skin grafting, it is not a contraindication provided deficiencies are corrected perioperatively, haemostasis is achieved before grafting, and the formation of haematoma is avoided afterwards.

Each of these children had a chronic cutaneous manifestation of a haematological or immunodeficiency disease. Healing was assumed to be abnormal because of a defect in either humoral or cellular defence mechanisms. This assumption delayed referral for surgery by some months in each case, but debridement followed by split skin grafting produced rapid healing.

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Pneumonia associated with contact with cyanobacteria

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We report an outbreak of possible cyanobacterial poisoning after contact with water containing toxic *Microcystis aeruginosa* at a freshwater reservoir in Staffordshire, England, and give details of two cases of pneumonia that occurred after the water was swallowed.

Case reports

Case 1—A 16 year old army recruit was admitted on 26 September 1989 with a five day history of malaise, sore throat, blistering around the mouth, pleuritic pain on the left side, and a dry cough. He had also developed vomiting with central abdominal pain, followed by diarrhoea for one day. On examination he had a fever, with a temperature of 38·4°C. Examination disclosed signs of left basal consolidation, which was confirmed on chest radiography. He was tender in the umbilical area. The table shows the results of the laboratory investigations. He was treated with erythromycin and cefuroxime (owing to his sensitivity to penicillin), and his temperature settled within 24 hours. He remained periodically confused for two days with episodes of hallucinations but was well enough for discharge after one week.

Case 2—A 16 year old army recruit was admitted on the same date with a four day history of malaise, vomiting, sore throat, blistering around the mouth, dry cough, and pleuritic pain on the left side. He had also developed central abdominal pain. On examination he had a fever, with a temperature of 39·2°C. Respiratory examination disclosed signs of left basal consolidation, which was confirmed by chest radiography, and abdominal examination showed epigastric and umbilical tenderness and tenderness in the right iliac fossa, with rebound tenderness. He was treated with penicillin and erythromycin, and his temperature settled within 24 hours. The tenderness in his right iliac fossa persisted for three days; the presumptive diagnosis was mesenteric adenitis. He was discharged after one week.

In both patients acute and convalescent serum samples showed no significant rise in antibody titre to leptospira, *Legionella pneumophila*, influenza virus A and B, chlamydia group antigen, *Coxiella burnetii*, *Mycoplasma pneumonia*, and adenovirus. Repeat chest *x* ray films showed considerable resolution of the consolidation after one week.

Results of laboratory investigations on patients with pneumonia after swallowing water from a freshwater reservoir

	Case 1	Case 2
White cell count ($\times 10^9$/l)	11·8	17·2
Total differential counts ($\times 10^9$/l):		
Neutrophils	10·30 (neutrophilia)	15·6 (neutrophilia)
Lymphocytes	0·52 (lymphopenia)	0·57 (lymphopenia)
Platelets ($\times 10^9$/l):		
26 September 1989	141	205
3 October 1989	703	420
Alanine transaminase (1U/l)	56	<35
Aspartate transaminase (1U/l)	94	<35
Blood gas tensions	Normal	Normal
Microbiological cultures:		
Blood	No growth	No growth
Sputum	Normal flora	Normal flora
Throat swab	Normal flora	Normal flora
Urine	No growth	No growth
Faeces	No enteric pathogens	No enteric pathogens
Leptospira enzyme immunoassay	Negative	Negative
Legionella indirect fluorescent antibody test (acute and convalescent serum samples)	<1·8	<1·8
Complement fixation tests for respiratory pathogens (acute and convalescent serum samples)	No significant rise in titre	No significant rise in titre

Comment

The two recruits had taken part in canoe exercises lasting several hours in Rudyard reservoir, during which they swallowed some of the water. The water contained a mass development of cyanobacteria, dominated by *Microcystis aeruginosa*. Microcystin peptides[1,2] were present in a sample of the bloom collected on 27 September 1989, and microcystin-LR was identified in the bloom material by high performance liquid chromatography.[3] Reservoir water sampled on 9 October by the Public Health Laboratory Service contained counts of *Escherichia coli* considered to be unsuitable for bathing by European Community standards. There was no evidence of enterovirus contamination in water sampled on 2 October (National Rivers Authority, personal communication).

In the two cases described the patients presented with left basal pneumonia four to five days after taking part in a canoeing excercise. One (case 1) had difficulty sleeping that night and awoke the next morning with a sore throat, dry cough, vomiting, and abdominal pains, and the other developed similar symptoms within 24 hours and difficulty in walking.

In addition to liver damage in mice after microcystin intoxication, atypical pulmonary thrombosis has been observed, including pulmonary intravascular formation of protein deposits and removal of platelets from the circulation.[4,5] The pulmonary consolidation and low platelet counts on admission (table) may have been a consequence of microcystin poisoning. (Platelet counts increased during treatment from 141×10^9/l to 703×10^9/l (case 1) and from 205×10^9/l to 420×10^9/l (case 2).)

Subsequently 16 soldiers were admitted to the medical centre at their barracks. Preliminary investigations showed that eight had been canoeing and had symptoms that might have been associated with cyanobacterial poisoning, including sore throats,

headaches, abdominal pains, dry coughs, diarrhoea, vomiting, and blistered mouths. More information is needed about the hazards of cyanobacterial toxins to health.

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Shy-Drager syndrome presenting as isolated paralysis of vocal cord abductors

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The Shy-Drager syndrome (multiple system atrophy with autonomic failure) is now recognised as a rare cause of respiratory stridor due to paralysis of the abductors of the vocal cords. Characteristically, this occurs late in the disease, after patients have developed features of autonomic failure, parkinsonism, and cerebellar ataxia.[1] We report a case of Shy-Drager syndrome in which paralysis of the abductors of the vocal cords preceded the development of other features of the disease by 20 months.

Case report

A 61 year old woman presented to the ear, nose, and throat clinic in November 1987 with a 12 month history of increasing nocturnal stridor and exertional dyspnoea. Indirect laryngoscopy showed bilateral paralysis of abduction of the vocal cords, but no cause for this could be found despite full investigation including computed tomography of the neck and base of the skull. After an elective tracheostomy she remained well, although regular follow up examinations showed no recovery of the function of the vocal cords.

In January 1989 she presented to the neurology clinic with a six month history of progressive unsteadiness of gait and dysarthria. She had also complained of frequency and urgency of micturition and dribbling urinary incontinence. This had been investigated by urodynamic studies, which had shown a poorly contracting bladder with a residual volume of 400 ml. There was no other important history, and she was not taking any drugs.

Examination showed her to be alert with normal intellectual function. Supine blood pressure was 145/75 mm Hg and standing blood pressure 115/65 mm Hg. The optic fundi were normal. She had a scanning dysarthria and first degree horizontal nystagmus in both directions of lateral gaze. There was no dysphonia. Her arms and legs showed normal power, but tone was increased in the left leg. The tendon reflexes were pathologically brisk, and the left plantar response was extensor. She had ataxia and dysmetria of her arms and legs with mild bilateral intention tremor and dysdiadochokinesia. Sensory examination yielded normal findings. Her gait was broad based, veering, and ataxic, and she was unable to walk heel to toe. There was no rombergism.

Results of haematological and biochemical investigations, including erythrocyte sedimentation rate, thyroid function tests, serum vitamin B12 and folate concentrations, and plasma protein electrophoresis, were normal. A test for antinuclear factor and the Venereal Disease Research Laboratory test gave negative results. A chest *x* ray film did not show any evidence of a neoplasm. An electrocardiogram showed normal R-R interval variation (>10%) during deep breathing. Examination of cerebrospinal fluid yielded entirely normal findings, and the results of cytological examination of cerebrospinal fluid were negative. Computed tomography of the brain showed mild generalised cerebral atrophy with more pronounced atrophy of the cerebellum.

Comment

The clinical features of this case, which included cerebellar ataxia, postural hypotension, and disturbed function of the urinary sphincter, are consistent with the Shy-Drager syndrome.[1,2] The syndrome consists of a progressive panautonomic failure with later development of parkinsonian features and cerebellar disturbance.

It is well established that patients with the syndrome may develop stridor and respiratory failure later in the disease because of bilateral paralysis of the abductors of the vocal cords. In one study, eight of 12 unselected patients developed this complication, five of whom needed elective tracheostomy.[1] One of the patients developed severe stridor and respiratory failure at an early stage of the disease before evidence of extrapyramidal or cerebellar disease. In a clinicopathological study of three patients with multiple system atrophy and paralysis of the laryngeal abductors pronounced selective atrophy of the posterior cricoarytenoid muscles was found at necropsy.[3] Denervation atrophy of these muscles was seen in two of the three cases, but no evidence of primary neuronal degeneration was found in the nucleus ambiguus, the motor nucleus to the laryngeal musculature. The pathogenesis of the paralysis of the abductors of the vocal cords therefore remains unclear.

The case reported here shows that paralysis of the laryngeal abductors can be the first sign of the Shy-Drager syndrome and may precede the development of other features of the syndrome by some time—in this case 20 months. We therefore propose that the syndrome should be considered in cases of stridor caused by apparently isolated paralysis of the abductors of the vocal cords.

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