PECULIARITIES IN THE CLINICAL COURSE OF GUILLAIN-BARRE'S POLYRADICULONEURITIS

D. Minchev, I. Nikolov, V. Hristov

Key-words: Guillain-Barre's polyradiculoneuritis — neurological alterations — electromyography — diagnosis

The polyradiculoneuritic syndrome, type Guillain-Barre-Strol, can be manifested from a clinical and clinico-diagnostic point of view with various symptomatics and wealth of differential-diagnostic possibilities (1—4, 7, 8). We observed a more peculiar course of the neurological symptomatics and an atypical characteristics of the illness in seven clinical cases treated in the Clinic of Neurology, Higher Institute of Medicine, Varna and Tolbuhin, during the period from 1970 till 1982.

The aim of the present work is to outline the peculiarities of the symptomatics and to discuss some pathophysiological mechanisms that accompany Guillain-Barre's polyradiculoneuritis illustrating two of our followed-up observations.

Case 1. The patient N. N. A., age 25 years, clinical record No 20354/1970. The present diseases started about 6 months ago when playing the accordion the patient showed a painful cramp of the fingers of the right hand which disappeared after massage. In the next weeks a general faintness, easy tiredness, induration of posterior right leg musculature during squating. After 4 months a weakness of both legs and paresthesiae under the knees develop for one week only. The right leg tucks when going at the talocrural joint resulting in patient's falling over. Simultaneously, paresthesia in the arms under elbows appears, and on the anterior upper part of the thorax he feels and sees by himself rapid muscullar fibrillations. These phenomena are soon followed by femoral musculature weakness that makes standing up from squatting position impossible.

Neurological status: craniocerebral nerves normal. Delayed and limited active movements of arms under elbows, diminished finger strength, slight hypotonia and absent periosteal and tendon reflexes, moderate hypotrophy of interosseal musculature, thenar and hypothenar. Similar deviations are observed in both legs under the knees — absent periosteal and tendon reflexes, bilateral peroneal paresis, torpid paretic gait and interosseal hypotrophy. There is a hypesthesia for any kinds of superficial sensitivity of distal type in both arms and legs. The laboratory examination shows the following pathological levels: RSE 40/88 mm, after 2 weeks 4/15 mm; Weltmann 7,5; McLagan 79 PU; after 2 weeks 6 U and 55 PU. LOR consultation examination — chronic peritonsillitis. Cerebrospinal fluid — Pandy++, N. Apelt+, Weichbrot+, Rivalta(—), Pavlovich(—). Total protein 95 mg %, after 20 days — 72 mg %, colloid curve of right type. Biopsy from m. triceps surae dextra (No 5602): a transversely striated muscle tissue with pseudohypertrophic pattern. Skin with normal histological structure. EMG: presence of spontaneous activity consisting of fibrillations, positive peak waves and scarce fascicullations in m. interosseus dorsi Ist, m. extensor digitorum brevis and m. abductor hallucis bilaterally. Predominantly polyphasic action potentials with prolonged duration and medial layout. Reduced conduction speed

through motor fibers of peroneal and tibial nerves. The 2-months long therapy with neurorestitution drugs, remedial gymnastics, electroprocedures without corticosteroids (because of diagnosed duodenal ulcer) did not cause any essential improvement.

On 5th February, 1971 (C. R. No 2380) the patient is admitted to hospital again without any change of the neurological status. Lumbar puncture is normal. Two weeks after maintenance of an adequate treatment including dehydrocortison up to 30 mg a sudden change sets in and 9 months after the onset of the disease a rapid progressive improvement of motor functions of the extremities occurs. Hand power increases up to 40/50 kg and the patient begins to walk on heels, to run and squat without any effort. However, myotonic painful cramps in biceps musculature of the arms more expressed at the left side persisted till his discharge from hospital. He was recommended a balneological treatment after discharge in April, 1971.

Case 2. K. S. R., age 11 years, C. R. No 16538/1981. The illness begins about 10 days before hospitalization with pains in the legs without any apparent ground, 1—2 days long. After the 3rd day the gait becomes unstable, the child is not able to go upstairs and to stand up from squatting position. Anamnestically, there were no previous diseases in postnatal development. The somatic status is normal. Neurologically, there is a markedly diminished strength of the legs. The examination reveals looseness of humeral joints, an expressed hypotonia of both legs and arms musculature. There is an outlined lumbar lordosis at standing, the gait has a myopathic features, and the child can not stand up from squatting position without using his arms. There is an absence of periosteal and tendon reflexes when legs are concerned.

Laboratory examination: rheumatic activity test — no abnormalities. Lumbar cerebrospinal fluid: total protein 256 mg %, glucose 86 mg %, chlorides 731 %, no cells. After 2 weeks, respectively, total protein 198 mg %, after 2 months 40 mg %, no cells. EMG: The data about fibrillations in most muscles, the increased percentage of polyphasic action potentials, the rarefied recording and diminished conduction speed through motor fibres of fibular, elbow and radial nerves bilaterally give an evidence for a diffuse damage of peripheral nerves — a polyneuropathia.

Corticosteroid therapy together with neurorestitution drugs and remedial gymnastics 2 months long caused an increase of arm strength and leg one, the child began to move independently, to squat, stand up on toes and heels. After a continuing ambulatory treatment with medicaments and remedial gymnastics in the 4th month after the onset of the illness the complaints completely disappeared but only periosteal and tendon areflexia of the legs persisted. The latter disappeared after 3 months, too.

The analysis of the clinical characteristics of these cases is remarkable for the following peculiarities:

1. A long onset (4 months) with painful muscle cramps and distal paresthesiae which are designated as rare in the literature (1, 9, 10).

2. The rather slow almost chronic course with a severe, but reversible symptomatics creates similarity of these cases to the chronic polyradiculoneuritis with tendency towards healing (8).

3. The proximal motor insufficiency with a myopathic appearance, distal muscular painful cramps and weakness without the involvement of the craniocerebral nerves, in combination with sensitive disorders of distal type presents a rare clinical combination described as "pseudomyopathic" (1, 3, 4, 8).

4. These facts indicate that when this disease is concerned the process does not affect only the peripheral motor neuron but also primarily as well as in a various manner the transversely striated musculature (myotonic manifestations and a histologic pattern of muscular pseudohypertrophy). This connects both exogenic polyneurites and chronic progressive degenerative diseases of the muscular apparatus, as J. McComas (6) proved in his clinical and experimental investigations.

In our opinion, the awareness of the clinical variations of symptomatics and course of the polyneuritis, type Guillain-Barre facilitates the correct diagnosis and timely therapeutic behaviour.

REFERENCES

1. Божинов, С., И. Георгиев. *Неврол., психиатр. и неврохирург.*, 1965, № 3, 147—181. — 2. Георгиев, И., и др. *Неврол., психиатр. и неврохирург.*, 1980, № 1, 3—7. — 3. Крайндлер, А., Б. Проскауер-Апостол. Детска неврология. С., Медицина и физкултура, 1967. — 4. Цукер, М. Б. Инфекционные заболевания нервной системы у детей. М., Медгиз, 1963, 219—222. — 5. Bodechtel, G. Diagnostic differential des maladies neurologique. Paris, 1965, 21—90. — 6. Mc, Comas, J. A. Neuromuscular function and disorders. London, 1977, 111—120. — 7. Guillain, G. J., A. Barre, A. Strol. *Bull. Soc. méd. Hop., Paris*, 10, 1916, 1462. — 8. Magnussen, M. *Acta Psychiatr. Neurol.*, 21, 1946, 561. — 9. Martin, J. J. *Acta Neurol. Psychiatr. Belg.*, 60, 1960, No 12, 1087. — 10. Martin, J. J., M. Philiport. *Welt. Neurol.*, 3, 1962, No 5, 409—430.

ОСОБЕННОСТИ КЛИНИЧЕСКОГО ПРОТЕКАНИЯ ПОЛИРАДИКУЛОНЕВРИТА ТИПА ГИЛЕН-БАРЕ

Д. Минчев, И. Николов, В. Христов

РЕЗЮМЕ

В работе приведены современные взгляды на этиопатогенез, большое разнообразие клинической картины и на протекание полирадикулоневритного синдрома типа Гилен—Баре. Проведено обобщение данных клинических наблюдений семи сходных между собой хронических случаев с преобладающими псевдомиопатическими проявлениями.

Авторы считают, что при этом заболевании воспалительный процесс не только охватывает периферический двигательный нейрон, но и первично затрагивает поперечнополосатую мускулатуру. Эти данные позволяют говорить о связи между экзогенными полиневритами и хроническими прогрессирующими дегенеративными заболеваниями мышечного аппарата.