Benzine-sniffing neuropathy

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Summary

Eight children and adolescents with a predominantly motor neuropathy of which the most likely cause was \( n \)-hexane are described. \( n \)-Hexane is one of the impurities in highest concentration in benzine, a petroleum product freely available at most corner stores in South Africa. It is bought freely by a large number of Black children in Natal and sniffed to produce a state of euphoria. Benzine sniffing by children constitutes a major health hazard in Natal.

Case reports

Case 1

A 9-year-old Black boy was hospitalized because of painful knees and weakness of both legs. Symptoms began 1 month before admission. Physical examination was unremarkable. Neurological examination revealed bilateral foot-drop with symmetrical distal weakness of the lower limbs; knee jerks were diminished and ankle jerks were absent. The upper limbs were normal. There was no Babinski sign, and sensation was intact. The CSF was normal, with a protein value of 0.16 g/l. Nerve conduction velocity in the right lateral popliteal nerve was 14 m/s. All other investigations were negative.

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Case 3

A 14-year-old girl complained of progressive weakness of the hands and legs for about 6 weeks. She had previously participated in games at school, and had never been seriously ill before. General systemic examination revealed no abnormality, and the cranial nerves were normal. There was generalized, symmetrical weakness of the limbs, more pronounced distally to the extent that she had bilateral wrist- and foot-drop. She was unable to walk without support. Muscle tone was decreased and all tendon reflexes were abolished. Plantar responses were absent. There was slight reduction of appreciation of pain, vibration and joint sense in the feet and toes but no other abnormality in sensation.

Investigations, which included a blood count, blood biochemical and glucose estimations and chest radiographs, were negative. The CSF was normal in all respects, with a protein level of 0.32 g/l, and the blood lead content was not elevated.

The patient had the clinical features of a predominantly motor neuropathy. On admission to hospital she had admitted sniffing benzine, and it emerged that she had begun the sniffing habit 11 months before, sniffing with friends several times every day; they had bought the benzine from a shop in small bottles and had sniffed by pouring some onto a cloth and holding it to their noses. She did not smoke or drink alcohol. At least 4 of her female friends and 3 of her male friends also sniffed benzine, but according to the patient, they had remained well. She had enjoyed the benzine because it 'made her feel drunk'. She had stopped sniffing several weeks before coming to hospital. She was kept in hospital for physiotherapy and when seen after 4 weeks she appeared to be gaining strength, albeit slowly.

Case 4

An 18-year-old girl was referred from Eshowe Hospital complaining that she had developed difficulty in walking and weakness of the limbs about 2½ months previously. There had been progressive deterioration for 2 months, but she thought that the condition was no longer worsening and possibly improving. She had a healthy baby aged 7 months and had never been seriously ill before.

On examination she appeared to be well nourished, and general systemic examination was negative. There was generalized weakness, most pronounced distally, with slight wasting of the small muscles of the hands. All the tendon reflexes

Polyneuropathy resulting from industrial exposure to the solvent \( n \)-hexane is widely recognized. Progressive and predominantly motor neuropathy has also been reported in individuals who have inhaled vapours from glue to produce a state of euphoria.

In this paper we report 8 patients who developed polyneuropathy after long-term sniffing of benzine.
and the plantar responses were absent. She walked with difficulty and with a high-stepping gait due to foot-drop. There was no defect in sensation, and the blood count was normal except for eosinophilia (20% of 10,0 x 10^9/L white cells). The CSF was normal with a protein value of 0.16 g/L. All other investigations were negative.

After her admission to hospital the patient admitted having been a regular benzine sniffer when she was 17, but maintained that she had since abandoned the habit. She had bought benzine from a chemist shop, at 8c a small bottle, and had inhaled the fumes as described above. Four of her friends did likewise, and they had sniffed about once a day; as far as she knew the friends had remained well. The patient did not smoke tobacco or dagga or take liquor.

This patient had the features of a pure motor neuropathy which seemed to improve slowly. She was ultimately sent back to the referring hospital for further physiotherapy.

Case 5
A 13-year-old boy was referred from a peripheral hospital because of weakness of the hands and legs, thought to be due to myopathy. He was unaccompanied by a parent, and his own history varied on different occasions. He said that his complaints had developed over a period of 1 week about 2 months previously, and since that time had not worsened.

On examination the only abnormalities were in the hands and legs, where he had slight peripheral wasting and gross peripheral weakness with bilateral wrist- and foot-drop. All tendon reflexes were absent and there were no plantar responses. He was able to walk, but with a high-stepping gait. Sensation was normal.

Biochemical investigations did not contribute further to the diagnosis. In particular, the CSF was normal with a protein content of 0.20 g/L. The creatine phosphokinase and aldolase levels were slightly elevated.

The features of this case were those of a pure motor neuropathy. Because of the patient's history of rapid development of weakness a diagnosis of Guillain-Barré syndrome was considered, but thought unlikely because the weakness was peripheral and the CSF normal. On further questioning he admitted having sniffed benzine on one occasion; subsequently he said that he had done so 'more than once', having stopped sniffing because it 'made him giddy', but he was always evasive and reluctant to answer questions on this theme. He was given orthopaedic appliances to relieve his foot-drop and discharged so that he could attend school.

Case 6
A 14-year-old boy was admitted complaining of pain and weakness of the knees and ankles which had developed over 5 days and which he said was progressive. On examination he walked with a high-stepping gait and there was bilateral foot-drop. He had distal weakness in the arms and particularly in the legs. Muscle tone was reduced and all tendon reflexes were absent. There was no sensory deficit.

All investigations were negative. The CSF was normal, with a protein value of 0.24 g/L, and the creatine kinase level was normal.

This boy displayed the clinical features of a pure motor neuropathy, the cause of which was unclear. On admission to hospital he denied using alcohol and benzine, but admitted that he occasionally smoked tobacco. Subsequently, when questioned further, he admitted having sniffed benzine 'many times a day', but said that this had been 3 years previously and then only for 6 weeks. The condition did not seem to progress while the patient was in hospital. He was discharged at his own request so that he could attend school, and was referred for outpatient observation.

Case 7
A boy aged 14 years said that his illness had started with pain and weakness of the legs in about 2 months before. It had become rapidly worse when he had fallen while walking to a shop, and since then he had had difficulty walking. He had never been ill before.

On examination the only abnormalities were in the limbs. There was pronounced distal weakness in the arms and legs and bilateral foot-drop, with a high-stepping gait. All tendon reflexes were absent. Sensation was entirely intact.

Serum creatine phosphokinase and aldolase levels were slightly elevated. The CSF was normal, with a protein content of 0.24 g/L, and all other investigations were negative.

The patient had the clinical features of a pure motor neuropathy. In the search for a cause he was repeatedly questioned regarding exposure to drugs and chemicals, which he denied. Ultimately he did admit that he had sniffed benzine, but denied that this had been a regular habit.

Case 8
A 12-year-old boy complained of weakness of the legs for about 6 months. This had initially progressed slowly, but he thought that some improvement was beginning to occur. The arms were unaffected and bladder function was normal. There was no history of significant previous illness.

On examination he had a high-stepping gait, which was not very pronounced, and there was weakness of dorsiflexion of the ankle and extension of the toes. Knee and ankle jerks were absent, but plantar and abdominal reflexes were normal. Power and reflexes were normal in the arms; there was no sensory loss in the arms or legs or in the saddle area. Radiographs of the spine were normal except for features of spina bifida occulta at the first sacral vertebra. The blood count was normal, as was the CSF.

At first the patient denied all knowledge of benzine sniffing, but later, when he was persuaded that he would not be punished, he said that he had sniffed benzine about once a week for a month before the onset of symptoms. His story varied, however; later he admitted having sniffed daily for several months. The benzine had been bought in small bottles from the local shop, and inhaled in the usual way. The patient also drank a mugful of Zulu beer every day. He had 2 friends who shared the benzine, and said that they had not been affected in any way.

This patient had definite signs of motor neuropathy, although he said that he was improving at the time of his admission to hospital. There seems to be little doubt that he had sniffed benzine, but the details regarding how often he did so were so variable that they should be accepted with some reservation.

Discussion
The patients described all displayed a fairly typical predominantly motor peripheral neuropathy. Only 1 child showed slight peripheral sensory deficit. In 2 of the patients in whom nerve conduction studies were carried out slow velocities were found, suggesting a demyelinating process. Although other diagnoses were considered, such as infectious polyneuritis and distal myopathy, these seemed unlikely on clinical examination and further investigation. The one common factor in these cases was a history of long-term inhalation of benzine, and it is therefore reasonable to assume that benzine was in some way responsible for the peripheral neuropathy. In the 1 case in which follow-up was possible the weakness had disappeared 1 year after stopping the habit of benzine sniffing.

Most of the children at first denied and in fact seemed frightened to admit to benzine sniffing. It was only after being assured that they would not be punished that they freely admitted to it.
Benzone (not to be confused with benzene) is a petroleum distillate consisting principally of n-pentane and n-hexane, with a boiling point range of 35-80°C. No lead is present (P. A. S. Canham — personal communication).

n-Hexane is regarded as a 'low-toxic' industrial compound, yet numerous reports have clearly demonstrated that it can cause damage to both the central and peripheral nervous systems. In large doses it may cause central nervous system depression.

In 1968, Sobue et al. reported on 93 cases of polyneuropathy among sandal workers in a cottage industry in Japan, thought to be due to n-hexane, and Herskowitz et al. described 3 adults working in a furniture-finishing operation who developed neuropathy caused by n-hexane. In both these reports the initial symptoms were predominantly sensory and included numbness, paraesthesiae and tenderness; motor signs only appeared later.

In our patients sensory loss was minimal, but their histories were probably inaccurate, since most of them were either referred from another hospital or unaccompanied by a relative. A report by Takeuchi et al. described gait disturbances in labourers who were exposed to petroleum benzine. Reports of polyneuropathy due to glue sniffing have also appeared. Again n-hexane seems to be the hydrocarbon responsible, yet, in contrast with reports on the polyneuropathy caused by industrial exposure to hydrocarbons, these glue-sniffing patients, like ours, had a predominantly motor neuropathy with very little sensory disturbance. Suzuki suggests that perhaps the presence of other hydrocarbons, especially toluene, may inhibit n-hexane metabolism and modify the clinical picture. The glue solvents were largely n-hexane and toluene in approximately equal amounts.

The effect of n-hexane on peripheral nerves has been studied in animals. Kurita exposed rats to n-hexane for 143 days, after which microscopic examination of peripheral nerves showed degeneration of myelin and axon cylinders in the sciatic nerves. Truhaut et al. exposed rats to 'technical-grade hexane' (45.8% n-Hexane) and found decreased conduction velocity, increased refractory time and decreased excitability of the sciatic nerves, and Schaumberg and Spencer showed that rats intoxicated with pure n-hexane consistently developed abnormalities in the peripheral and central nervous systems, weakness and gait disturbance developing after 45 - 69 days of exposure. The characteristic structural abnormality was giant axonal swelling in the myelinated nerve fibres.

The pathological changes in nerves of human subjects exposed to n-hexane have ranged from destruction of the myelin sheath with well-preserved axons in those cases caused by industrial exposure to axonal distension by neurofilamentous masses and axonal loss in glue sniffers, in whom the changes were very similar to those of acrylamide neuropathy, suggesting a 'dying-back' phenomenon. The varied pathological changes reported may be due to other hydrocarbons which have a modifying effect, as was suggested for the difference in clinical signs in glue sniffers and those exposed to industrial hydrocarbons.

Conclusion

Benzone, which is readily available from corner stores in most of South Africa, has a high concentration of n-hexane, an important cause of polyneuropathy in those exposed to it chronically. Benzone sniffing is a common practice among Black children in Natal, and every effort must be made to combat this form of solvent abuse by restricting its sale and by a concentrated attempt at health education at schools and through the media.

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REFERENCES


Afbeelding van die miokardium met tallium-201

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Summary

Myocardial imaging after the intravenous injection of radionuclides offers a non-invasive, highly sensitive and reliable method for the detection of myocardial infarction and regional myocardial ischaemia. Considerable attention has been given to various isotopes as a means of independent assessment of regional blood flow distribution in the normal, ischaemic or infarcted myocardium.

The clinical use of cationic tracers such as potassium-43 and rubidium-81 is based upon the principle that tracer uptake is proportional to regional myocardial blood flow. A region of absent tracer uptake suggests the presence of previous infarction or fibrosis, while a new perfusion defect appearing after stress suggests transient ischaemia. These isotopes, however, have significant physical limitations as imaging agents. The physical and biological properties of thallium-201, however, make it ideally suited for imaging of the myocardium with currently available imaging equipment.

REFERENCES