The following are the results of further investigations. Feb. 19:—Blood count: red cells, 4,380,000 per c.mm.; haemoglobin, 87%; white cells 5,200 per c.mm. Agglutinations: negative in dilutions of 1:25 and 1:50 against *Br. abortus*; positive in dilutions of 1:125, 1:250, and 1:1,250. April 16:—Blood count: haemoglobin, 95%; red cells, 4,680,000 per c.mm.; white cells, 6,600 per c.mm. (polymorphs 68%, lymphocytes 30%, monocytes 1%, basophils 1%). Agglutinations: negative in dilutions of 1:25, 1:50, 1:250, and 1:1,250 against *Br. abortus*; positive in dilution of 1:125.

Case 2

This patient, a woman aged 38, first complained of feeling ill on Jan. 24, 1948. She had shivering, sweating, headache, and pain in the right ear. On examination she was found to have an evening temperature swinging to 102° F. (38.9° C.), but no other abnormality was found. On Feb. 8 a blood count showed: haemoglobin, 84%; red cells, 4,690,000 per c.mm.; white cells, 4,000 per c.mm. (polymorphs 46%, lymphocytes 49%, monocytes 4%, basophils 1%). Agglutinations: positive in all dilutions up to 1:500 against *Br. abortus*.

The following chemotherapy was given: sulphamezathine, 48 g. (Feb. 2 to 5 and 9 to 11), penicillin, 1,200,000 units (Feb. 9 to 11), and sulphadiazine, 51 g. (Feb. 27 to March 3). Despite this treatment the patient's condition deteriorated; she had distressing rigors with profuse sweating and pyrexia rising to 105° F. (40.6° C.).

On March 4 she was admitted to the Staffordshire General Infirmary. She was still seriously ill, the temperature rising to 104° F. (40° C.), and the spleen was slightly enlarged. Treatment with sulphadiazine and blood transfusions was begun on March 5, as follows: sulphadiazine, 30 g. (March 5 to 9) and 6 g. (March 13); blood transfusions, 10 oz. (280 ml.) (March 5), 15 oz. (425 ml.) (March 8), and 4 oz. (110 ml.) (March 13). The patient felt much better after the first transfusion and there was an immediate lessening of the severe rigors and sweating. She was discharged from hospital afebrile on March 18.

She felt well and remained afebrile until April 6, when she developed pain under the right costal margin and a temperature of 102° F. (38.9° C.): a diagnosis of acute cholecystitis was made and she was readmitted to hospital on the following day. The condition showed no signs of settling despite 25 g. of sulphadiazine over a period of four days, and finally a cholecystectomy was performed by Mr. B. R. Sworn on April 19. The gall-bladder was acutely inflamed, and a pure culture of *Br. abortus* was obtained from the contents.

There was some increase in the pyrexia and malaise after the operation, so it was decided to try further treatment with sulphonamides and blood transfusions. Sulphadiazine, 12 g. (April 22 and 23) and 6 g. (April 27), and blood transfusions, 8 oz. (225 ml.) (April 23) and 6 oz. (170 ml.) (April 27), were given. There was a mild reaction to the second transfusion, a rigor, and transient rise of temperature, but immediately afterwards the pyrexia subsided completely and the patient said that she felt better than she had been throughout the whole illness. She was discharged from hospital on May 5 and has since remained well.

Comment

Both these cases of abortus fever had failed to respond to ordinary chemotherapy: Case 1 did not improve with penicillin and Case 2 was not affected by penicillin, sulphamezathine, or sulphadiazine. In each case there was rapid response to sulphadiazine and small blood transfusions. Case 2 returned three weeks later with acute cholecystitis due to *Br. abortus*; the temperature did not subside following cholecystectomy but responded to further sulphadiazine and blood transfusions.

While it is impossible to draw conclusions from only two cases, we feel that these results are encouraging and that this method of treatment should be given a more extended trial.

Our thanks are due to Dr. D. B. W. McKendrick, who called our attention to this method of treatment of abortus fever.

Medical Memoranda

Pneumococcal Meningitis in an Infant of 8 days: Recovery

The value of intrathecal penicillin in pneumococcal meningitis is now established, but the optimum dosage still seems to be a matter for controversy. Shalom (1945) reported eight cases of pneumococcal meningitis with three deaths, the patients having been given intrathecally 4,000 units of penicillin daily, together with penicillin intramuscularly and full doses of sulphadiazine orally. As purer preparations of penicillin have become available larger doses have been used. Smith, Duthie, and Cairns (1946) reported 18 cases (with only one death, due to fat embolism) treated with varying amounts of intrathecal penicillin, but they never gave more than 20,000 units in one injection, though 16,000 units 12-hourly were given in a few cases. They state that injections of 40,000 units or over may cause fits or gumming of the subarachnoid space. At that time they were using a preparation of penicillin containing 500 to 700 units per mg.

The following case is reported because of the extreme youth of the patient and because of the very large doses of penicillin which were given intrathecally without any ill effect.

CASE HISTORY

A male child aged 5 days was admitted on Jan. 16, 1947, with his mother, who had lobar pneumonia, for breast-feeding. He was fully examined on admission. The cord had separated and the stump was dry and clean. He was slightly jaundiced and apparently had been so since he was 2 days old. His heart and chest were normal and his spleen was not palpable. There was no neck rigidity and his mouth and throat were healthy. He weighed 6 lb. (2.7 kg.). According to the mother his weight at birth was 7 lb. 4 oz. (3.3 kg.).

In spite of adequate amounts of milk for a child of his size and age he had lost weight. On Jan. 19 he passed one green relaxed stool and his temperature was 100° F. (37.8° C.). No physical signs were found, and his jaundice had faded. On Jan. 20 the temperature was 102° F. (38.9° C.). He was not taking his feeds well, and there was slight neck rigidity, but no other signs. A lumbar puncture was performed, and a turbid yellowish cerebrospinal fluid was obtained. Penicillin, 30,000 units, was injected intrathecally. The report on the cerebrospinal fluid was as follows: Opalescent fluid; lymphocytes, 6 per c.mm.; polymorphs, 82 per c.mm.; total protein, insufficient fluid; globulin, ++; chlorides, 700 mg. per 100 ml.; film, Gram-positive diplococci ++++; culture, profuse growth of pneumococci, penicillin-sensitive.

He was also given 0.5 g. of sulphadiazine, followed by 0.25 g. four-hourly for seven days, then 0.125 g. six-hourly for seven days (total 14.6 g.); 30,000 units of penicillin daily intrathecally for eight days (total 240,000 units); 30,000 units of penicillin intramuscularly three-hourly for 16 days (total 2,880,000 units); and subcutaneous salines to combat dehydration when he was not taking feeds well.

After three days there was some clinical improvement—less neck rigidity—and he took his feeds better. At the end of eight days the pyrexia had almost settled and the cerebrospinal fluid was nearly normal. Intrathecal penicillin was discontinued. Further recovery was uneventful. Owing partly to the fact that she was ill herself, the mother's milk dwindled in amount, and the baby was gradually weaned on to a proprietary milk preparation. He was discharged on Feb. 7 when fit and gaining well, his weight being 7 lb. 2½ oz. (3.25 kg.).

DISCUSSION

It must be unusual for so young an infant to recover from pneumococcal meningitis. As the child was already in hospital, early diagnosis and treatment were possible.

A preparation of pure white crystalline penicillin, containing 1,650 units per mg., which is much less irritating than the older impure preparations is now available. Certain points are thought to be of importance in the avoidance of reactions to the injections and of the introduction of secondary invading organisms. The penicillin used was prepared in separate doses of 30,000 units in 3 ml., each in a sealed ampoule; this prevents contamination of the penicillin solution. The small volume of the dose is, I feel, important, as large quantities of fluid may raise the tension of the cerebrospinal fluid considerably, particularly in small infants. I always aim, however,

at removing an equivalent volume of cerebrospinal fluid, plus 1 ml., before the injection. This should, of course, obviate that danger.

I wish to thank Dr. B. A. Young, medical superintendent, St. Alfege's Hospital, and Dr. B. Gottlieb, senior resident physician, for permission to publish this case, and the latter also for his help in preparing the paper.

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Therapeutic Scurvy

The following case is of interest in that it shows the importance of viewing the body requirements as a whole when placing any patient on a dietetic regime.

CASE REPORT

The patient, a married woman aged 42, with one son aged 16 alive and well, was admitted to hospital complaining of swelling of the left leg for four weeks, with bruising of two weeks' duration. Five weeks before admission she noticed that her teeth were tender and that her gums were swollen and bled easily. Several teeth were extracted without undue haemorrhage. A few days before admission she got a severe pain in the left thigh, with a "cramping" sensation in the calf and the foot. She was sent into hospital as a case of thrombophlebitis.

Vision was blurred, but there was no definite night blindness. On admission she noticed dryness of the skin. There were no pins-and-needles or paraesthesia, no soreness of tongue, and no haematuria. She had had no serious illnesses, but since the age of 8 had been subject to attacks of migraine, which had become more frequent two years ago. She consulted her doctor, who diagnosed nervous dyspepsia and put her on a diet. This consisted of 2 pints (1.14 litres) of milk a day, which was simmered for some while before being consumed. She also had three eggs a week, steamed fish, bread-and-butter, and milk puddings; but she had no vegetables or fruit of any kind. No drugs were taken except stomach powders.

On examination she was seen to be thin and pale, and her lower gums showed purple-red proliferations between the teeth. In the upper jaw, where the teeth had been extracted, there was no abnormality. Examination of the chest and abdomen revealed nothing abnormal. Blood pressure was 120/60. The left leg was markedly swollen, with discoloration of the whole thigh, the appearance being like an extensive bruise. Both legs showed many small petechiae. A blood count showed: haemoglobin, 58%; red cells, 2,800,000; white cells, 6,000; normal differential count. Blood ascorbic acid was 0.1 mg. per 100 ml. Urinary excretion of ascorbic acid was 2.15 mg. in 24 hours.

It was considered that minimal doses of vitamin C would be of value. She was put on an ascorbic-acid-free diet and given 15 mg. of ascorbic acid daily. After three days she noticed improvement in her teeth and the left leg rapidly became more comfortable. Nine days later objective improvement in the leg and gums could be seen. In four weeks there had been no further bleeding, the bruising had gone, and there were only traces of the petechial haemorrhages, but no fresh ones. After approximately three weeks the urinary ascorbic acid excretion was 7.1 mg. in 24 hours and the plasma ascorbic acid was 0.25 mg. per 100 ml. She was given 150 mg. of ascorbic acid daily and discharged from hospital, but it was noticed that large doses of vitamin C did not produce any particular beneficial effect.

The patient has been followed up since discharge. She still complains of being tired, but the haemorrhages have disappeared and apart from flatulent dyspepsia she has no symptoms.

COMMENT

This was a case of scurvy due to grossly deficient diet, and it serves to emphasize the importance of giving adequate vitamin C with many of the diets used for treating dyspepsias, especially if the milk is boiled. The very small amount of ascorbic acid which was required to cure the condition is also noteworthy. The minimum daily requirement of ascorbic acid for an adult is generally accepted as being in the region of 25 mg., but this patient was cured by a dose as low as 15 mg. daily. The impossibility of diagnosing scurvy from the gums of edentulous people is once again remarked.

I should like to acknowledge the help given me by Dr. J. R. P. O'Brien, of the Biochemical Laboratory, Radcliffe Infirmary, Oxford.

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Facial Seborrhoea in Arteriosclerotic Pseudobulbar Palsy

Profuse seborrhoea of the face associated with Parkinsonism is so characteristic of encephalitis lethargica that an exception to the rule is not without interest. In the following case the condition was due to arteriosclerotic degeneration of the brain.

CASE REPORT

The patient, a man aged 46, had never had seborrhoea until his present illness. Six months before admission his wife noticed that he seemed dull and sleepy, that he dribbled saliva, and that both hands were "shaky." He became slow in movement, irascible, and difficult to handle. His speech was slurred and his expression fixed.

On examination he looked older than his years, and his typically Parkinsonian face was thickly covered with a glistening film of sebum. The rest of the body was not so affected. He was given to fits of causeless weeping, but pathological laughter was not observed. He was disorientated for time and place, and showed defective memory for recent events. Insight and judgment were faulty, and he displayed a consistent disregard for cleanliness and the conventions. Speech was slow and slurred; it was embarrassed by Cheyne-Stokes respiration. There was no gross defect of sight. The fundi showed marked arteriosclerosis. There was a supranuclear paralysis of conjugate upward movement of the eyes, lateral and downward movements being intact. The tongue was spastic and could barely be protruded. Marked spastic rigidity of both arms and both legs was present. The left arm and leg were weaker than the right, and there was a left extensor plantar response; the right plantar was flexor. There was a coarse tremor of the fingers of both hands, but it was not typically Parkinsonian. Voluntary effort of one limb increased the rigidity of the opposite side of the body; consequently his incapacity was more pronounced when standing or attempting to walk than it was when lying down. He walked with a spastic shuffle and festination. There was no gross sensory loss, but his mental state precluded tests of the finer modalities. Sphincter control was intact.

The left ventricle was much enlarged. Blood pressure was 280/140. The urine contained a trace of albumin and a few hyaline casts. Blood urea was 60 mg. per 100 ml. The spinal fluid was normal in all respects, and the Wassermann reaction was negative in both fluid and blood.

Eight months after the first appearance of symptoms he had a series of epileptiform convulsions, between which he remained in an attitude of decerebrate rigidity. Twenty-four hours later he died.

The post-mortem report (Dr. J. G. Cummings) on the findings in the brain was as follows: "A recent haemorrhage filled the left hemisphere. Colloidin sections were cut from various parts of the brain and stained with haematoxylin, van Gieson, P.A.H., da Fano, and by the Gross method. Weigert-Pal preparations were also made. There is a widespread vascular abnormality, with marked proliferation of the intima and endarteritis. Atheromatous patches are present. The most marked damage is in the midbrain and pons, where in addition to vascular changes there are small zones of demyelination in relation to the vessels. The cerebellum shows slight changes of a similar type and also some loss of Purkinje cells. Very few zones of cellular infiltration are to be seen, but in a few cases in the midbrain and pons there is some perivascular lymphocytic infiltration in relation to damaged capillaries. The appearances are those of arteriosclerosis; there is no evidence of encephalitis."

COMMENT

This case is of interest in two respects. First, it illustrates the sound rule that encephalitis lethargica should not be diagnosed except during an epidemic of that disease: sporadic cases resembling it almost invariably turn out to be some other condition. Secondly, facial seborrhoea has not hitherto been described in pseudobulbar palsy. Its occurrence in this case is perhaps explained by the unusually rapid onset of facial rigidity, which, as in encephalitis lethargica, came on within a few weeks, in contrast to the usual ingravescent onset in arteriopathic Parkinsonism. The seborrhoea is limited to the face, and it is only in the face that rigid muscles, by their insertion into the skin, can impose immobility on the integument in which the sebaceous glands lie. It is suggested that the seborrhoea which occurs as a temporary phenomenon with rapidly developing Parkinsonian rigidity of the face has a mechanical explanation and is not due to a central autonomic disturbance. This view is in harmony with the current belief that the sebaceous glands are not under nervous control.

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