residual chlorine reading of 0.4 p.p.m. At the same time the ratio of chlorine to ammonia was increased from two to four parts of chlorine to one of ammonia with the object of accelerating the process of sterilization. After this step had been taken a few cases of dysentery continued to occur, but seldom was it impossible to find a history of contact with a previous case.

Discussion

The character of the epidemic suggested some general source of infection such as a common food or water. The fortunate circumstances under which *B. dysenteriae* Sonne was isolated from a sample of water precluded the possibility of a sampling error having been made. The disturbing feature was the fact that the sample of water satisfied the usual standards for purity, and that 0.15 p.p.m. residual chlorine was still present. This appeared to be a direct contradiction until it was found experimentally that *B. dysenteriae* Sonne could survive after treatment with this strength of chloramine for periods longer than those applicable to many of the consumers. The position was rendered more dangerous by virtue of the fact that the chlorine-ammonia ratio of 2:1 was such as to delay considerably the sterilization process, as shown by Elliott (1933). Notwithstanding the satisfactory results of the single tests performed on the raw well-waters and that subsequent tests of the treated water were invariably negative, the fact remained that cess-pits were in existence in the drainage area and the water therefore remained suspect.

The apparent connexion observed at the onset of the epidemic between one particular milk supply and a group of cases was interesting in the light of the finding of *B. dysenteriae* Sonne in the water supply. The dairyman concerned was in the habit of rinsing his bottles with cold tap-water after they had been sterilized by steam, and probably introduced the infection to his supply in that manner.

Flies were considered as possible vectors which may have produced an explosive epidemic, particularly as the town sewage is discharged untreated into the sea at no great distance from the town. At the time of the present epidemic, however, there were remarkably few flies in evidence—a fact commented upon by many observers. When the epidemic had practically subsided the usual summer increase in the fly population developed without stimulating further infection. This fact appeared to minimize the importance of flies in this particular epidemic, for with the persistence of the carrier state (E.P.H.L.S., Nov., 1942; Hobbs and Allison, 1942) ample opportunity must have occurred for the transference of infection from faeces to town during the period of heavy fly infestation.

Summary

An outbreak of Sonne dysentery involving approximately 400 cases is described.

Milk was excluded as a general source of infection, although probably indirectly responsible for one small group of cases.

B. dysenteriae Sonne was isolated from one sample of tap-water which passed the usual bacteriological tests satisfactorily and which contained 0.15 p.p.m. residual chlorine.

The evidence for incriminating the water supply as the source of infection is discussed.

We wish to acknowledge with thanks the willing co-operation of the director and staff of the water company, the milk retailers, and the blood donors during the investigation.

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According to a law introduced in April, 1942, everyone in Norway who has reached the age of 15 years must have his lungs examined by screen photography when the authorities demand it. Persons exempt from this obligation are those who have been x-rayed at school or who are notified cases of pulmonary tuberculosis and have been x-rayed within twelve months of the time they reach the age of 15. When the examination has been completed, information to this effect is stamped on the person's identity card (*Tidskrift for Norges Laegeforbund*, July 1, 1942, No. 13).

INFECTIVE HEPATITIS TREATED WITH GLUCOSE, INSULIN, AND ASCORBIC ACID

BY

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Infective hepatitis (syn., infective jaundice, epidemic catarrhal jaundice) is an endemic disease in Omdurman. During 1942 50 cases were admitted to Omdurman Civil Hospital, a native hospital of 215 beds. Of these cases 42 fell in the age group 10 to 33 years. The duration of the disease was 2 to 7 weeks (average 23 days). From time to time minor epidemics of the disease have occurred, and these have often included one or two severe cases resembling acute liver atrophy which have ended in death from profuse bowel haemorrhage. Various forms of treatment have been tried, including massive doses of glucose, intravenous drip infusions of glucose and alkali, intravenous sodium thiosulphate, and repeated medical drainage of the gall-bladder. None of these measures has appeared to have had the slightest effect upon the course of the disease. Recently El Kaimakam H. J. Bell (then venereologist to the Sudan Defence Force) read a paper to the Sudan Branch of the British Medical Association on "Arsenical Toxaemia." In his paper Major Bell described some very striking results which he had obtained by the use of glucose, insulin, and ascorbic acid in the treatment of late toxic conditions resulting from the treatment of syphilis by neoarsphenamine. These cases included arsenical jaundice. Major Bell's paper suggested a possible new line of treatment for infective hepatitis, particularly as there is a good deal of evidence to suggest that arsenical jaundice may be due to the synergic action of neoarsphenamine and the causal agent of infective hepatitis, probably a virus. Accordingly five consecutive cases of infective hepatitis admitted to Omdurman Civil Hospital were treated on these lines, and a summary of the case records is given below.

Case I

A Sudanese male aged 26 was admitted on Feb. 16, 1943, with a complaint of jaundice for 4 days, accompanied by nausea, flatulent dyspepsia, and constipation. There was no history of syphilis or of treatment by arsenicals.

Condition on Admission.—Afebrile; moderately severe jaundice; tongue coated; liver dullness normal; spleen not palpable. Urine: bilirubin and bile salts present; no albumin. Stools clay-coloured. Blood: no malarial parasites. White cell count, 8,200 per c.mm.—polymorphs 53%, lymphocytes 44%, large mononuclears 1%, eosinophils 2%. Capillary resistance test normal.

Progress and Treatment.—Feb. 17: Condition unchanged. Treatment started: glucose by mouth, 5 oz. daily; insulin, 10 units twice daily; tab. ascorbic acid (25 mg.), three tablets thrice daily. Temperature rose to 99.2° in the evening. Feb. 18: Afebrile; trace of jaundice. Feb. 20: Afebrile; no jaundice; no symptoms.

Case II

Sudanese male aged 38, admitted Feb. 7, 1943. Severe lumbar pain for 5 days and jaundice for 3 days with flatulent dyspepsia, nausea, and constipation. He had no history of syphilis or of treatment by arsenicals.

Condition on Admission.—Afebrile; very deeply jaundiced; tongue coated; liver dullness slightly diminished; spleen not palpable. Bilirubin and bile salts present in urine, with a trace of albumin. Stools clay-coloured. No malarial parasites in blood. White cell count, 9,800 per c.mm.—polymorphs 51%, lymphocytes 45%, eosinophils 4%. Large mononuclears were absent. Capillary resistance test normal.

Progress and Treatment.—Afebrile throughout. Condition remained unchanged until treatment started. Feb. 16: Treatment with glucose by mouth, 5 oz. daily; insulin, 10 units twice daily; tab. ascorbic acid (25 mg.), three tablets thrice daily. Feb. 18: Jaundice greatly decreased. Feb. 20: A faint icteric tinge only could be detected on the conjunctivae. Feb. 22: Jaundice gone; no symptoms.

Case III

Sudanese female aged 22, admitted Feb. 14, 1943, complaining of jaundice, dyspepsia and continuous nausea for 7 days, and constipation. There was no history of syphilis or of treatment by arsenicals.

Condition on Admission.—Afebrile; moderately severe jaundice; tongue coated; liver dullness normal; spleen not palpable. Bilirubin and bile salts present in urine, and a trace of albumin. Stools clay-coloured. No malarial parasites in blood. White cell count, 8,500 per c.mm.—polymorphs 55%, lymphocytes 40%, large mononuclears 3%, eosinophils 2%. Capillary resistance test normal.

Progress and Treatment.—Temperature rose to 99.2° on evening of Feb. 15 and to 99.8° on evening of the 16th; otherwise afebrile. Feb. 16: condition unchanged. Treatment started: glucose, 5 oz. by mouth daily; insulin, 10 units twice daily; tab. ascorbic acid (25 mg.), three tablets thrice daily. Feb. 17: Trace of jaundice. Feb. 19: Jaundice nil; no symptoms.

Case IV

Sudanese male aged 9 years, admitted Feb. 15, 1943, having had jaundice for 7 days, with constipation. There was no history of treatment by arsenicals.

Condition on Admission.—Afebrile; moderately severe jaundice; no signs of congenital syphilis; tongue coated; liver dullness normal; spleen not palpable. Bilirubin and bile salts present in urine; no albumin. Stools clay-coloured. No malarial parasites present in blood. White cell count, 5,700 per c.mm.—polymorphs 39%, lymphocytes 56%, large mononuclears 3%, eosinophils 2%. Capillary resistance test normal.

Progress and Treatment.—Afebrile throughout. Feb. 16: Condition unchanged. Treatment started: glucose by mouth, 2 oz. daily; insulin, 5 units twice daily; tab. ascorbic acid (25 mg.), one tablet thrice daily. Feb. 18: Faint icteric tinge only to be seen on the conjunctivae. Feb. 19: No jaundice; no symptoms.

Case V

Sudanese female aged 70, admitted Feb. 17, 1943, complaining of jaundice for 30 days accompanied by flatulence and nausea. There was no history of syphilis or of treatment by arsenicals.

Condition on Admission.—Afebrile; emaciated; intense jaundice; liver dullness normal; gall-bladder and spleen not palpable; nothing abnormal discovered clinically elsewhere. Bilirubin and bile salts present in urine; no albumin. Stools clay-coloured. No malarial parasites in blood. White cell count, 4,800 per c.mm.—polymorphs 61%, lymphocytes 34%, large mononuclears 4%, eosinophils 1%. Capillary resistance test normal.

Progress and Treatment.—Afebrile throughout. Feb. 18: Treatment started: glucose, 5 oz. by mouth daily; insulin, 10 units twice daily; tab. ascorbic acid (25 mg.), three tablets thrice daily. Feb. 21: Condition unchanged.

Note.—In view of the patient's age, the intense degree of jaundice without haemorrhages and without diminution of liver dullness, and the emaciation, this may well be a case of carcinoma of the head of the pancreas.

Summary

The treatment is described of five cases of infective hepatitis, taken at random, by glucose, insulin, and ascorbic acid. In Cases I, III, and IV there was complete remission of all symptoms and signs in 3 to 4 days. In Case II, a severe one, there was marked improvement in 4 days. In Case I the disease lasted only 9 days (the lowest time recorded in Omdurman Civil Hospital); in Case III it lasted 14 days; in Case IV it lasted 12 days. All these times are well below the average of 23 days. In Case V, in which, however, for the reasons stated above, there was cause to doubt the accuracy of the diagnosis, there was no change.

Although it is appreciated that it would be rash to base conclusions on the results of a series of only five cases, nevertheless, in view of the importance of this disease and in view of the striking results obtained in three of the cases, it has been considered desirable to place these notes on record without delay.

I wish to acknowledge my indebtedness to the Director, Sudan Medical Service, for permission to publish these case records; to Major H. J. Bell for his valuable paper on arsenical toxæmia; and to Dr. Mahmoud Hussein for his assistance in compiling the records.

LIVER DEFICIENCY ANAEMIA IN A CASE OF ACUTE INFECTIVE HEPATITIS

BY

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AND

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The following is an account of a case of liver deficiency anaemia which occurred acutely during the course of an infective jaundice. There was slight anisocytosis and practically no poikilocytosis, and the anaemia responded with a clear-cut reticulocytosis to injections of liver, the only haematinic employed. There was no associated achlorhydria or even hypochlorhydria, nor were there any nervous symptoms. The condition would appear to have been due solely to the effects of liver damage, as there had been no evidence of gastric disturbance since admission and the anaemia developed while the patient was in hospital.

Case History

The patient is a gamekeeper on a moorland estate in Yorkshire, and was known to one of us (J. N. H.) before he developed jaundice. He was a robust man of 52 years who had never had any previous illnesses, who did not smoke, and who was practically teetotal. His son, aged 18, had had jaundice for two weeks—four weeks before the patient contracted it—and the patient himself had had it for five weeks before he was sent to the County Hospital, Otley. He had continued to work almost up to the day of admission, Oct. 23, 1942. The incubation period of four weeks is mentioned by Dr. W. N. Pickles (1942) in his Milroy lectures. He also remarks that the disease frequently visits Wensleydale, and we have noticed quite a crop of cases in Wharfedale during the past two years.

By the time the patient was admitted he had lost a considerable amount of weight, largely owing to anorexia, which had caused him to take practically nothing to eat during the two previous weeks. He was profoundly jaundiced, and suffered from intense itching of the skin due to this. His stools were clay-coloured, and his urine was loaded with bile pigments and contained a small quantity of urobilinogen. His liver was grossly enlarged, extending down to the umbilicus, and its surface smooth; his spleen protruded four fingerbreadths below the left costal margin on inspiration and felt very hard. Examination revealed no other noteworthy physical findings, and the nervous system was normal.

A blood count on admission gave the following results: R.B.C., 4,800,000; Hb, 98% Sahli (throughout); C.I., 1; W.B.C., 3,300—polymorphs 58%, lymphocytes 33%, monocytes 5%, eosinophils 2%, ribbon forms 2%. The polymorphs showed toxic granulations. The van den Bergh test gave a direct positive result. The Wassermann test was negative.

The patient was put on a carbohydrate diet with copious glucose drinks, and vitamins in the form of Abbott's pentacaps 2 t.d.s. for the first three weeks. By that time his appetite had completely returned, and he then took a normal diet with added carbohydrate drinks. The leucopenia was checked again on Oct. 29 and Nov. 4 and 12, and showed totals and differentials very similar to the original count except that the percentage of ribbon forms rose to 12.

Towards the latter end of this period we had noted with concern that, although the patient was improving generally and eating well, a greenish-grey pallor was showing through the jaundice, so on Nov. 20 we did another complete blood count, and were astonished to find: R.B.C., 2,300,000; Hb, 63%; and C.I., 1.37. The film revealed slight anisocytosis, with macrocytosis, hyperchromia, polychromasia, and a few nucleated red cells. The white cell count—3,200—and the differential count showed no significant change from those noted above. The reticulocytes appeared to be approximately normal, although an actual count was not made on this specimen.

A sternal puncture was done immediately, and after examination the smears were sent to Edinburgh. Prof. L. S. P. Davidson and