Sciatic nerve palsy associated with intramuscular quinine injections in children

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The purpose of this paper is to show that, in children, gluteal injection of quinine dihydrochloride (QDH) may result in damage to the sciatic nerve.

Forty-six children were seen with foot drop following intramuscular injections in the same limb. They were analyzed for the type of injection, injection site, route of injection, the Health Unit where the drug was administered and the personnel that administered the prescription, the type of paralysis and its duration. In 22 children, QDH was the principle drug administered. Of these, five (23%) had a sciatic palsy that persisted for at least one year. As well as the sciatic nerve palsy, some of the children developed other complications including equinovarus deformity, leg length discrepancy and trophic ulcers. We conclude that intramuscular injections of quinine dihydrochloride into the gluteal muscles in children can cause sciatic nerve palsies and that unfortunately some of these may be permanent.

Introduction

Adverse reactions may be produced by many drugs and this is certainly true with quinine dihydrochloride[1] which is an alkaloid derived from Cinchona Bark. It was introduced into Europe from South America in 1633 and has been used for the treatment of many fevers. It was regularly used against malaria until Mepacrine appeared in 1939[2], but it lost its popularity largely on account of its adverse effects which include ototoxicity resulting in tinnitus, deafness and vertigo (Cinchonism), visual impairment and even complete blindness which may arise very suddenly[3]. Other side effects include skin rashes, nausea, vomiting and diarrhoea with abdominal pain, fever, hypertension, convulsions and respiratory distress. Rarely, it causes ventricular tachycardia. Intramuscular injections of QDH can form abscesses but sciatic nerve toxicity has not previously been reported.

In spite of these adverse reactions, the use of quinine for treatment of malaria has recently become much more popular in Uganda because of the emergence of chloroquine-resistant malaria. It is also used intravenously for severe malaria[4] and is given very slowly by this route when there is nausea and vomiting. However, its administration by gluteal intramuscular injection in children has resulted in damage to the sciatic nerve.

Patients and methods

We made a prospective study of all children with foot drop after a gluteal intramuscular injection who presented to a special clinic at the Round Table Polio Clinic, Mulago Hospital in one year (1996-1997). As the result of a circular sent to all Health Units in Uganda, we received patients from all over the country. Clinical and stool examination, together with a history of adequate examination were used to exclude patients with poliomyelitis.

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Results

During one year there were 59 children with sciatic nerve palsy among a total of 311 who attended. Thirteen of these 59 were excluded as they defaulted before adequate information had been gathered. Another 24 had received more than one drug injection and therefore also had to be excluded. Thus there were 22 children with a sciatic nerve disorder following gluteal intramuscular injection of QDH.

There were 19 children under five years of age and three between five and ten years (Fig 1). Unfortunately the exact site of the gluteal injection could rarely be identified. It was recorded as the upper and outer quadrant in one, the centre of the buttock in three and was unknown in 17. The symptoms of sciatic nerve damage appeared immediately in 15, in less than six hours in one and after six hours in six (Fig 2).

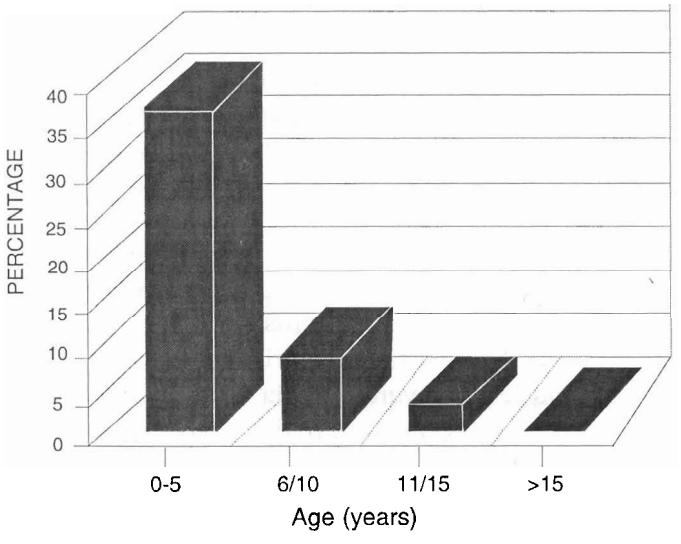

FIG 1 *Age distribution*

The presenting symptoms are listed in table I and the physical signs in figure 3, with most showing a combined motor and sensory deficit. Recovery within one year is recorded in figure 4, from which it is evident that some 15 of the 22 (68%) still showed signs of nerve damage at that time.

Although most patients showed some signs of neurological recovery during the survey (Fig 4), 10 patients still had marked deficit after one year and in five of these it was complete motor and sensory palsy.

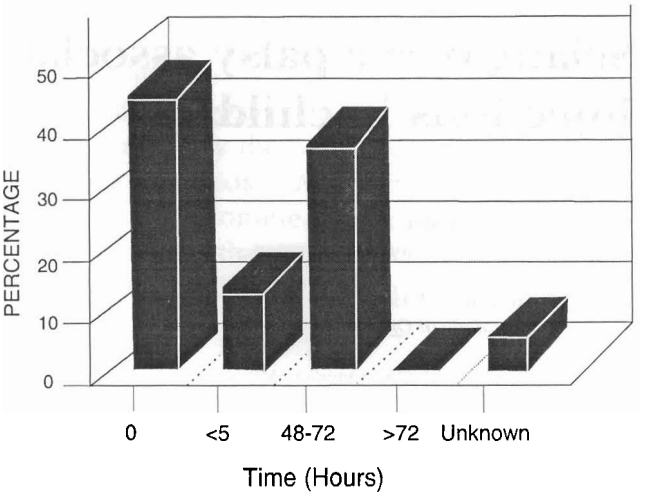

FIG 2 *Time of onset of paralysis*

TABLE I *Symptoms*

Symptoms	Patients	%
Hyperaesthesia	12	(23)
Foot drop	9	(17)
Other muscle weakness	9	(17)
Numbness	7	(13)
Pain	7	(13)
Deformity	4	(8)
Ulcerations	2	(4)
Pins and needles	2	(4)
Itching	1	(2)
TOTAL	53	

Additional persistent complications included foot drop, trophic ulcers, and limb shortening (Table II).

TABLE II *Other persistent complications*

Complications	Total+	%
Deformity	7	(32)
Trophic Ulcers	3	(14)
None	12	(55)
TOTAL	53	

FIG 4 *Neurological recovery*

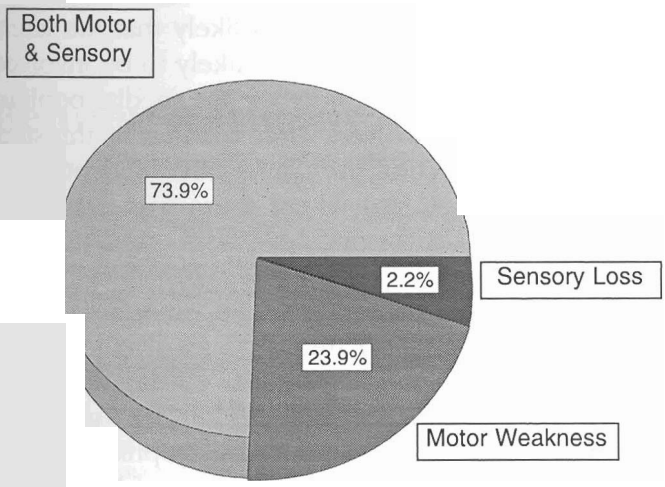

FIG 3 *Neurological signs*

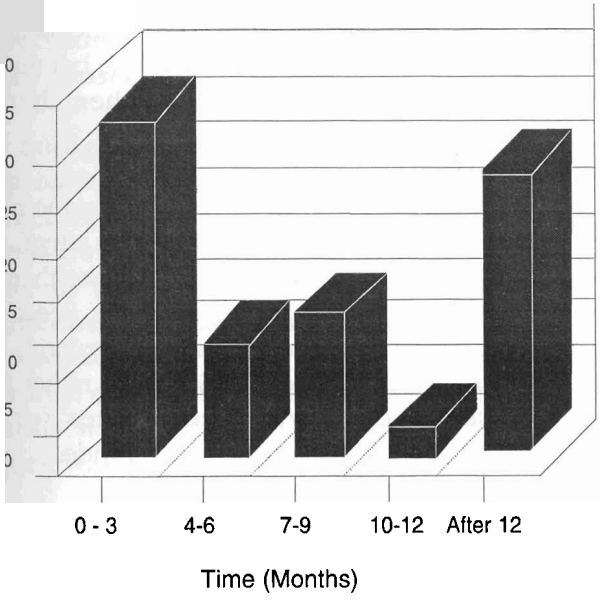

FIG 4 *Neurological recovery*

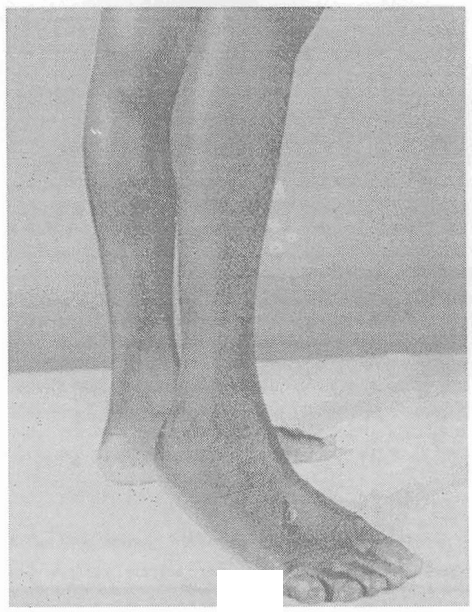

FIG 5 *Eight-year-old child with right equinus deformity*

Aspirin was given for the pain. An ankle brace and stretching exercises for the right ankle joint were prescribed. He was then followed up at monthly intervals. At one year, he still had a right foot drop (Fig 6) and had no sensory recovery.

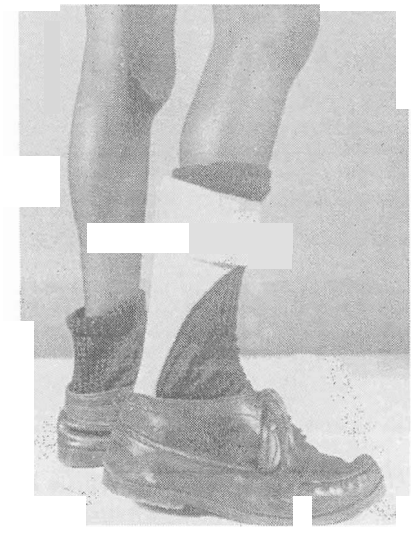

FIG 6 *Same child treated with ankle brace*

Case presentations

1 An eight-year-old boy was given an intramuscular quinine injection into the gluteal muscles of his right buttock. He developed a right foot drop 48 hours after the injection and had associated intense pain in the right foot. On examination he had weakness of the dorsiflexors and evertors of the right foot and associated sensory loss on the lateral border and plantar surface of the foot (Fig 5).

2 This two-year-old baby, who was given intramuscular quinine at the centre of the right buttock, immediately developed weakness and sensory loss in his right foot (Fig 7). He was prescribed analgesics, an ankle brace (Fig 8) and stretching exercises. He had not recovered either sensory or motor function one year later and had

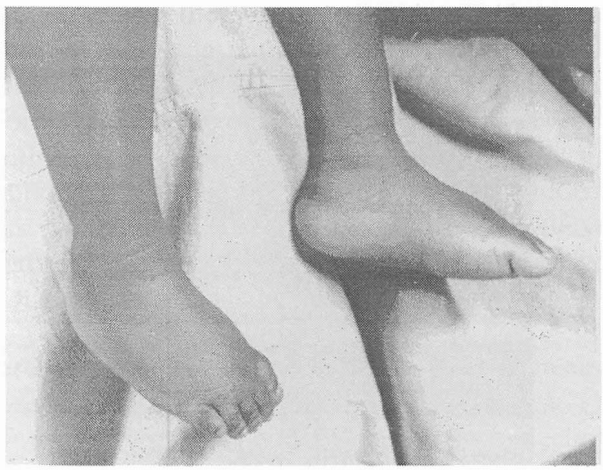

FIG 7 *Two-year-old child with right foot drop*

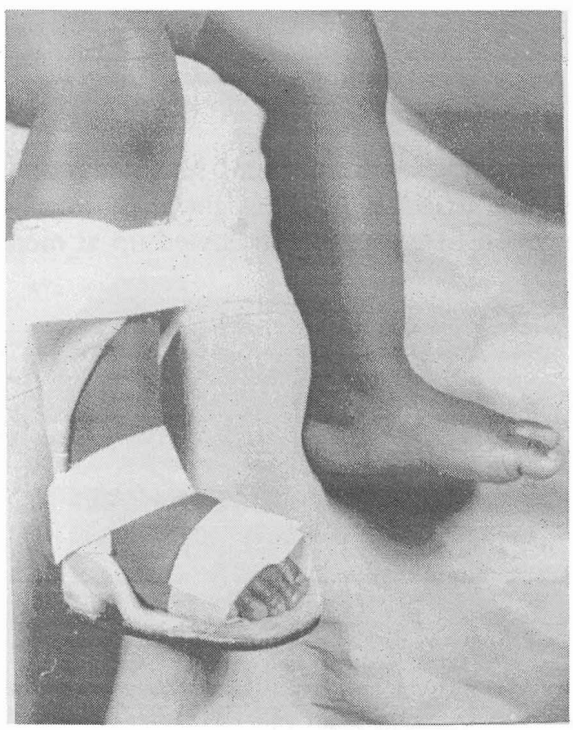

FIG 8 *Same child fitted with ankle brace*

developed trophic ulcers on the plantar aspect of the big toe and second toe.

Discussion

Quinine dihydrochloride must be recognised as one of the important drugs which may result in permanent sciatic nerve palsy after gluteal intramuscular injection[5]. Further anatomical studies are needed to determine how and where this damage occurs but it seems likely that the lateral popliteal component is most likely to be involved. A bad prognosis exists when the medial popliteal component is involved. Unfortunately, in this study the actual injection site could only be identified in five children (23%) where there was a palpable intradermal scar or nodule.

The common symptoms of hyperaesthesia (23%) and foot drop (17%) are due to involvement of the lateral popliteal component. When the nerve damage is permanent the children may develop a fixed equinovarus deformity and trophic ulceration of the tips of the toes. One third of the patients were showing some signs of recovery within three months but five out of 22 (23%) had not recovered after a year.

There is no specific treatment although it is important to maintain joint movement and avoid damage to anaesthetic skin. An ankle brace may be helpful. There is no real evidence that exploration of the sciatic nerve with or without excision of the damaged portion and nerve grafting is of any use but tendon transfers should be considered when there is permanent residual paralysis.

Avoidance of injecting drugs into the sciatic nerve is vital and it is recommended that the lateral side of the thigh is a much less dangerous site than the gluteal region for giving intramuscular injections. In severe and chloroquine resistant malaria, it is safer to use slow intravenous infusion rather than intramuscular (or intraneural) routes for the administration of quinine dihydrochloride.

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