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TICK PARALYSIS OR POLIOMYELITIS

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In his excellent summary of our knowledge concerning tick paralysis, Ransmeier (1949) notes that many cases of this condition (including his 4 cases) were referred as poliomyelitis. The seasonal incidence is the same, and many of the clinical manifestations overlap. Differential features in tick paralysis are stated to be the usual absence of marked fever, normal or near-normal cerebrospinal fluid findings, the relative absence of muscle spasm, little stiffness of the back or neck, marked ataxia preceding the paralysis, and the development of a diffuse, bilateral, ascending type of paralysis which, except in severe fatal cases, disappears rapidly on the complete removal of the tick. Sensory involvement is usually absent in both tick paralysis and poliomyelitis.

It is apparent that the differential features listed are not absolute but are a matter of degree, and the following case report is interesting in this regard:

CASE REPORT

The patient, a European male aged 16 months, had always been a healthy boy. On 13 June 1957 his parents noted that he was rubbing his right ear and his knees seemed to be hurting him. On the morning of 15 June he was fretful and refused to walk (he had been walking for some months). The next day he appeared mildly feverish and started 'shaking', and when seen by his family doctor on 17 June there was just detectable weakness of the right leg. When seen in consultation as a possible case of poliomyelitis on 18 June, he showed slight neck stiffness and some non-specific weakness of both legs, which were tender on movement. The knee and ankle jerks were absent, he was afebrile, and the rest of the physical examination was negative. The right ear canal could not be visualized because of wax (the history of ear rubbing was not obtained at this time). His condition steadily deteriorated over the next 2 days, with increasing tenderness and weakness of both legs, and when seen again on 20 June he was obviously a very sick boy with neck retraction and respiratory difficulty. No leg movements could be elicited, he was unable to sit or lift his head, and the leg-straightening test was strongly positive.

There was no sensory loss. Other non-neurological physical signs were still absent but this time a tick was visualized in the right ear canal. Over a period of some hours the tick was floated out (alive) with liquid paraffin and within 12 hours the child was laughing, showed no further respiratory distress, and could lift his head. By 26 June he was sitting up and standing with assistance and a re-examination on 30 August 1957 showed only very slight residual weakness of the dorsiflexors of the right foot, with an apparent complete return to normal by the end of September.

No treatment was given during the illness save for ½ tablet of aspirin twice a day for the first 5 days.

At the time of the illness the child had not been out of Germiston for 3 months and there had been no cases of poliomyelitis in the municipality during that period. The family owned two dogs from whom ticks had occasionally been obtained and which played with the child in a sand-pit in the back yard.

The tick was identified by Dr. F. Zumpt, of the South African Institute for Medical Research, as a nymphal specimen of *Otobius megnini* (Duges), commonly known as the spiny ear tick.

A blood count at the height of the illness gave a leucocyte 12,300 per c.mm., with 25·5% neutrophils, 6·5% monocytes, 66% lymphocytes and 2% eosinophils. No poliovirus was identified from the blood but Type-I poliovirus was isolated (confirmed on a repeat check) from a stool specimen by the Poliomyelitis Research Laboratory, Rietfontein.

DISCUSSION

Were it not for the identification of poliovirus from a stool specimen (this information reached us 2 months after the clinical illness), the acceptable diagnosis in this case would have been one of tick paralysis. The preliminary ataxia, apyrexia, moderate leucocytosis, little stiffness of the back and neck, and the diffuse, bilateral, ascending type of paralysis, together with the relatively dramatic initial recovery on removal of the tick, would all be in favour of this diagnosis. Although most authors on the subject state that the syndrome is produced by gravid female hard ticks (family *Ixodidae*) only, Ross and Oxer (quoted by Theiler, 1949) report that sufficiently large numbers of nymphs of the Australian paralysis-tick, Ixodes holocyclus can produce paralysis in experimental animals.

Otobius megnini (which is a soft tick of the family Argasidae) is considered (Parish, 1949) a serious pest of domestic animals in semi-arid regions of the world and nymphs have been collected from the ears of cattle, sheep, goats, dogs, deer and man. The ear tick is unusual in that it lives its whole parasitic existence on the one host where the larval and

nymphal stages are passed in the external ear canal. After a last engorgement the ticks fall to the ground for their final moult into adults and mating and ovideposition. The adults do not feed. The only soft tick previously reported (in Russia) as being related to paralysis in humans is *Ornithodorus lahorensis*, when present in large numbers.

Whether the finding of the tick was coincidental, or whether the finding of poliovirus was coincidental, or whether the two agents were synergistically related, cannot now be established, but this case does arouse doubt in one's mind about the validity of the diagnosis in certain other reported clinical cases of tick paralysis, especially where complete recovery did not occur in a few days. Most of the literature on tick paralysis antedates the time when tissue-culture techniques became readily available for the identification of poliovirus and I can find no reference to cases where a search for this virus has been conducted along modern lines. In the 4 African cases reported by Veneroni (quoted by Stanbury and Huyck, 1945), by Zumpt and Glajchen (1950), and by Erasmus (1952), the clinical syndromes described could have been due to a viral aetiology, though not necessarily so. Is it unreasonable to suggest that in a certain number of reported cases of tick paralysis the tick may have acted as a local non-specific irritant with a consequent increased likelihood of poliovirus involvement of the corresponding segment of the spinal cord as has been frequently reported with other non-specific irritants?

SUMMARY

The close similarity between the clinical picture of tick paralysis and poliomyelitis is pointed out.

A case is reported which could easily have been one of tick paralysis and where the removal of a tick (*Otobius megnini*) from the ear canal led to a dramatic clinical improvement, but from which Type-I poliovirus was recovered from the stool.

If this case was one of tick paralysis it is the first human case reported caused by a single soft tick (family *Argasidae*).

It is suggested that in certain other recorded cases of tick paralysis a viral aetiology has not been convincingly excluded and the possibility is mooted that a tick bite may act as a localizing and precipitating factor for systemic viral infections.

I wish to thank Dr. A. Jankowitz for letting me see the patient, and Drs. J. H. S. Gear and F. Zumpt for their help.

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