CASE REPORTS

CEREBRAL OEDEMA IN SCARLET FEVER

BY

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The literature contains no reference to cerebral oedema as an early complication of scarlet fever. It is likely, therefore, that the condition is extremely rare.

Case history

The patient, a boy aged fifteen years, gave a history of measles and chickenpox in infancy and of rheumatic fever nine years ago. His mother stated that the present illness started with sore throat, occipito-cervical headache, backache and a temperature of 101.2°F. These symptoms persisted for three days, vomiting occurred once, sordes was obstinate, marked thirst was satiated with abundance of fluids, and, in spite of profuse perspirations daily, the temperature gradually rose to 103.8°F. Early on the fourth day his doctor was called and prescribed sulphonamide; later on that day a deep red flush appeared on his neck and face. Throughout he had been drowsy, but talked quite sensibly. On the fifth day his condition had definitely improved, his throat was easier, his temperature had dropped two degrees, but as the rash persisted and had spread, he was sent to hospital.

On admission he looked ill, his face sallow and puffy, his cheeks desquamating in large flakes. On his neck was a dusky purplish rash, the unusual colour being due to multiple petechiae, and on the body and limbs a generalized punctate eruption of moderate severity. The palate and tonsils were swollen and brightly injected, the palate having a punctate appearance; the tonsils were covered by a yellow lacunar exudate which was easily removed. Some viscid mucus covered the mouth and fauces, while the tongue was at the ‘white-strawberry’ stage. The temperature was 101°F., the pulse of poor tension, pulse rate 100, respirations 20. He was given an intramuscular injection of concentrated and refined streptococcus antitoxin (Parke Davis and Co.), the dose being 9000 units.

On the following morning exfoliation of the mouth and tongue was complete; the tongue was then a ‘red-strawberry’, and the tonsils clean and bright red. The rash on the neck was still dusky and showed early desquamation, whilst the body rash had almost gone. The temperature was 97.6°F., the pulse 80.

In spite of the marked improvement in the clinical signs the patient appeared to be dull and listless, but answered unsmilingly in monosyllables when spoken to. Throughout the day he was able to read, or at least he pretended to, but made repeated attempts to get out of bed, occasionally requiring moderate
restraint as he was apparently unable at these times to realize fully what was being said to him or what he was doing. In the evening his condition had deteriorated still further: he was then definitely stuporose.

Examination of the heart, lungs and abdomen was completely negative; examination of the central nervous system, on the other hand, gave some interesting results. Superficial and deep reflexes were either absent or greatly diminished: the knee-jerks were weak and elicited with difficulty, the plantar responses were faint but definitely flexor, and the abdominal and epigastric reflexes absent. There was no stiffness nor retraction of the neck, Kernig's and Brudzinski's signs were negative, and there was no ankle clonus. There was no photophobia, nor nystagmus; the pupils were equal and regular with a normal degree of dilatation whilst ophthalmoscopic examination showed clear discs but some dilatation of the retinal veins. In reply to a shouted question he denied headache or pain. His co-operation in this examination was anything but active: his actions those of an automaton with a wooden and expressionless face. His temperature was 97.8° F., his pulse 70 and his respirations 18. The urine was normal in amount and contained no abnormal constituents.

He passed an extremely restless night, with little sleep till seven o'clock in the morning. When seen at ten o'clock on the seventh day of the illness, he was practically comatose, but could still be roused with difficulty. It was found he had had incontinence of urine between the hours of seven and ten. Examination gave the same results as on the previous evening. Lumbar puncture was considered, but his condition had deteriorated to such an extent that urgent treatment rather than leisurely diagnosis became imperative.

His head was lowered, he was given magnesium salts, one ounce by the mouth and one ounce by rectum, followed in a few minutes by castor oil, one ounce with thirty minims of coramine. These measures proving ineffectual, a soap and water enema was administered with a profuse watery result. Within a few minutes he sat up in bed and, looking round with a questioning gaze, engaged the nurse in animated conversation. Examination later in the day revealed normal and brisk reflexes, both superficial and deep, in an intelligent and smiling patient who had no memory of the previous two days.

His progress thereafter was wholly uneventful, his temperature remaining between 97° and 98° F., his pulse 54/60 for the next three days. He was able to get up on the fourteenth day of his stay in hospital, and to leave on the twenty-sixth day. He has remained perfectly fit since his discharge.

Discussion

The patient's condition on the day of admission to hospital was believed to be due to the toxaemia of a severe scarlet fever infection; on the second day, with a normal temperature and pulse and a clean tongue (strong presumptive evidence that the immediate toxaemia had been overcome), it was thought to reflect an innate dull mentality; whilst on the third day, in the presence of a gross and progressive deterioration in the mental picture, it became obvious that the cause was then some obscure brain condition.

A normal temperature, bradycardia, marked stupor or even coma, diminution or absence of superficial and deep reflexes, incontinence of urine and dilatation of retinal veins (all signs of some general cerebral irritation), led to the provisional diagnosis of cerebral oedema. Even without lumbar puncture, a procedure of doubtful diagnostic value in a non-inflammatory lesion of recent onset, the differential diagnosis gave rise to no great difficulty, as all the usual
complications of scarlet fever, and causes extraneous to it, could be rapidly excluded.

Serum anaphylaxis was ruled out, as the signs, both in time and type, were wholly inapplicable to such a condition, and as refined and concentrated serum, which reduces the risk of serum shock to a negligible minimum even in a susceptible patient, had been employed.

Meningismus has been said to occur as a rare and fleeting phenomenon at the onset of hypertoxic scarlet fever. It is doubtful if it exists as a clinical entity, and it is likely that the syndrome results from an attack of abortive serous meningitis. Meningitis in all its forms, and its sequelae, could be dismissed in the absence of headache, neck rigidity or retraction, positive Kernig’s and Brudzinski’s signs, pyrexia and suppurative otitis media. It is generally agreed that practically all cases of meningitis are secondary to infection of the middle ear and are extremely rare before the tenth day.

Uraemia was unlikely as the pupils were not contracted, the discs were clear, there was no headache, vomiting or convulsions, the tongue was clean, the breathing normal and the urine free from albumin with no retention or diminution in amount.

Cerebral oedema is usually represented by an increase in the cerebrospinal fluid, especially in the meshes of the pia, whilst the brain substance may be infiltrated with fluid and the amount of fluid in the ventricles may be increased. The symptoms are generally ill-defined. It is probable that two factors were involved in its production in the case under discussion: (a) toxaemia and (b) water intoxication.

(a) Toxaemia. The toxin in scarlet fever is absorbed into the system and gives rise to the rash and the constitutional symptoms. The rash is produced by dilatation of the skin capillaries, the fragility of walls of which is increased so that minute petechiae may occur in the flexures, or even elsewhere in the more severe case. The severity of the rash may therefore be taken as a true index of the toxicity of the infection and of the damage, potential or actual, to the capillaries in the internal organs. The primary object of treatment is to neutralize this toxin at the earliest possible moment, before it has become fixed or etched in the capillary walls, that is, before the fifth day. No matter at what stage antitoxin is given, if the amount is adequate, it will overcome the immediate toxaemia, that is, it will neutralize the whole of the circulating toxin in a few short hours, but the later the treatment the greater will be the damage to the capillaries.

In the present case, which was of toxic type as was shown by the severity of the constitutional symptoms and the presence of a petechial rash on the neck, antitoxin treatment was not instituted till the fifth day of the illness. Some complications, resulting from direct damage by the toxin itself, was not therefore unexpected. Probably the complication of this type most commonly seen is albuminuria, and it is believed to result from the action of toxin on the vessels of the kidney, especially those of the parenchyma and glomeruli. It would not therefore be unreasonable to suppose that cerebral oedema may occur as evidence of analogous damage to the minute vessels of the brain. The lesion may in
some degree be compared with that in cerebral concussion. Cannon (1901) says that this condition is characterized by the formation of diffuse multiple small thrombi and by the presence of punctate extravasations in the minute capillaries which cause a relative ischaemia of the parts, and that brain substance, like other tissues, when deprived of oxygen, takes up fluid from the circulating media and becomes oedematous.

(b) Water intoxication. This condition may have resulted from excessive water intake following profuse sweats: both these factors were present during the four days before admission to hospital. Helwig, Schutz and Currey (1935) suggest that injudicious forcing of fluids produces an upset in the salt-water balance of the body, or a disturbance in the normal isotonicity of the blood, with a resulting cerebral oedema with intoxication.

The object of treatment was to relieve the water-logged condition of the brain, and incidentally to reduce any increase of intracranial pressure which may have resulted from increase in the amount of fluid in the ventricles, by stimulating its circulation and consequent oxygenation, and by causing depletion of tissue fluids.

**Summary**

A case of cerebral oedema, occurring in the course of toxic scarlet fever infection, is recorded. The complication was an early one and probably resulted from toxaemia and from water intoxication. The immediate response to simple and appropriate treatment would seem to confirm the diagnosis.

**REFERENCES**
