Tick-Borne Meningopolyneuritis
(Garin-Bujadoux, Bannwarth)

RUDOLF ACKERMANN, M.D., PETER HÖRSTRUP, M.D.,
AND ROGER SCHMIDT, M.D.

Neurologic University Clinic of Cologne, Federal Republic of Germany

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We studied 100 patients with tick-borne meningopolyneuritis (Garin-Bujadoux, Bannwarth), the neurologic component of European erythema chronicum migrans disease. They had intense radicular pain, asymmetric polyneuritis combined often with uni- or bilateral facial palsy, lymphocytic meningitis without or with only slight meningismus, and a course lasting three to five months. Neurologic abnormalities were preceded by the bite of a tick or an insect in 37 percent of patients or by an erythema in 41 percent. In addition, many patients had extraneural involvement, such as fever or fatigue. The outcome was favorable in all cases, and occurred faster with antibiotic treatment, but a few patients had slight residual peripheral nervous system deficits.

Tick-borne meningopolyneuritis, which occurs in most European countries, is a clinical syndrome characterized by intense radicular pain, signs of peripheral nervous system involvement, and chronic lymphocytic meningitis. Compared with most non-suppurative infections, the signs and course of this syndrome are unusual. Extraordinary features include the variety and asymmetry of the peripheral nerve involvement, the absence of meningeal signs in most of the cases, and a course of four to six months. These features caused many errors in the discovery of this disease. It was assumed that different presentations were different diseases. In addition, the syndrome was rediscovered several times and given different names such as Paralysie per les Tiques, Meningo-Myelo-Radiculitis nach Zeckenbiss, Polyradiculonévrite atypique avec hyperalbuminorachie et pléiocytose, and Neuro-Radikulomeningo-Encephalo-Myelitis. Only a few authors were aware of a possible causative connection with the preceding tick bite and erythema reported by some of the patients. Today we know that tick-borne meningopolyneuritis is one manifestation of erythema chronicum migrans disease, which is a tick-borne spirochetosis.

Forty years ago the German neurologist Bannwarth [1,2] tried to establish the clinical characteristics of the syndrome. The principal signs were intense radicular pains, lymphocytic meningitis without meningeal signs, and involvement of the peripheral nervous system, particularly facial palsy. He thought that the preceding erythema reported by some of his patients was erysipelas. Therefore, he presumed that the disease had a rheumatic etiology. Peripheral nervous signs often started in the region of the preceding erythema and pain. Bannwarth called the syndrome chronic lymphocytic meningitis with the clinical syndrome of neuralgia or neuritis. None of his patients reported a tick bite.

At the time of Bannwarth’s studies, this possible mode of transmission as well as the migrating character of the erythema had already been reported in a local medical
journal about a single case from Lyons, France. At the location of a tick bite the man had developed a spreading erythema, intensive pain, peripheral pareses of one arm, and meningitis with a favorable outcome. But this publication by Garin and Bujadoux in 1922 [3] remained unnoticed for decades.

Both Hellerström (1930) [4] and Gelbjerg-Hansen (1945) [5] made the connection between the neurologic syndrome and the preceding erythema which they recognized as erythema chronicum migrans described at the beginning of this century by Afzelius [6] and Lipschütz [7]. Gelbjerg-Hansen recommended that physicians ask patients about preceding tick bite and erythema in all cases of unexplained meningoencephalitis.

Schaltenbrand [8] as well as Bammer and Schenk [9] explained the accentuation of erythema, radicular pains, and multiplex type of polyneuritis, which occurred in the location of the tick bite, as centripetal spreading of the infection along peripheral nerves and lymph vessels. Erbslöh [10] and Wolf [11] emphasized the seasonal appearance of the syndrome in summer and autumn as well as its changing frequency over the years. They believed that these features favored the hypothesis of arthropod transmission.

In 1973, Hörstrup and Ackermann studied the spectrum of this unique syndrome in 47 patients [12]. The signs, symptoms, and course show that the syndrome is a unique nosologic entity. Additional clinical observations during the past ten years [13] have deepened the knowledge about this multifaceted and protracted disease.

METHODS

This report is based on 100 patients seen at the Neurologic University Clinic of Cologne, North Rhine-Westphalia, Federal Republic of Germany, between 1956 and 1983. Sixty-four were male and 36, female. Tick-borne meningopolyneuritis was diagnosed by the presence of prolonged radicular pains, chronic lymphocytic meningitis, and, in most cases, signs and symptoms of peripheral nervous system involvement. Before the etiology of the disease was known, the protracted course and locally produced IgM in the CSF also supported the diagnosis.

RESULTS

General Characteristics

Ages of the patients ranged from 12 to 76 years; peak periods were the third and sixth decades (Fig. 1). Only 27 (27 percent) patients reported a tick bite, 10 (10 percent) an "insect bite," and 41 (41 percent) a preceding erythema. The onset of the ill-

![FIG. 1. The age distribution of patients.](image-url)
ness occurred from February to December, most commonly in July and August (27 and 25 patients) (Fig. 2). Most of the patients came from in or around Cologne. Arthralgia was reported by one patient and another had a severe gonarthrosis at the same time as the meningopolyneuritis. Patients often had extraneural involvement such as fever or fatigue.

**Radicular Pain and Other Sensory Irritations**

At the beginning of nervous system involvement, all but four patients had severe radicular pains, paresthesias, or hyperesthesias, which were the reason for hospitalization in one-fifth of the patients. During the following weeks the pains remained in the same region, involved other regions, or moved completely to other locations.

**Peripheral Nerve Lesions**

Peripheral nervous system abnormalities often started and were most severe in the region of the pains. They showed no relation to the region of the tick bite and the erythema. Ninety-one of the patients showed motor and 57, sensory lesions (Table 1). Sensory loss was usually less severe than motor. Four of the patients had only sensory signs, and five had no neurologic abnormalities on examination. The lesions developed over a period ranging from days to a maximum of one month, but late recurrences were not seen.

Cranial neuritis was very common (68 patients) (Table 2), and in 28 of the patients was combined with pareses of extremities. Twenty-two of the patients showed

| Localization of Pareses | N | Trunk | Extremities |
|------------------------|---|-------|-------------|
| Cranial nerves         | 40| 5     | 4           |
| Extremities            | 22| 5     | 12          |
| Cranial nerves and extremities | 28| 10   | 17          |
| Without pareses        | 9 | 4     |             |
| Abdominal muscles      | 1 |       |             |
| **Total**              | 100| 24    | 33          |
TABLE 2
Tick-Borne Meningopolyneuritis: Involvement of Cranial Nerves
(N = 68)

| Cranial nerves | I  | II | III | IV | V  | VI | VII | VIII | IX-X | XI | XII |
|----------------|----|----|-----|----|----|----|-----|------|------|----|-----|
| Frequency      | —  | 6  | 3   | 1  | 9  | 8  | 63  | 3    | 4    | 3  | —   |

pareses of extremities without cranial neuritis. Most common was facial palsy (63 percent), which was bilateral in 27 cases (Table 2). Slight papilla edema was seen in five of the patients. One had an engorged papilla with hemorrhage. Other cranial nerves were involved less frequently.

The pareses of extremities ranged from mild to severe. In most of the cases, they were distributed asymmetrically (Fig. 3) in the manner of polyneuritis multiplex. They could not be attributed to single nerves. Tendon reflexes were often diminished, sometimes including extremities without pareses. In severe cases, denervation potentials and reduced motor conduction velocity were found during the later course of the disease. Sensory impairment as well as the early sensory irritations were usually distributed asymmetrically and, particularly at the extremities, nonsystemically. All types of sensation were involved, especially superficial sensations.

Meningitis

Only 19 patients had meningismus, often found only on extreme flexion. It was sometimes associated with headache, nausea, vomiting, and photophobia. All other 81 patients were free of meningeal signs. Cerebrospinal fluid analysis showed a pleocytosis of 11 to 906 cells/mm³, mostly lympho-, reticulo-, and plasma cells; total protein was 45 to 360 mg/dl. Among 37 patients investigated by laser nephelometry, locally synthesized IgM could be demonstrated in 29, IgG in 21, and IgA in 12. Spinal fluid abnormalities lasted up to five months (Fig. 4).

Encephalitis and Myelitis

Cerebral involvement was relatively rare. Twenty patients had poor memory, impaired concentration, or behavioral changes, and one had slight somnolence. A

![Diagram of cranial neuritis](image)

![Diagram of extremity pareses](image)

![Diagram of extremity pareses and cranial neuritis](image)

FIG. 3. The distribution of pareses.
positive Babinski sign and retention of urine were seen in one patient each. Abnormal electroencephalograms were found in many patients. These abnormalities included generalized slowing or dysrhythmia.

**Clinical Course**

Erythema, pains, and neurologic signs with meningitis followed the tick bite in a characteristic sequence (Fig. 5). The erythema began a mean of two weeks after the tick bite, radicular pains five and one-half weeks after that, and neurologic signs eight and one-half weeks later. The erythema lasted a mean of one week, pains seven weeks, and neurologic signs and meningitis nine weeks.

The outcome was favorable in all 100 patients. Even severe pareses returned to normal in most of the cases. After several years, only a few patients had slight residual pareses. One patient had a moderate facial palsy after nine years. Eleven patients treated with antibiotics seemed to recover faster from pains and pareses.

**Laboratory Findings**

Some of the patients had increased sedimentation rates or elevated serum IgM values. Neutralizing tick-borne encephalitis virus antibodies could not be demonstrated in any patients.
DISCUSSION

Tick-borne meningopolyneuritis can usually be recognized by the neurologic picture alone, but many patients also have a history of tick bite or erythema chronicum migrans, or have locally synthesized IgM in the CSF. It is important to make the correct diagnosis because of the urgency of specific treatment. Our clinical evaluation of 100 patients confirms previous reports of a few or single cases. Based on these series, the common features and wide variety of neurological involvement in this disease are now clear. However, other diseases with polyneuritis multiplex, chronic lymphocytic meningitis, or tick bite must be considered in the differential diagnosis. The determination of antibody titers against the causative spirochete will facilitate the diagnosis and may allow the recognition of additional aspects of this multifaceted disease. For example, cases may exist without CSF alterations or courses may occur that are as chronic as neurosyphilis.

Tick-borne meningopolyneuritis is very similar to the nervous system involvement of Lyme disease; but Lyme disease seems to be more severe, involves the central nervous system more often, and produces longer courses with more recurrences [14].

We could not confirm the hypothesis of centripetal migration of the infection along peripheral nerves. In our experience, the area of peripheral nervous impairment did not occur within the dermatome of the tick bite or the erythema. Today, it is known that the spirochete spreads systemically. However, the pathogenesis of peripheral nerve lesions, which sometimes resemble neuralgic amyotrophy or the vascular forms of polyneuritis, remains unclear.

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