A CASE OF TRANSVERSE MYELITIS FOLLOWING T.A.B. INOCULATION

BY

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Neurological complications following T.A.B. vaccination are rare. Miller (1954) has recently reviewed neurological sequelae of prophylactic inoculation. He has summarized 49 neurological syndromes following use of T.A.B. vaccine from the literature, to which he has added one case of his own. He includes one case of transverse myelitis, reported by Lemke (1943). In view of this rarity, a case is reported here in which a transverse myelitis of the cervical spinal cord occurred in a National Service soldier, beginning fifteen days after his receiving a second injection of T.A.B. vaccine.

Case Notes

Private D., a National Service man aged 18 years, of two months’ service, was admitted to Chester Military Hospital on 6th January, 1954, complaining of numbness and weakness of the legs and arms.

Family History.—Mother reported to have had brain fever, aged 8 years. Father died when patient was aged 8 years. No sibs.

Past History.—Had never been ill before. Left school at the age of 15 years. He tried various jobs, but finished up by working underground in the mines, where he was training to be a fireman. He did this for one year, leaving it to join the Army.

He joined the Army in November, 1953, and received inoculations and vaccinations as follows:

- 19th November, 1953 ... Schick Test: Negative.
- 28th November, 1953 ... T.A.B.: 0.2 ml.
- 4th December, 1953 ... Tetanus Toxoid: 1.0 ml.
- 21st December, 1953 ... Vaccination: Typical primary vaccination.
- 21st December, 1953 ... T.A.B.: 0.4 ml.

All inoculations and vaccinations were in the left arm.

On 5th January, 1954, the day before admission, he was out on an outdoor exercise. It was very cold, and he noticed that he felt “freezing cold” and was not shooting as well as usual, having difficulty in holding his rifle. On return to his barracks in South Wales, however, he felt perfectly well and went to bed feeling quite fit. He awoke during the night and noticed a feeling of tightness across his upper chest. He went to sleep again and on awakening in the morning he felt that his arms were very stiff, with pain under both armpits. He tried to
get out of bed and found that his arms were weak; he was unable to take any weight on them, to grip objects or to extend his arms, which were in a position of flexion across his trunk. Although he was able to stand, his right leg felt numb and weak. He was dressed by his mates and helped to breakfast; he then noticed his right leg was weak and uncontrolled. He was carried back to his bed and seen by his unit Medical Officer, who recorded temperature of 97.4°F., pulse 80, a vague transient headache, weakness of extension of both forearms and of grip, absent triceps jerks, a flexor plantar response on the left side, and a doubtful plantar response on the right side. Loss of sensation to pinprick over the right leg and both hands was noted. Nil else abnormal was recorded.

He was transferred by ambulance car to Chester Military Hospital. He stated his left leg began to feel cold and numb en route, and on arrival at Chester he could not use it.

There was no history of sore throat or cough, or of injury. He had been constipated for one day.

Physical Examination on admission late on 6th January, 1954, showed a pleasant lad, intelligent, co-operative, not drowsy. Temperature was 98°F., pulse rate 70, respiration rate 20.

Examination of the central nervous system showed no abnormality in the cranial nerves. There was loss of power of the abductors of both shoulders, and flaccid paralysis of all muscles of the arms and forearms with bilateral wrist drop. He was unable to sit up and intercostal movements were diminished. There was complete flaccid paralysis of the left leg, paralysis of extension of the right hip and leg and weakness of flexion of the right hip and leg. Both triceps jerks were absent, the biceps jerks and supinators were weak, the right knee jerk was diminished and the left knee jerk and both ankle jerks were absent. All abdominal reflexes were absent and plantar reflexes were not obtained. Sensation was diminished to pinprick and cotton-wool below the second thoracic dermatome approximately. Other systems showed no abnormality.

By morning on 7th January, 1954, all reflexes were absent, the upper level of loss of sensation to pinprick was more definite at the level of the second thoracic dermatome, and it was noted that loss of sensation was greatest peripherally. Retention of urine occurred.

Lumbar puncture produced a clear colourless fluid, pressure was 140 mm. with negative Queckenstedt test. Protein was 45 mgm. per 100 ml., no excess of globulin was found, sugar was 70 mgm. per 100 ml., chloride 760 mgm. per 100 ml., cell count was two lymphocytes per c.mm., W.R. and Lange were negative. Blood count showed 10,400 white blood cells per c.mm., polymorphs 58 per cent., lymphocytes 38 per cent., monocytes 3 per cent., and eosinophils 1 per cent. B.S.R. was 3 mm. in the first hour (Westergren). Throat swab showed normal commensals. Catheter specimen of urine was normal chemically and sterile on culture. Radiographs of cervical spine were normal.

Routine changing of position was instituted. Four-hourly temperature
charting, intake and output chart, daily enema, twice-daily catheterization, splinting of extremities and physiotherapy were all commenced, with vitamin B1 given parenterally. Penicillin 500,000 units four-hourly intramuscularly was commenced.

On 11th January, some general improvement was noticed, and knee jerks and biceps jerks were obtained. On 14th January it was noticed that the right plantar response was definitely extensor whilst the left was equivocal. Repeat of lumbar puncture showed protein content of 70 mgm. per 100 ml., no excess of globulin, 3 lymphocytes per c.mm. Other constituents were normal and pressure was 140 mm.

Improvement in strength and power continued gradually and on 22nd January he was passing urine normally, and by 26th January he had regained complete sphincter control. All reflexes were now present though weak, especially the left triceps jerk. The right supinator jerk was inverted. Early in February both plantar responses were extensor and deep tendon jerks became brisk in the legs only, with some spasticity in the left leg. Repeat lumbar puncture on 10th February showed clear fluid with pressure of 160 c.mm., protein 60 mgm. per 100 ml. and 6 lymphocytes per c.mm.

On 23rd February a detailed review showed considerable improvement. He was walking with assistance. There was weakness of the left trapezius, of abduction, internal and external rotation of the left shoulder. The left triceps was not acting, the right triceps was weak; there was still only slight power in extension of the wrists, best on the right side, whilst flexion of the wrists and all movements of the hands were nil. There was considerable wasting and weakness of the sternocostal head of the left pectoralis major. The spinal muscles had regained strength, though not completely; intercostal muscles above T4 were now contracting well. The legs still showed marked weakness in the hips and flexors of the left knee, and there was definite spasticity in the left leg. Examination of sensation then showed diminished sensation to pin-prick below T2 with greatest loss over the right leg; vibration sense was diminished in the right leg. No change in thermal sensitivity was found. The triceps jerks, especially the left, were diminished. The right supinator jerk was inverted. Knee jerks were exaggerated, both ankle jerks were brisk and both plantar responses were still extensor. At this time it was noted that the left cremasteric reflex and left lower abdominal reflex could now be elicited.

Improvement continued slowly, and on final discharge from Chester Military Hospital in October, 1954, he walked well and looked after himself unaided. He still had difficulty in use of the hands. There was some wasting of both groups of spinati bilaterally, of both pectorals with marked atrophy of the sternocostal head of the left pectoralis major, of the left triceps and of the small muscles of both hands. There was slight weakness of the left shoulder, especially of abduction above the horizontal and of internal rotation, and of the right shoulder to lesser degree. Weakness of extension of the left elbow, of extension of the left wrist and fingers, of flexion of the right wrist and fingers was marked, and there was little power in the intrinsic muscles of both hands and thumbs,
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especially on the left side. All intercostals contracted well, and spinal muscles were relatively normal. There was some wasting and weakness in the left buttock, with slight weakness of dorsiflexion of the left ankle and extension and adduction of the left hip. Sensory loss to pinprick had practically recovered, but there was still a level in the region of T6-7. The right leg was still less appreciative of pinprick. Vibration sense was normal. Reflexes showed increased tendon jerks apart from a very weak left triceps jerk and weak right triceps jerk. Both plantars remained extensor and abdominal reflexes were absent on the right-hand side. Bladder and bowel function were normal.

. DISCUSSION

It was considered that the spinal cord lesion was probably a thrombosis affecting the cervical cord. The absence of other apparent cause of the transverse myelitis—e.g., trauma, syphilis or other infection, bacterial or viral—and its relatively close sequence to his T.A.B. and other inoculations led to the consideration that it might be associated with those inoculations.

A post-vaccinal condition was thought to be unlikely as the cause. Post-vaccinal encephalomyelitis most commonly occurs following primary vaccination, especially in adults. Its incidence has been reported as 9 cases in 75,000 vaccinations (Fyfe and Fleming, 1943) and 45 cases in over 5,000,000 vaccinations (Greenberg and Applebaum, 1948). A total of some 815 cases have been recorded out of at least 30 million vaccinations. These cases are predominantly of affection of the brain, spinal cord and meninges, with an almost constant period of 10-12 days elapsing after vaccination, though isolated extremes of 2-26 days have been recorded. Though fatal in from 20 to 50 per cent. of cases, when recovery occurs this takes place rapidly and completely. Included are three cases of transverse myelitis. All cases occurred on the twelfth day after vaccination. The two cases reported by Fyfe and Fleming (1943) showed thoraco-lumbar girdle pain with meningeal irritation and minimal affect of the pyramidal tract on one side, with clear lumbar fluid under slightly increased pressure and 15 and 35 mononuclear cells per ml. Recovery was complete in nine and eight days respectively. The third case, incompletely recorded, died (Committee on Vaccination Reports, 1928 and 1930). Such cases contrast sharply with the case reported in respect of interval following vaccination, clinical picture and progress.

Neurological complications from tetanus toxoid are practically unknown. Wooling and Rushton (1950) report a case of brachial neuritis following five days after administration of tetanus toxoid.

Neurological sequelæ after T.A.B. inoculation have been classified as syndromes of radiculitis, or plexitis, and mononeuritis, of polyneuritis and Guillain-Barré syndrome, of Landry’s paralysis and myelitis, and of cerebral and meningeal forms. Such sequelæ occur most commonly after a second inoculation, usually coming on within fourteen days and most frequently on the third day after inoculation (Miller, 1954). In the case of myelitis described by
Lemke, however, three T.A.B. injections were given in September, 1941, and a further injection in April, 1942. Eight days after this, symptoms of a brachial neuritis commenced which was followed several days after by spread to the cervical spinal cord at the level of the seventh cervical segment. Death followed. In the case reported here, onset was on the fifteenth day after a second dose of T.A.B. vaccine twenty-three days after the first dose. The immediate effect was upon the cervical spinal cord involving the fifth cervical to first thoracic segments with no evidence of cerebral, brain stem or meningeal involvement. There was no further spread. Death did not occur, but recovery was far from complete, there being residual weakness in the muscles of the upper limbs, particularly those innervated from the eighth cervical and first dorsal segment, and slight spastic weakness of the lower limbs.

It is now thought that the pathogenesis of such neurological complications following prophylactic inoculation is that of an anaphylactic reaction affecting blood-vessels (Miller, 1954). Success in treatment of encephalomyelitis following rabies inoculations, where a similar mechanism has been postulated, has been reported with antihistamines (Pickar and Kramer, 1949) and cortisone (Garrison, 1952).

Anti-histamine drugs and cortisone were not used in the case reported here. However, in the light of the above remarks, it is felt that consideration should be given to their immediate use in all cases showing evidence of neurological lesions after prophylactic inoculation, especially where there is evidence of spinal cord involvement.

SUMMARY

A case of transverse myelitis following prophylactic T.A.B. inoculation is described. Certain features are briefly discussed.

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