A Comparison of Infant and Adult Bantu Siderosis

By

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In a previous publication the author corroborated most of the findings of Higginson and his Rand colleagues (1953) on the frequency of siderosis and also on the distribution of the main deposits of iron in the viscera of the Bantu. They demonstrated that siderosis is common in the South African Native, that it probably causes no symptoms and therefore is distinct from haemochromatosis. They also found that the liver and spleen were the two internal organs most commonly affected, being followed by the intestine, abdominal lymph nodes, but rarely the pancreas, heart and skin which so commonly gives the Prussian Blue reaction for ferric iron in haemochromatosis. They failed to agree with the Gillmans that Bantu siderosis followed malnutrition, as they observed that even in bodies with good nutrition large amounts of the ferric iron were deposited in the viscera.

To explain this interesting finding in the Bantu the Rand workers claim to show that Bantu siderosis results from the ingestion over many years of excessive iron following the large amounts of maize consumed, and to a certain degree by cooking of their food and beer in iron utensils. This excessive intake of iron revealed itself by the deposition of ferric iron along the lines of food absorption, namely, in the small gut, abdominal lymph glands, liver, spleen and other organs of the reticulo-endothelial system. In the face of such excessive iron ingestion the mechanism whereby the body eliminates the iron breaks down. It follows that because of the high iron intake over the years siderosis is more likely to be found in the older age-groups than in infants and children in whom it is believed to be seldom encountered.

Higginson et al find additional support for the theory that siderosis is a consequence of the high iron content of the Bantu’s diet in the alleged rarity of hypochromic anaemia in tropical Africa, where an iron loss due to the hookworm and bilharzia parasites is compensated by the high iron intake. According to these authorities microcytic anaemia in Central Africa can be expected to be extremely uncommon, and arguing on the same lines they report that in the Union an iron deficiency anaemia in the Bantu is very rare.

Gelfand (1954) conducted certain investigations on this subject in Salisbury on Africans from Central Africa where hookworm, bilharziasis and malaria are endemic. His results were in the main identical with the Johannesburg workers, namely (1) that the liver and spleen were the two organs most likely to contain ferric iron, but the pancreas rarely; (2) that the disease may occur in well-nourished individuals. However, some of his findings were different. For instance, siderosis was fairly often found in infants and young children, and doubt was cast upon the suggestion that siderosis could be attributed to a high iron content of the diet as the infant in Mashonaland is fed almost solely on the breast, and only later will the mother commence to give him thin maize porridge.

The object of the present study was to compare the distribution of ferric iron in selected tissues of 50 infants with that of 50 adults.

PROCEDURE

In each case, whether adult, child or infant, the procedure was the same. A small amount of tissue about 1 inch long and ¾ inch wide was removed from the liver, spleen, pancreas, suprarenal, lung, heart, kidney and small intestine. These were placed in a clean glass Petri dish. Hydrochloric acid (N/10) was added to the specimens followed by 2 per cent. potassium ferrocyanide. The tissues were then inspected for any change of colour, and a greenish blue or deep blue colour was regarded as being positive for ferric iron.

RESULTS

Siderosis was found in 35 out of 50 infants (70 per cent.), whereas in the adults it was present in 42 out of 50 cases (84 per cent.). It would thus appear that while siderosis is common in the infant, siderosis in the adult is more frequently seen and that the condition becomes more common with an increase in age. In both the infant and the adult the liver, followed closely by the spleen, was the organ which showed this excess of ferric iron.

In the adult the liver was positive in 40 and the spleen in 34 out of 42 cases tested. In the infant out of 35 cases examined the liver was positive in 34 and the spleen in 27. Although in this respect the liver and spleen are commonly affected in the infant and adult, there is a marked difference between the frequency with which the other organs are affected. In the adult the lung, suprarenal, small intestine and kidney may not infrequently contain an
excess of ferric iron—usually not more than one or two or occasionally three in addition to the liver and spleen—whereas in the infant it is exceptional to find another organ besides the liver and spleen showing siderosis. This is best revealed by the following figures. Of the 42 adults showing siderosis in the liver and spleen, 18 had changes in other viscera, whereas in the 35 infants with siderosis in the liver and spleen, six showed it in the other organs tested. Further, it was far more common to find more than three organs affected in the adult, whereas this was not so in the infant. (Eight adults to 0 infants.)

The distribution of ferric iron in other organs when both the liver and spleen are positive is given as follows:

**Adults**

Forty-two subjects with siderosis of the liver and spleen.

- In ten cases three organs besides the liver and spleen were affected.
- In four cases four organs besides the liver and spleen were affected.
- In three cases five organs besides the liver and spleen were affected.
- In one case eight organs besides the liver and spleen were affected.

**Infants**

Thirty-five positive cases (liver and spleen).

- In six cases three organs other than the liver and spleen were affected.
- In no cases four organs other than the liver and spleen were affected.
- In no case five organs other than the liver and spleen were affected.

By comparing the frequency with which organs other than the liver and spleen are affected, we see readily the differences between the adult and infant.

**Adults**

- Lung 11, suprarenal 7, intestine 10, kidney 6, heart 1, pancreas 4.

**Infants**

- Kidney 1, intestine 1, suprarenal 3, lung 4.

It is clear from these figures that siderosis in the adult is much more widespread in the body than in the infant.

**DISCUSSION**

This work would tend to support the previous work of Higginson *et al* that siderosis in the Bantu is not, as far as we are aware, associated with any adverse effect and therefore is not responsible for any symptoms. This study shows that siderosis is also common in Central Africa and has a wider geographical distribution than the Union workers have postulated.

The cause of siderosis is in my opinion unknown. It does not appear to be due to the high iron intake of the African diet. It may be argued that because of malaria, which is endemic throughout the Federation, excessive amounts of iron are deposited in the tissues. In the present series the infants examined at autopsy came from the Salisbury African township, where malaria is to-day a rare disease. It is also doubtful if this disease would account for siderosis as it is seen in the South African Bantu from areas where malaria does not occur.

Siderosis in Central Africa is a common finding in the Africans as it is in the South African Bantu. It is also common in the infant, but its distribution in the body is far more widespread in the adult than in the infant. It is rare to find in the infant viscera other than the liver and spleen showing this phenomenon in contrast to the adult. In this respect this agrees with the Union workers, but it shows that the infant very often has siderosis. Reports so far from the Union indicate that it is rarely found at this age. The explanation for this is by no means easy. As siderosis was found in the neonatal period and the stillborn, one cannot attribute an excessive deposition of iron to a high maize intake, as it is later that the infant is given maize as a thin porridge. It suggests another explanation. One suggestion which comes to mind is that the foetus receives the deposits of ferric iron from its mother via the blood stream. But ferric iron is not ordinarily present in the blood of a patient suffering from haemochromatosis.

**REFERENCES**


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