POSSIBLE ENCEPHALITIS FOLLOWING B.C.G. VACCINATION

F. M. A. SAVAGE, M.A., B.M., B.Ch. (Oxon.)

Medical Registrar
Lusaka Central Hospital

During September/October 1966 intensive BCG vaccination campaigns were carried out in the schools of Lusaka. During this time we admitted to the wards of the Central Hospital, Lusaka, four young school children with an illness that we could only diagnose as a form of encephalitis. They were of markedly different severity, but all had in common recent BCG vaccination scars acquired during the campaign. None could give accurate dates for the vaccination, but in all cases it was "about 3 weeks," and the appearance of the scars was such as to suggest similar dates.

While our information on these cases is somewhat incomplete, it is felt that they are worth recording, so that others in a position to make similar observations may have these brought to their notice. At no other time during my stay in this hospital, nearly 1 year, have there been cases of encephalitis in this age group, and never more than two even suspected cases of encephalitis in any age group simultaneously.

CASE 1.

The first case that came to our notice was a young schoolboy of 16 years of age, admitted on 18 October 1966 with a five-day history of dizziness, headache, vomiting, and inability to breathe freely.

The illness began one morning when he "tried to get out of bed but couldn't." He had no history of previous illness, recent visits outside Lusaka, or of head injury. He had attended out-patients for 3 days but had felt no improvement.

On admission he had a temperature of 100 deg. F., which had fallen to 99 deg.F. next morning. Apart from this, and the BCG vaccination scar, he had no abnormal physical signs. He had no meningism or other neurological abnormalities, and his blood pressure was 100/60. When first examined he twice collapsed heavily on the floor, but with no alteration in his blood pressure or conscious state. He was due to sit exams in a few weeks' time, and his whole behaviour, and perhaps the wording of his history, led us initially to regard this as a primarily non-organic episode. In view of his mild pyrexia, he was treated with chloroquin and asprin overnight. On the following day, his temperature seemed to have settled, but he began to complain of the muscles at the back of his neck, which he said were pulling violently and would not let his head come forward. He appeared to have some neck stiffness, but this was not present a few hours later. On the third day his temperature rose again to 101 deg. F. A lumbar puncture was performed, which showed a cell count of 104 (60% neutrophils, 40% lymphocytes.) Protein content, glucose level, and pressure were all normal, and Kahn was negative. His blood urea was 29, blood sugar 87 mgm %, Hb 14.8, blood WBC 3,500 with a normal differential. His fever continued for seven days, and settled spontaneously without further treatment. He was discharged feeling well and behaving quite normally on 27 October. He did not return for follow up.

CASE II

The second case was admitted on 29 October. He was a 15-year-old schoolboy, with a 3-day history of dizziness, headache, and of shaking when standing. As in Case 1, he had no history of previous illness, or of head injury, and the only abnormal physical signs were mild pyrexia and recent BCG vaccination mark. The illness in this case was much milder, and he felt completely well within 48 hours of admission. At no time did he have any evidence of neck stiffness, and a lumbar puncture was performed only because of the apparent similarity to the first case. The CSF showed an identical picture, with a cell count of 118 WBC's, 30% neutrophils, 70% polymorphs. Pressure, glucose, and protein were all normal, and the Kahn negative.

CASE III

The third case was more serious than either of the above, and the encephalitic picture only appeared on a second admission. He first presented on 16 October, complaining of diarrhoea with small worms in it, and abdominal pain, present for 5 months, and of recent onset of palpitations and headache. He had a fever of 101.4 deg. F, vague abdominal tenderness and a palpable spleen. No pathogens were found in his stool, and blood culture was sterile. He was treated as an out-patient. He returned on the 27 October, complaining of headache and vomiting, and had fallen out of bed on the previous night. He was semi-conscious, with a tendency for his eyes to deviate to the left, and a suggestion that his right pupil was slightly bigger than his left. He had generally brisk reflexes, but his plantar reflexes were flexor. There was no meningism, and no definite evidence of neurological assymetry. His blood pressure was 120/70, blood sugar 124, urea 27, Hb 14.6. Lumbar puncture was performed, and the fluid showed a cell count of 88, 30% polymorphs, 70% lymphocytes. Again, pressure, protein, and sugar content were normal. An electroencephalogram showed diffuse slowing, compatible with encephalitis. He became very confused and restless, but without further treatment other than a routine chloroquin course, steadily improved. He continued ataxic and confused, but became well enough by 17 November to be discharged, and shortly after to return to school, though we fear he suffered permanent intellectual damage. His CSF at the time of discharge contained 11 cells, with a similar differential and normal protein content. He did not return for further follow up.

CASE IV

At the same time as these cases were in male ward, a girl of 8 years old was admitted to female ward, also with a 3-week-old BCG vaccination mark. She had a sister with proven TB, and was so ill that we

felt compelled to treat her from the outset as tubercular meningitis, but it occurred to us that she might conceivably be a member of the same group.

She was admitted on 15 November, confused and excitable, and with pronounced megingism and bilateral papilloedema. All 4 limbs were held rigid, but reflexes and plantar responses were normal. The cerebrospinal fluid contained 240 cells, 60% neutrophils, 40% lymphocytes. Protein content was 60 mgm %, pressure was markedly raised, but glucose content was normal.

She recovered on TB treatment with striking rapidity.

The striking absence of significant fever at any time during her admission, the absence of X-ray evidence of pulmonary TB, the normal CSF glucose content and her rapid recovery prevented us from being entirely happy about the diagnosis of TBM.

DISCUSSION

Clearly it is impossible to present a water-tight "case" for a casual relationship between vaccination and encephalitis, for the objections and alternative explanations are numerous.

Post-vaccinial encephalitis has usually been described following smallpox and rabies vaccinations, and has rarely been reported after injections of bacterial vaccines and foreign proteins. The usual time of onset has been 11 days following vaccination or injection. Despite numerous intensive BCG vaccination campaigns, the only widely recognised complications have been those due to local reaction, i.e. vaccination site ulceration and regional lymphadenopathy.

The cerebrospinal fluid findings would be well explained by the preparalytic stage of poliomyelitis, or by viral encephalitis. As we did not carry out any viral studies, we can neither confirm nor refute these two hypotheses, but neither alone could account for all cases. The remarkable concurrence of four cases of viral encephalitis could be explained only by chance, for, as mentioned previously, we seldom see more than one such case at any one time.

Under normal circumstances, the mildest of these cases would not have had a lumbar puncture, and we are presumably "missing" cases of preparalytic polio among the many brief febrile illnesses that are seen both in the ward and in out-patients. However, the majority of acute, confirmed, paralytic polio that we see occurs in the 1- 2-year-old age group, and it appears regularly throughout the year. There was no corresponding "epidemic" among the young children, and we were not seeing paralytic polio among the older age groups at that time. Poliomyelitis would not be a satisfactory explanation for the more serious cases, and encephalitis would not be a satisfactory explanation of the mildest case.

"Injections" are frequently incriminated for precipitating paralysis in the injected limb in polio cases, but this usually occurs within a day or two of injection, and in previously infected subjects. Though prehaps worth mentioning, an explanation along these lines is hardly plausible in view of the time relations of the events, and the total absence of paralysis.

SUMMARY

Four cases of an encephalitis-like illness 3 weeks after BCG vaccination are described, and alternative explanations of the occurrence are discussed. As none of these explanations account for all the features of the illnesses, it is felt that it is worth recording the observation.

ACKNOWLEDGMENTS

I wish to thank Dr. M. M. Nalumango, Permanent Secretary, Ministry of Health, for permission to publish this report and Dr. J. C. Davidson, under whose care the patients were admitted.

CASE REPORTS

HAEMOSIDEROSIS, OSTEOPOROSIS AND SCURVY

M. N. LOWENTHAL, M.B., Ch.B.(Rand.) M.R.C.P.(Edin.)
Physician Specialist
General Hospital, Ndola

J. A. SIDDORN, M.B., Ch.B.(Birm.)
Medical Registrar
Central Hospital Kitwe

R. P. PATEL, M.B., B.CH.(Dublin)
House Physician
Central Hospital, Kitwe

J. FINE, M.D.(Glas.) D.P.H.
Pathologist
Central Hospital Kitwe

Haemosiderosis (also known as siderosis or Bantu siderosis) is known to be a common condition in the Africans of South Africa (Higginson et al. 1953, Wainwright 1957), Rhodesia (Buchanan 1966), Tanzania (Haddock 1965), and Ghana (Edington 1959). This is also true of patients admitted to the Ndola General Hospital and in traumatic cases on whom forensic autopsies are performed at the Ndola Hospital mortuary (Lowenthal, unpublished data). The subject has been reviewed by Bothwell et al. (1965).

The clinical associations of haemosiderosis are portal fibrosis and cirrhosis often leading to liver failure, diabetes mellitus, porphyria cutanea tarda, and scurvy (Bothwell et al. 1965). Pathologically the condition is characterized by the excessive deposition of iron in the form of haemosiderin granules in the reticulo-