Original Articles.

AN OUTBREAK OF TRUE BERI-BERI IN AN ASSAM JAIL.

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THE climate and physical configuration of Assam resemble so closely those of Burma and the political connection between them on several occasions before our occupation of the Assam Valley was from time to time so close that it is matter for some surprise that a disease prevailing so extensively, as does beri-beri in Burma, does not appear to have been introduced into Assam. Any such introduction, if it ever occurred, has been on so small a scale or the circumstances were so unfavourable to its spread that it has escaped recognition. Moreover, the opening out of tea gardens and the consequent importation of a numerous foreign labour force introduced an unfortunate confusion of nomenclature. These new and unnatural social conditions brought into prominent notice a heterogeneous group of diseases all characterised by anæmia, which prevailed extensively amongst the new and unacclimatised population living as they were in unaccustomed surroundings and under, to them, irksome conditions of discipline and labour. This group of diseases, of which anchylostomiasis and malarial cachexia are the most important, became known by the name of "beri-beri," following the example of Ceylon, where similar social conditions had, one may suppose, produced similar results. Hence for many years "beri-beri," or its synonym "anæmia of coolies," occupied an important place in the vital statistics of the Assam Valley, and obscured the older use of the name "beri-beri" to express an endemic form of multiple neuritis. A record in Assam of "beri-beri" unqualified by any explanatory synonym is therefore more likely to refer to some form of anæmia than to a case of endemic multiple neuritis. Although there may have been cases of true beri-beri reported, I do not know of a record of any local outbreak. Therefore a short note on a small outbreak which occurred in the Gauhati Jail between August and October 1902 may not be devoid of interest.

The first case was an elderly man admitted to hospital on August 13th, who died on the 17th, with sign of cardiac dilatation—venous engorgement, practical suppression of urine and pulmonary œdema. On *post-mortem* examination the heart and kidneys appeared healthy, and no cause for the cardiac dilatation could be found save a very slight degree of pulmonary emphysema affecting only the anterior borders of the lungs, apparently much too slight to have caused such rapidly fatal results. The urine at a time when only two or three ounces were passed in the twenty-four hours was, curiously enough, free from albumen.

This example of what Schenbe describes as the acute cardiac or pernicious form of beri-beri remained a puzzle till cleared up by the recognition of the second case which occurred in a young Garo. He was being treated for dysentery at the time the first case died, and a few days sufficed to cure the dysentery. But by that time he was observed to be suffering from slight anasarca and scanty urine. By the 2nd September there was great dilatation of the heart, and the anasarca was especially marked at the root of the neck to such a degree indeed as to obstruct deglutition. No knee-jerks could be obtained. The urine was nearly suppressed. Death occurred on the 8th September. The *post-mortem* showed great general subcutaneous œdema fluid in all the serious sacs, with cardiac dilatation and a few small ulcers in the rectum. The abdominal organs were all engorged with blood and the lungs œdematous.

The third case was in some ways the most typical. It was more chronic and showed successively the œdematous and atrophic type. He complained on admission (1st September) of slight fever (to 101°). The feet were œdematous and the urine diminished to 12 oz. in the 24 hours. From September 6th to 17th improvement occurred, the urine averaged 32 oz. in the 24 hours, but no knee-jerks were obtained. On the 19th a relapse occurred, the urine again diminishing, and the œdema increasing with a slight rise of 60 to 99. Marked epigastric pulsation appeared, and for the first time sensation was found to be deficient on the dorsum of the right foot and front of the right leg. The pain of pin prick was delayed, and in places the head could not be distinguished from the point of a pin. On the left foot and leg there was a less degree of delay and blunting of sensation. The legs were crossed only with difficulty. From the 19th to 24th September the urine averaged only 10 oz. in the 24 hours and on one occasion fell to 5 oz. The evening temperature range was between 99° and 100°. On the 25th there was marked tenderness of the muscles of the legs and forearms and the cardiac dulness was increased in all directions. A basal systolic murmur appeared. No hydro-thorax and no ascites could be detected. On October 1st the upper half of the body was much more œdematous than the lower and ascites appeared. From the 9th October the urine began to increase and no rise of temperature occurred; muscular tenderness diminished. But as the œdema subsided marked muscular wasting of the forearm and legs became evident. Recovery was very gradual. Six months later the muscles were still wasted and the patellar tendon tap threw the vastus internus into chronic spasm without producing a normal response.

The fourth case admitted on September 12th with slight anasarca improved so much that within a few days he was unfortunately discharged from hospital. His knee-jerks were present. On October 18th he was readmitted. No knee-jerks were obtained and sensation was absent over a considerable portion of the front of the legs and dorsal surface of the hands and forearms. Later patches of anæsthesia appeared even on the soles of the feet. Eventually præcordial pain and dilatation of the heart with a mitral systolic murmur developed with great diminution of urine. Death occurred on November 27th. The *post-mortem* appearances were similar to those of Case (2).

The fifth case was admitted with slight œdema of the feet and over the sacrum and absent knee-jerks, but the most marked symptoms were weakness of the legs, a peculiar gait and slight Romberg's symptom. There was doubtful blunting of sensation of shins. After being under observation for two months and-a-half he was released on expiry of sentence. The gait was much improved, but the other symptoms persisted.

The sixth case was admitted on September 23rd with increased knee-jerks, which, in the course of three days, disappeared. The legs were weak and could not be crossed without assistance. Five days later the heart became dilated and death occurred on October 1st.

The seventh case also admitted on September 23rd with absent knee-jerk and œdema. He also died of cardiac dilatation with great dyspnœa and obstinate vomiting on October 25th.

The eighth case, admitted on 2nd October with slight œdema, some cardiac dilatation and absent knee-jerks, was only under observation for eight days before his sentence expired.

The ninth case was one of the acute cardiac variety like the first and died within three days.

The tenth case was admitted on 28th October complaining of loss of strength in lower limbs and of sudden contractions of muscles of the thighs. On examination the knee-jerks were brisk. There was slight anæsthesia over patches of the inner aspect of both calves. On attempting to rise from bed a sudden contraction was produced in the hamstring muscles, which threw him down. There was slight pretibial œdema. On November 3rd the anæsthesia of the pretibial and inner aspect of the legs was complete—there was also slight anæsthesia of forearms and hands (extensor surface). On November 15th the knee-jerks had disappeared; there was some tenderness of the calf muscles. The area and degree of anæsthesia had diminished. On December 4th there was slight ascites and œdema of the abdominal walls. On the 6th the urine (not previously much reduced in amount) suddenly rose to 60—70 oz. in the 24 hours and concurrently the œdema and ascites disappeared. On the 11th he was released on expiry of sentence.

With regard to individual symptoms, one or two points are noteworthy—

(1) The absence of any trace of albuminuria, even when the daily secretion fell to a few ounces or even to the point of practical suppression, was noted in cases 1, 2, 3, 4 and 6.

(2) An early and fleeting accentuation of the knee-jerk was noted in cases 4, 6, and 10, in which the reflex disappeared under observation.

(3) Extremely sudden fluctuation in the extent and degree of anæsthesia was noted in cases 4 and 10. In the former, between October 18th, when loss of sensation was first detected, to the 28th, when the maximum was reached, the anæsthesia had spread from the dorsum of the left foot and front of the left leg successively to the right foot and leg, to the dorsum of the left hand and portions of the forearm, the two ulnar interdigital spaces of the right hand, the fourth left interdigital space and ulnar border of the little finger, the whole extent or surface of the left forearm, the dorsal surface of the outer two toes of the left foot and small portions of the sole. The greater part of this extensive anæsthesia cleared up in about two days, in spite of an increase in the severity of the cardiac symptoms, and on the last day of life anæsthesia was practically confined to the pretibial areas.

In case 10 the anæsthesia never reached the same pitch, either in area or intensity, but, after persisting for a couple of weeks in both legs and forearms, disappeared in the course of a comparatively few days, so that on release only the left calf shewed patches of anæsthesia.

The investigation of sensation was confined to testing the ability to distinguish the head from the point of a pin and the acuity of the perception of the pain of a pin-prick. Both these cases showed for a few days patches of absolute analgesia. It is interesting to note that in case 10 the sensory loss occurred before, and reached its maximum just when, the knee-jerks disappeared. This case also displayed the curious phenomenon of spasm of the hamstring muscles, which I have not seen described in this disease.

(4) Pyrexia preceded the onset of symptoms of beri-beri in case 6, who, from 1st to 8th September, was in hospital with irregular fever, which was supposed to be malarial, but did not yield to quinine. Case 3 also complained first of fever, but in his case œdema was present from the time he first came under observation in September 1st. It may, however, be remarked that in this case a second bout of fever on 19th September ushered in an aggravation of the other symptoms. Both the fever and the aggravation of the heart symptoms lasted till October 6th, from which date the temperature fell and the other symptoms also began to improve.

(5) As regards the œdema—this shewed a preference for the root of the neck and the angle of the jaw in cases 2 and 3, for the presternal region for some time in case 4, and for

the dorsum of the hands in case 10, and for a while the abdominal cavity and abdominal wall. In all these, however, there was more or less dropsy of the feet, in the erect, and of the back and sacrum, in the recumbent, position. In all the *post-mortems* marked pericardial effusion was formed, usually with fluid in one or more of the other serous cavities.

It is worth mentioning that Case 2 suffered in the month of May from a painless chronic non-inflammatory effusion into the right knee-joint, which lasted about a month and did not re-act to any treatment. In June a precisely similar condition affected the left knee. No cause was discovered to account for the effusion, and it is possible that it may have been of atrophoneurotic character. It preceded definite symptoms of beri-beri, however, by nearly two months.

In none of the cases did anæmia play any part, except perhaps in case 2, in whom also a few ankylostoma were found *post-mortem*; and in only one case did the spleen show signs of old malarial infection.

Of the *post-mortem* signs, dilatation of the heart, especially of the right side, pericardial effusion and œdema of the lungs were constant. The blood was in all the fatal cases markedly fluid. In two cases (2 and 6) there were a few small ulcers in the large intestine.

Six deaths out of these ten cases is a heavy mortality. Especially as of the remainder only one was known to have recovered—the other three being released with symptoms still persisting. No doubt other mild cases escaped recognition—two indeed were under observation, whose symptoms though suspicious were not marked enough to allow of their being reported as beri-beri. But with all allowances, the fatality is greater than seems usual in large series of cases.

As regards the existing cause of the outbreak nothing could be proved. The shortest period of confinement in the jail, which was followed by the development of the disease, was two months in case 8. Case 9 had been in this jail for three months, all the others for over six months, up to as long as nine years in one case.

FURTHER NOTES ON THE CULTIVATION OF THE BACILLUS LEPRÆ AND THE TREATMENT OF LEPROSY BY THE INJECTIONS OF A LEPROLIN MANUFACTURED FROM CULTURES.

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(*Continued from p.* 169.)

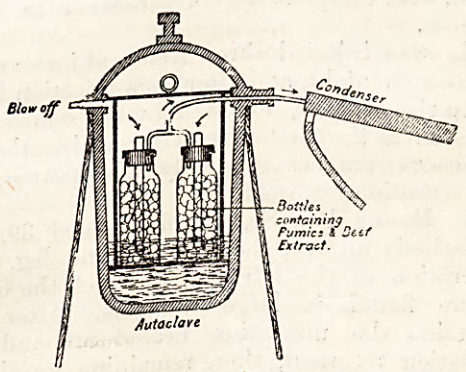
THE method of preparing the distilled nutrient medium has been improved, thus rendering the cultivation of the bacillus lepræ in this medium much more potent for the preparation of leprolin.

Small pieces of pumice stone are washed and dried in the sun, and then made to absorb beef extract; they are then placed in bottles having a wide central tube leading to the bottom, and an outlet tube at the top.

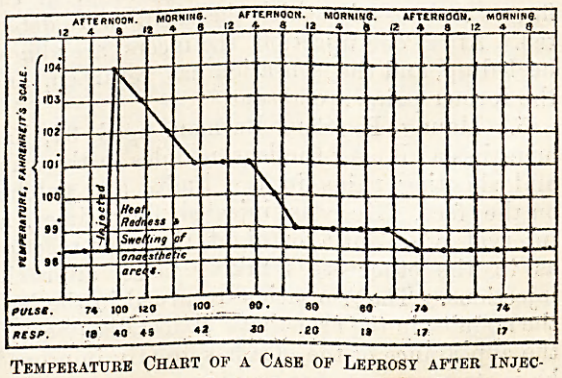
The bottles are placed inside the autoclave, and the tubes from the top of the bottles led out from the autoclave through the valve or blow-off openings, and on to the condenser. By this means the superheated steam passes through the bottles and acting on the heated beef extract in the pumice, carries over to the condenser more nutrient value out of the beef extract, all salts and non-volatile substances being left behind in the bottles. The nutrient fluid is collected at the end of the condenser in sterilized Pasteur flasks and inoculated, when cool, from cultures of leprosy.

The flasks are then allowed to incubate at 37 C. for a period of one month to six weeks and examined, when they should present a turbid appearance and a stringy white deposit, which on being shaken up forms stringy, white, curly, heavy cloudiness in the flasks.

They are then passed through a sterilized Pasteur filter and again re-filtered through fresh sterilized filters, and then reduced to about one tenth the original bulk, by exhaustion over sulphuric acid in a vacuum; the fluid is then mixed with an equal quantity of glycerine and kept in an ice box until required for use.



SECTION THROUGH AUTOCLAVE SHOWING ARRANGEMENT FOR MANUFACTURE OF NUTRIENT MEDIUM FOR GROWING BACILLUS LEPRÆ.



TEMPERATURE CHART OF A CASE OF LEPROSY AFTER INJECTION OF 20c.c. OF LEPROLIN IN THE RIGHT BUTTOCK.