LATHYRISM IN THE CENTRAL PROVINCES

Lathyism in the Central Provinces

By Dr. MARTIN LUDLAM, M.B., CH.B. (EDIN.),
Friends Mission Hospital, Itarsi

A small investigation was carried out by two of us near the village centre of Babji in the Hoshangabad District of the C.P., in order to find out what could be learnt of the extent, cause, prevention and cure of the cases of lathyism occurring in that area. The time at our disposal was limited to two days and our means of transport to two bicycles, hence only a very inadequate picture of the true situation can be given. Moreover it should be understood that as our coming was unheralded the cases we saw were only those who happened to be present in their own homes as we passed.

Five villages were visited and a total of about 50 cases of lathyism were seen of whom 43 were more closely examined.

All ages from 5 years upwards and both sexes were affected. 14 of the cases were under 15 year of age but only 4 women were seen with the disease. The poorer section of the community was mostly affected and especially those whose means of livelihood was only indirectly from the land. The cause of the condition was unquestionably associated with the consumption of large quantities of teora dal (a kind of pea) normally used for the feeding of cattle but also consumed in greater or lesser quantities by the villagers depending on the availability and price of better class grains. The disease occurred among those who where paid cash and those who were paid in kind; those who were paid in kind (teora dal) could not avoid the disease, and those who were paid in cash could buy only those grains which were available, namely teora dal. It should be clearly understood that the villagers knew perfectly well the cause of their lameness, though some people (unaffected) and who were possibly interested in the growing of the inculmicated grain either blamed consumption of buttermilk with the dal, or else the consumption of a kind of grass. We found no foundation for such alternative suggestions.

The time at our disposal did not allow us to examine or question those who, though eating large amounts of the dal, yet did not suffer any apparent ill effects. Those who were affected had been eating the dal in small amounts for many years, and in considerable quantities for the past three years. The average duration of lameness was fifteen to eighteen months though some had been affected as long as three years and others as short a time as three months. To quote the villager, they said that they had been eating almost nothing but teora dal "since the dearness period" which of course coincided with the dislocation of transport, and scarcity of grains, etc., brought on by the threat of a Japanese invasion, the August disturbances, and the Bengal famine. Conditions were aggravated by poor jawar and wheat crops, lack of alternative seeds, procurement of grain by the Government mainly for the towns and the indifference of the landowners. Nevertheless, these people were not apparently under-nourished, nor were they suffering from any obvious deficiency diseases, and some of them were remarkable for their powerful build and good muscular development.

The symptoms of the disease were little different from those usually described in the textbooks. The onset was always fairly rapid, the first symptom often being pain in one or other thigh with weakness of one or both legs. Within eight
to ten days the disability became severe and bilateral in distribution, but always confined to the lower limbs. Sometimes the onset came with dramatic suddenness so that the patient collapsed; sometimes patients actually blamed the fall for bringing on the condition.

Examination revealed in all cases the presence of a spastic paresis involving all the muscles of the legs. The patellar reflex was always extremely exaggerated, and patellar clonus sometimes and ankle clonus always could be elicited. The plantar response was extensor. Ordinary sensation (hot and cold not tested) seemed unimpaired. Joint sense in the lower extremities remained good and even in a most severe case vibration sense was not impaired. This severe case who could just walk and no more, had incontinence of both urine and faeces. The abdominal reflexes were always impaired in proportion to the severity of the condition. In the cases examined there was no obvious disability or abnormality of the reflexes in the upper extremities. At this time we saw no cases who were actually bedridden; most could move fairly quickly with the aid of one stick, while the worst cases could only make slow laborious progress with the aid of two sticks. The attitude of most was cheerful and perhaps rather fatalistic.

Summary and Comments. The investigations revealed that the consumption of large quantities of toora dal daily over a long period (more than 3-6 months) causes in most cases a spastic paresis of the lower extremities. The consumption of the dal in small quantities (1/3 of the total grain consumed) over periods does no harm.

The onset of the condition is rapid and we fear, irreversable. The signs indicate that the posterior columns of the spinal cord are not affected but that the disease is mostly confined to the pyramidal tracts. It does not seem that the disease is primarily due to any vitamin deficiency but that the lesion is produced by a toxic principle in the diet.

The poison may be cumulative in action, being analogous to digitalis in this respect. Because of the rapid onset and irreversability of the condition it may be that the precipitating cause of the lesion spinal is vascular in nature. The fact that women seem much less liable to the disease may be due to the fact that the female nervous system has for some reason a greater resistance to the toxin. One woman seen, stated that she had actually recovered from the disease but this case was quite unique; on examination there was no apparent lameness but the reflexes were markedly exaggerated.

The preventability of the disease, the poor prognosis, and the otherwise excellent condition of the patients makes the disease especially tragic. Unfortunately patients rarely stay in hospital long enough for one to form a proper estimate of the best kind of treatment. In this connection we should add that we have been treating two patients in our hospital for the past 4 weeks. Each of them has been lame for about 15 months. The treatment has been the provision of an Indian diet (a not very good diet) avoiding toora dal, and the administration of yeast, ascorbic acid, shark liver oil daily, thus providing maintenance doses of the main essential vitamins. One of the patients was severely anaemic (Hb 50%) so in addition this patient received iron and liver injections in full doses. The first patient has shown little or no improvement; but the anaemic patient who at first was almost bedridden, improved rapidly, and can now walk short distances at a reasonably fast rate with the aid of one stick. We cannot say however that this is due to regression of the
spinal lesion.

In conclusion we should like to point out that this lameness although certainly associated with the consumption of tea daal, or more correctly lathyrus sativus may not be actually due to this plant, as is suggested in some textbooks (we only have textbooks for reference). At the same time we understand that the nutrition experts in this country are not of the opinion that this condition is directly due to the consumption of this grain, and not due to the consumption of an allied but contaminating grain. Certainly no one who has seen tea daal as sold in the bazaar or growing in the fields would think there could be any other grain accidentally associated with it.

Reprinted from the I.M.J. Vol. 40, No. 4, April 1946

Midwives Union Page.

Ruptured Uterus

Most Midwifery Text Books teach us that Patients who manage to rupture their Uterus during the course of Labour, invariably die from shock. Here however is the Case history of an Indian mother who not only ruptured her Uterus, but managed to survive the ordeal. Aged 32. Para 7. All previous normal deliveries, never on any occasion Ante Natal Care.

Friday Oct. 18th. Labour pains began during the night, about 10 p.m.
Saturday Oct 19th. 4.30 a.m. Pains suddenly stopped and there was some vaginal bleeding which later ceased.
9.40 a.m. Relatives got a jutka and brought her 9 miles to Hospital. arrived 12.30 m.d.
12.30 m.d. Admitted to Hospital, condition very shocked, skin cold and clammy, pulse rapid and thready, rate 140. Respiration 28 Temperature 98.4. B.P. 110:90 H.B. 35%. on palpitation, foetal parts easily felt per abdomen. No Foetal Heart.

Diagnosis. Ruptured Uterus

Treatment. Coramine 1. c.c. by hypo inj. Subcutaneous Saline 2 Pints with 50 c.c. 25%. Glucose given. Shock treated with warm blanket and hot water bottles.
5.30 p.m. Shocked condition a little better. Prepared for Theatre.
6.10 p.m. Morphia gr. ½, with Atropin gr. 10. lh, given by hypo inj. 6.30 p.m. Taken to Theatre, head of the table lowered a little. Rectal drip Saline with Glucose fixed up and given throughout the Operation. Pulse remained rapid and at times almost imperceptible. I.e. Coramine by hypo inj given again. Although general condition was very poor, a Laparotomy was thought advisable without delay.
6.45 p.m. General Anaesthetic was administered, Chloroform, throughout the Operation. The Laparotomy revealed an Abdomen full of blood clot. When this was removed the Amniotic sac was seen intact in the abdominal cavity. Membranes were ruptured, and the Foetus, stillborn, full term was extracted. Placenta was removed and Pituitrin ½ c.c. was given into the Uterus. A large tear of the posterior surface of the Uterus was revealed, although this did not extend into the Broad Ligament. The rupture of the Uterus was sutured, as her general condition would not stand Hysterectomy, and the incision was closed in 3 layers.

7.30 p.m. On return to the Ward her condition was fair, Pulse thready and still