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ASSOCIATION OF PATIENTS WITH PARKINSONISM AND EPILEPSY WITH EEG CHANGES

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A b s t r a c t: EEG is an important diagnostic method for epilepsies. EEG changes in patients with Parkinson's disease and epilepsy are not unknown or unexpected events. We investigated 250 Parkinson's disease patients. Epileptic seizures were recorded in 10 patients. Four patients had generalized tonic clonic seizures; 3 partial complex seizures, 2 of which with secondary generalization; while 3 patients had right-handed simple motor partial seizure. Clinical and neurophysiological investigations, neuroimagining techniques electroencephalographic investigations (EEG, EEG in sleep) were conducted. Our patients were well controlled with antiepileptic therapy.

Key words: parkinsonism, epileptic seizures, EEG.

Introduction

EEG is an important diagnostic method which is used in patients with epilepsy. EEG changes in patients with Parkinson's disease and with epilepsy are not unexpected or unknown. Cerebrovascular diseases, neoplasmas, neurodegenerative diseases, epileptic seizures are problem which need attention, and present particular diagnostic and differential diagnostic problems. In literature, there is brief information about the presence of epileptic seizures in patients with Parkinson's disease, also or EEG changes in this type of patients.

The aim of this study is to describe in detail the types of epileptic seizures and the EEG changes, before and after accepting therapy, in 250 patients with Parkinson's disease, a followed over 6-year period. The patients were

diagnosed with detailed anamnesis, acomplete neurological examination, with the use of morphologic (CT and MRI) and neurophysiological investigations (EEG, EEG in sleep, VEP, SEP and BAEP).

Ten patients with different types of epileptic seizures and their EEG changes were keyed and presented in this study.

Clinical and neurophysiological investigations, materials and methods neuroimaging techniques, keyed and CT of brain, electroencephalographic investigations (EEG, EEG in sleep) were made in 250 patients with Parkinson's disease. The period of investigation was 6 years (1996–2001), the age of the patients ranged from 43 to 72 years. Epileptic seizures were registered in 10 patients (6 women and 4 men) aged 50 to 65 years.

EEG was performed with an 18-channel apparatus, in 4 standard montages by the International system 10–20.

VEPs (visual evoked potentials) were made on the a Tönnis apparatus for evoked potentials, with visual "Pattern-shift" stimulation in the standard technique. EP were acquired with 128 biosignals, in which the latency, amplitude and morphology of VEP were analyzed.

SEPs (somatosensory evoked potentials) was carried out with the same apparatus as for EP, meanwhile teasing the nervus medianus and nervus tibialis. SEP was performed with superficial electrodes fixed above the somatosensory region of the head and scalp. Acquired SEP analysed the latency, amplitude and morphology of EP.

BAEP (Brainstem auditory evoked potentials) were obtained with sound stimulation, a click with minimal power in one ear, and another is a bar with a whisper. Admission of EP was with electrodes which are put into the vertex and the procesus masstoideus. Characteristic BAEP responses consisting of 5 positive-negative waves were obtained, each one with a selfsource of genesis. Latency, amplitude and morphology of EP were analysed.

Generalized tonic clonic seizures (GTCS) were registrated in 5 (2%) of patients, in 2 of them this was registrated in sleeping. Three patients had partial complex seizures (1.2 %), the seizures were with secondary generalization in 2 of them. Simple motor partial seizures were registrated in 3 patients.

Results

In patients with GTCS (2%), in precisely 2 of them, the illness was manifesting in the form of slowness in daily activities, bradylalia, gait disability, rigidity and cognitive changes.

In another 2 patients, the symptoms were manifested by headaches, tremor in the right hand, weakness and rigidity in the same hand, one of them

frequently had a queer gait disability and cognitive changes. Three years after the beginning of the illness, sudden loss of consciousness, froth in the mouth and urination after an attack was noticed. In 2 patients the attacks were nightly. All 4 patients were examined in detail. In the first 2 patients, EEG demonstrated irregular basic brain activity, with presentation of FIRDA (focal intermittent rhythmic delta activity). Cortico-subcorticaly atrophic and leucoencephalopaty changes were noticed paraventricularly. EP (VEP and SEP) was normal. In another 2 patients the EEG verified bilateral, centrotemporal, almost continuous, mild left side lateralization, on an average voltage of theta waves of 5 Hz. Paroxysmal intermittent activity was not verified. EEG was made in sleep, which verifies the presence of slow waves, with bilateral synchronization, with clearly irritative phenomena, without clear lateralization. BAEP was carvied out in 2 patients which signifiee decrescence of amplitude in the first and second component. All 4 patients recived therapy with Carbamazepine.

Psychological testing was done of all the patients, only in 1 patient was the result normal, the other 3 patients had psychomotor slowness, concentration problems, weariness and affection of motor performances. Control EEG was made in 1 patient (who had night seizures), which verified a significant slowness, in a patient with normal psychological tests. Seizures were partially complex in 3other patients, in 2 patients with secondary generalization. In 1 of these 3 patients, EEG was focally replaced with a spike-wave complex in the temproparietal region. In 2 patients with secondary generalization seizures, EEG verified focal activity with biphasic discharges (spikes with secondary generalization changes) and forms of bilateral paroxysm of spikes and irregular theta waves.

In the first patient, problems were present with rigidity, tremor and bradykonesia, in which after 8 months attacks appeared, showed interesting and unexpected phenomena of improved Parkinsonism symptoms. In 2 other patients the problems started with tremor, hemidistonia in the right hand, and in one of them palsy of vertical gaze. In 1 patient the seizures were associated with affective symptomatology and the presence of changes in consciouness.In one of the two remaining patients, attacks were attended by intellectual and cognitive symptoms with ideoaffective changes, and in 1 of them an attack had psychosensory symptomatology and sensory hallucinations. EP was normal in all 3 patients, CT of the brain was normal in the first patient, in the other 2 patients focal brain atrophy was verified. Simple motor seizures in the right hand were registered. Their initial symptoms were choreoatetotic movements of the head and extremities, bradylalia, rigidity, bradykinesia and vegetative symptoms. In these patients, attacks of short tonic and clonic contractions were registered for 6 months, localized in the right hand extremities; their duration

was 3–5 minutes. In all 3 patients the EEG was normal, but showed a focus in the left motor region and an increase of beta activity.

CT and MRI of the brain produced normal findings, also EP was normal. Patients were put on therapy with Carbamazepine.

Parkinson's disease is a neurodegenerative illness of frequent Discussion n.p. presence. In our series of 250 patients, 10 of them had epileptic seizures and all of them were elderly (50–65 years old, 6 women and 4 men). Four patients had generalized tonic clonic seizures, 3 partial complex seizures and 2 secondary generalization, while 3 patients had right-hand simple motor partial seizures. All our patients were well controlled with antiepileptic therapy (Carbamazepine in required doses) over a 2-and 4-years period. Five to six months after starting therapy, EEG was improved in 6 patients, only in 1 patient EEG changes were present even altough they had not epileptic seizures. In the further period control EEGs were made and all had normal finding.

Despland, PA (1992), has discussed epileptic seizures in older patients, stressing the diagnostic problems and differential diagnoses from which depended therapy and prognosis of the disease.

Bogousslavsky *et al.* (1992) explained the epileptic seizures in 48 patients with cerebrovascular attack, describing postictal palsy and epileptic sequences which were present during the night.

Jankowicz, E *et al.* (2000) denonstrated marked delta activity in EEG in patients without therapy, patients who were treated with neuroptective therapy showed lessened delta activity. In cases treated with high doses of Levodopa, associated with presentation of hyperkinetic side-effects, slower waves were noticed in the EEG. High Levodopa doses certainly evoked cerebral dysfunction.

Our experiences showed that in 250 patients with Parkinsonism, 4 % of older patients showed epileptic seizures, 2 % with GTCS and 1.2 % with partial complex seizures.

Krakow, K *et al*. (1999) presented the incidence of EEG changes in patients with Parkinson's disease. These were more frequent than in the rest of the population, but features of epileptic attacks were less.The most fregnent changes were generalized slowness of EEG with presentation of delta activity.

Yoshioka, A *et al.* (1999) discussed that it is possible to explain features of myoclonias with electrophysiological methods (Magnetic electroencephalogaphy and SEP). It is supposed that myoclonias were of the cortical origin, but their patophysiologic mechanism is unknown as yet.

In discussion, it is important to point out the risk of dementia in patients with epileptic seizures, Parkinson's disease and EEG changes. The risk of

dementia is especially high in patients during the early hases of Parkinson's disease.

Conclusion

Epileptic seizures are not very rare in patients with Parkinson's disease. In their etiopathologenesis probably are included common pathophysiological mechanisms. These epileptic seizures are symptomatic, maybe a consequence of degenerative brain process and respond well to antiepileptic therapy.

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Резиме

АСОЦИРАНОСТ НА БОЛНИ СО ПАРКИНСОНИЗАМ И ЕПИЛЕПСИЈА СО ЕЕГ ПРОМЕНИ

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ЕЕГ е многу важна дијагностичка метода што се применува кај пациентите со епилепсија. ЕЕГ промените кај пациентите со паркинсонизам и со епилепсија се сè уште недоволно познати и неочекувани. Ние испитувавме 250 пациенти со разни форми на паркинсонизам. Епилептични напади беа регистрирани кај 10 пациенти. Четири пациенти имаа генерализирани

тоничко-клонични напади, 3 пациенти беа со парцијални комплексни напади, од кои двајца со секундарна генерализација, додека 3 пациенти имаа едноставни моторни напади на десната рака. Кај сите нив беа направени клинички и неврофизиолошки инвестигации (ЕЕГ, ЕЕГ во спиење). Нападите кај сите пациенти беа добро контролирани со антиепилептична терапија.

Клучни зборови: паркинсонизам, епилептични напади, ЕЕ

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