Quinine Intoxication in a Child Treated by Exchange Transfusion

Quinine poisoning is rare in children. The mortality rate is high and in patients who survive serious intoxication there may be residual blindness and deafness. The following report shows that in childhood, exchange transfusion is an effective treatment.

Case Report

An 18-month-old Jamaican girl was brought to hospital 3 hours after being found at home eating her mother's quinine sulphate tablets (300 mg). She was drowsy and twitching. A gastric washout retrieved a considerable quantity of tablet particles. The dose of quinine taken is unknown.

The child gradually became less responsive and the twitching became generalized. An intravenous infusion was begun in an attempt at forced diuresis. The fits were controlled by repeated doses of intravenous diazepam. A plasma level of quinine 4 hours after the ingestion of the tablets was 0.49 mg/100 ml. No other toxic substances were detected in blood or urine.

In spite of treatment the child's condition continued to deteriorate. She became unrousable, her pupils became dilated and fixed, no reflexes could be obtained, and the fits were no longer controlled by diazepam. A lumbar puncture was performed, and this produced normal CSF.

In view of the clinical deterioration, it was decided to carry out an exchange blood transfusion, despite the apparently low levels of circulating quinine. In the course of 3 hours 1800 ml whole blood was exchanged using a central venous catheter. The plasma quinine levels estimated during the exchange transfusion are shown in the Fig. The quinine level in the CSF is also shown. The child stopped convulsing after 1000 ml blood had been exchanged and she became more responsive. Her blood pressure remained about 90/50 mm Hg throughout the exchange transfusion.

Twenty-four hours after admission the girl was alert, but unresponsive to sound and to light, her pupils remaining fixed and dilated. 5 days later, however, she had regained her hearing, and her visual acuity on formal testing was estimated as 6/12.

Comment

The signs and symptoms of quinine overdose are well described (Goodman and Gilman, 1970). Tinnitus, tremor, vomiting, and hypotension occur early, rapidly followed by convulsions and loss of consciousness. Some individuals display an idiosyncratic hypersensitivity to quinine, and may show toxic manifestations at therapeutic plasma levels. In the present case, despite recovering tablet particles by gastric lavage, the child lapsed into unconsciousness and began to convulse within 3 hours of ingesting quinine. This presumably reflects the fact that quinine is rapidly absorbed from the stomach and small intestine, maximum plasma levels being reached within 4 hours.

Quinine in therapeutic doses is rapidly metabolized and is excreted in the urine within 24 hours (Goodman and Gilman, 1970). The slow fall in the level of blood quinine in this child, before exchange transfusion, suggests that its clearance may be impaired at toxic levels, or that she continued to absorb quinine from the intestine. Some 70% of quinine in the plasma is bound to plasma proteins (Goodman and Gilman, 1970).

Therapeutic levels of plasma quinine in adults are in the region of 0.7 mg/100 ml. In the present case, the maximum level of plasma quinine recorded (0.49 mg/100 ml) was considerably less than the levels of 26–33 mg/l. quoted by Hillman and Harpur (1961) in their report of exchange transfusion in the treatment of quinine poisoning. An

---

**Fig.**—The concentrations of plasma quinine in a child before and during exchange transfusion for the treatment of quinine intoxication.

---


QUTUB H. QAzi* and MARGARET W. THOMPSON
Downstate Medical Center, Brooklyn, New York, U.S.A.; and The Hospital for Sick Children, Toronto, Canada.

*Correspondence to Dr. Q. H. Qazi, Department of Pediatrics, Downstate Medical Center, 450 Clarkson Avenue, Brooklyn, N.Y. 11203, U.S.A.
explanation of this discrepancy may be that the child was hypersensitive to quinine. However, exchange transfusion, which is the logical method of treatment, produced both a dramatic improvement in the child’s clinical condition and a fall in plasma quinine concentrations.

Quinine does not readily cross the blood-brain barrier (Goodman and Gilman, 1970), but in the present case the concentration of quinine in the CSF was about 35% of that in the plasma. It is generally thought that quinine has a direct toxic action on the retina which may take months to recover. In the present case it is of interest that, though the child was initially both deaf and blind, she recovered both faculties within 5 days.

**Summary**

An 18-month-old child became unconscious and convulsed within 4 hours of taking an unknown number of quinine sulphate tablets. Her clinical condition did not improve, and an exchange blood transfusion was carried out. The plasma quinine concentrations fell, and the child stopped fitting and regained consciousness during the procedure. She made a rapid recovery from the transient deafness and blindness which occurred with quinine intoxication.

The plasma levels of quinine were kindly estimated by the Poisons Centre, Guy’s Hospital, London.

**References**


A. W. Burrows, G. Hambleton, M. J. Hardman, and B. D. R. Wilson*  
Children’s Department, St. Thomas’s Hospital, London S.E.1

---

*A Correspondence to Dr. B. D. R. Wilson, St. Thomas’s Hospital, London S.E.1.

**A Case of Fetus in Fetu**

Meckel (circa 1800) described a condition where a parasitic twin was found included within the abdomen of its partner and called it *fetus in fetu*. Willis (1958) pointed out the separate natures of *fetus in fetu* and retroperitoneal teratoma, the difference being that the latter is a true tumour, while the former is not. Since Young reported a detailed study of a case in 1809, only sporadic case reports have appeared from time to time, 14 cases having been traced in the 20th century. Nearly all the cases have been intra-abdominal.

**Case Report**

A 20-month-old Sinhalese girl was admitted with a history of abdominal distention of 2 months’ duration. She had been born normally, with a birthweight of 2.7 kg. She had two elder brothers and a younger sister who were quite normal. There was no history of consanguinity, or of twin pregnancy.

She was 8.2 kg in weight, 77.5 cm tall, and appeared well nourished. She was mildly anaemic but not jaundiced. The abdomen was distended, particularly on the right side, and there were prominent veins in the epigastrium. There was an ill-defined lobulated firm lump extending from the right hypochondrium to the right lumbar region. It did not move with respiration, was slightly mobile laterally, and was not ballotable. The liver was palpable 2.5 cm below the right costal margin, but the lump could be felt apart from the liver.

Hb was 6.6 g/100 ml. Her urine showed a trace of protein and RBC 30/mm³. Liver function tests were normal. ESR 2 mm/1 hr, urea 25 mg/100 ml. Blood group O Rh positive.

The plain x-ray of her abdomen (Fig. 1) showed bony structures within the intra-abdominal lump. An IVP showed that the lump was not connected to the kidneys and both kidneys functioned normally. A preoperative diagnosis of *fetus in fetu* was made from the x-ray appearances.

At laparotomy (P.R.W.) through a transverse incision, the fetus was found enclosed in a sac between the liver and the right kidney with the structures of the porta hepatis, spread over it. Its pedicle arose from the posterior abdominal wall, and had blood vessels on the surface, which continued over the wall of the sac. The fetus was

---

![Fig. 1.—A plain x-ray of the abdomen of patient (oblique view), showing the bony structures within the right upper abdominal region, preoperatively.](http://adc.bmj.com/)

---

*Short Reports*  305