Medical Memoranda

Intracranial Hypertension in a Child during Treatment with Nalidixic Acid

A case of bulging fontanelle, papilloedema, widening of skull sutures, and vomiting in a 6-month-old boy after nalidixic acid therapy is described. The symptoms occurred on two different occasions when he was treated with the drug for a urinary tract infection, and subsided rapidly when the medication was discontinued. The suspicion of a connexion between the nalidixic acid and the increased intracranial pressure was confirmed when, on a third occasion, the same symptoms could be provoked after administration of the drug under carefully controlled conditions.

Case History

Episode 1 (Fig. 1).—A previously healthy 6-month-old boy weighing 6,480 g. was treated at another hospital with nalidixic acid (100 mg. orally four times daily) for a urinary tract infection. On the third day of treatment he vomited, and on the fourth day was found to have a bulging anterior fontanelle. There was a slight papilloedema of the right side. No explanation for this increase in intracranial pressure was found and he was admitted to the hospital, where x-ray examination of the skull showed sutures wider than normal. Electroencephalography, echoencephalography, pneumoencephalography, and blood pressure were normal and he was afebrile. High pressure in the subarachnoidal space was found on bilateral exploration (operation 1). The fontanelle was normal after the operation.

Episode 2 (Fig. 1).—Two weeks after the first symptoms the patient was discharged to the other hospital, where treatment of the urinary infection was resumed with nalidixic acid in the same dose as before. On the evening of the first day bulging over the trephine hole on the left side was noted. On the second day a bulging fontanelle appeared and he was readmitted to this hospital. There was a slight papilloedema on the right side. The general condition of the boy was quite good. A puncture was made through the trephine hole (operation 2), but the bulging reappeared rapidly. The cerebrospinal fluid was sterile. Nalidixic acid was now suspected to be the cause of the symptoms and was discontinued. Next day the bulging over the trephine hole and over the fontanelle had disappeared. Two weeks later the eye-grounds were normal. Urological examination showed a hydronephrosis and a stenosis of the ureter on the left side. Non-protein nitrogen and creatinine clearance were normal. Urological surgery was postponed until the cerebral symptoms could be explained.

Episode 3 (Fig. 2).—Three weeks after the end of episode 2 the boy was again admitted to this hospital. He was then 8 months old, weighed 8,195 g., and was in excellent condition. The fontanelle, eye-grounds, and physical and neurological examinations were all normal and no bacteria were present in the urine. Nalidixic acid was again given and the plasma levels of total free extractable drug were determined by spectrophotofluorometric assay according to McChesney et al. (1964). The analyses were kindly run by Mr. A. Robertson, of Winthrop Laboratories, Newcastle upon Tyne. The fontanelle was checked at short intervals. Signs of increased intracranial pressure occurred soon after the onset of medication. The patient vomited twice within the first 24 hours. A tense fontanelle was noted after about 36 hours and was definitely bulging and pulsating after 48 hours. A slight papilloedema on the right side developed at about the same rate. The medication was immediately discontinued. One day later the fontanelle was normal.
Mendelson's Syndrome: Its Treatment by Tracheostomy and Hydrocortisone


Hall (1940) described the cases of 15 patients who had inhaled vomitus during or after anaesthesia for childbirth. He described two types of case: (1) those patients in whom the inhaled vomitus consisted of solid material; this caused mechanical obstruction of the airway; and (2) those in whom the inhaled vomitus was entirely liquid; these latter patients developed marked cyanosis, tachycardia, and tachypnoea several hours later.

Mendelson (1946) also noted the two types of reaction, depending on the quality of the vomitus, which had been noted by Hall. When the vomitus contained solid material there was an acute airway obstruction with massive collapse of the lung. When the vomitus was liquid there developed, after a latent period of one to two hours, a syndrome of dyspnoea, cyanosis, tachycardia, and in Mendelson's cases generalized bronchospasm. Mendelson was able to cause a similar syndrome in rabbits by instilling either N/10 hydrochloric acid or acid gastric juice into the tracheobronchial tree. When distilled water, neutralized gastric juice, or 0.65% sodium chloride solution was used the syndrome did not develop (Mendelson, 1946).

Others have confirmed that pulmonary oedema may develop after the inhalation of acid gastric contents (Hartzell and Mininger, 1946; Parker, 1954; Hausmann and Lunt, 1955).

The presence of oedema at the blood/gas interface in the lungs may upset gaseous exchange with the atmosphere, and if extensive it may be fatal. It is now recognized that tracheo-

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**References**