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CONGENITAL HYPERTROPHIC PYLORIC STENOSIS IN THE AFRICAN

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Congenital hypertrophic pyloric stenosis in the African appears to be an extremely uncommon condition, since very few cases in the African have been reported in the literature. Ladd, Ware and Pickett[1] state that they have noted no particular racial disposition, and ascribe the fact that they have never observed a case in a Negro infant to pure chance. Donovan[2] reported 2 cases in Negro infants, and later[3] he states that he has found the condition in all races and nationalities and he has seen about 2 Coloured babies with this condition in each 100 cases. Many authors have stated that the condition is rare in Negro infants. Benson and Warden[4] describe the condition in 63 Coloured infants out of 707 consecutive cases reviewed by them during a 16-year period from 1940 to 1955 at the children's hospital of Michigan.

Before 1955 the condition had not been described in the African infant. Shepherd Wilson and Gelfand[5] described the first case in an African male aged 2 months. The following year Luder[6] reported another case in a male Ganda infant aged 1 month. Since then, at Johannesburg, Griffiths[7] has described 1 proved case seen at Baragwanath Hospital in a 1-month-old Bantu male and has stated that a single case, which was treated medically, was seen at Coronation Hospital. Hamilton[8] stated that the literature appeared to contain reports of only 4 cases of congenital pyloric stenosis occurring in Africans, and he described another proved case in a male Dama aged 1 month. Unfortunately the references to these 4 cases were not published with his article, but it would appear that he was referring to the 4 cases described by the abovenamed authors. Thus to date only 5 cases, 4 of which were proved by operation, have been described in Africans.

During a period of 6 years at King Edward VIII Hospital, Durban, this diagnosis has been made on 3 occasions in the Bantu paediatric department. This department has a very large turn-over, both in the in-patient and out-patient section, and the fact that only 3 cases of pyloric stenosis were diagnosed out of an admission rate of approximately 5,000 cases a year, is an indication of its rarity. It seems unlikely that the diagnosis is missed, for the condition has never been discovered as a chance finding at autopsy.

As the condition is usually slowly progressive and does not cause sudden death, presumably the affected infant at some stage would be brought to the out-patient department. The African mother has become increasingly hospital-conscious, and thousands of infants attend for such complaints as failure to thrive, vomiting and constipation; this latter symptom being particularly distressing to the enema-conscious African mother. It is unlikely therefore that cases of pyloric stenosis in any numbers are dying outside the hospital.

There was 1 proved case in each of the years 1953, 1954 and 1956, and these are summarized briefly as follows:

Case 1

A 4-week-old African male was admitted on 24 June 1953. He was a first-born child, his birth was normal and he was breast fed. His birth weight is unknown. The mother, a pure African who came from the kraal, was extremely 'raw' and was unable to give a reliable history even with the help of an interpreter. It appeared that the infant was subject to persistent vomiting and constipation, but the duration could not be ascertained. I was able to interview the father and was satisfied that he too was a pure-bred African.

The infant weighed 6 lb. He was small and miserable, and there was depression of the fontanelle, but skin turgor was satisfactory. All systems were apparently normal. No peristaltic waves were observed after feeding and no mass was palpable. Test feeding was carried out. After 48 hours it was obvious that the infant was vomiting most of what he took in. The vomiting was not projectile in type, but on the following day on 1 occasion it was noticed to be so. Still no mass was palpated. The stomach was washed out and Eumydrin (1:10,000 solution) was given in a a dose of 2·5 c.c. 20 minutes before each feed. For the next 48 hours the vomiting lessened and the infant appeared to be improving. At this stage, after a feed, gastric peristalsis was visible and a pyloric tumour was palpated. After this examination projectile vomiting occurred again. Eumydrin was tried for a further period of 48 hours. Breast milk was now inadequate so that complementary feeds were required. Since dehydration was developing and practically nothing was being passed *per rectum*, operation was deemed advisable. The diagnosis of pyloric stenosis was confirmed at operation and Rammstedt's pyloroplasty was performed.

The post-operative progress of the infant was satisfactory for 16 days when, unfortunately, he developed bronchopneumonia which failed to respond to antibiotic treatment, and he died 2 days later.

Comment. This case tragically illustrates the danger of keeping these babies in hospital longer than is absolutely necessary, because there is the ever-present risk of cross infection. In this case we were influenced by the fact that the

mother was primitive and had to return to a distant kraal with an artificially fed infant who had not improved as well as we had hoped. Had we been able to discharge him early and follow up his case outside the hospital, this death from bronchopneumonia might well have been prevented.

Case 2

A 8-week-old African male was admitted on 10 May 1954. He was a first-born child, his birth was normal and he was breast fed. His birth weight was unknown. Both parents were pure Africans. The infant was well until he was 5 days old when he began vomiting a little after each feed. The vomiting was not projectile in type from the beginning, but had been so for the past 5 weeks. Stools were passed normally for some time after birth, but lately there had been marked constipation and the mother said many days would go by without a stool being passed, and then only a very small quantity would be passed.

His weight was 7 lb. 5 oz. Mild dehydration was present, but there was evidence of marked weight loss. The infant was alert with a lusty cry. All systems were normal apart from the gastro-intestinal system. The abdomen was not distended. He was given a breast feed, and after this peristaltic waves were seen moving from left to right, and an easily palpable tumour was detected on the right side just below the costal margin. The surgeon consulted agreed on the diagnosis. The administration of intravenous fluids were started, and the stomach was washed out and left empty. Operation was performed and the diagnosis was confirmed. The infant made an uninterrupted recovery and was discharged 8 days after operation weighing 8 lb. 6 oz.

Case 3

A 4-week-old African male was admitted on 27 December 1956. He was a first-born child, his birth was normal and he was breast fed. His birth weight was unknown. Both parents were pure Africans. The infant was well until he was 5 days old, when he started vomiting after feeds. The mother described the vomiting as 'shooting out'. The infant never passed a stool unless given an enema.

His weight was 6 lb. 5 oz. He was thin and there was evidence of some dehydration. All systems were normal apart from the gastro-intestinal system. The abdomen was soft. Visible peristalsis was present. It was thought that a pyloric tumour was palpable. Rectal examination revealed a trace of green formed stool. This was the state of affairs on admission in the evening. Throughout the night frequent small feeds of Hartman's solution were given. The following morning, 12 hours later, it was reported that the infant had vomited each feed of Hartman's solution. No stool had been passed. Mild dehydration was still present. A bottle of Hartman's solution was offered and while sucking this, typical peristaltic waves were seen, and now a pyloric tumour was definitely palpated, followed by projectile vomiting. Intravenous fluids were given and the stomach was emptied. Operation confirmed the diagnosis. By the fifth day after the operation the infant was fully breast fed, in excellent condition and improving. On discharge on the ninth day his weight was 7 lb. 6 oz.

SUMMARY

Three cases of congenital hypertrophic pyloric stenosis in Africans are recorded. The condition would appear to be extremely rare in this race.

Note: Since completion of this paper 2 more cases of pyloric stenosis in African infants have been diagnosed in this department.

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