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Paranoid syndrome and herpes simplex encephalitis – coincidence or casual nexus?

Over 90% of all non-purulent encephalitis and cerebrospinal meningitis have viral etiology, resulting mainly from exogenous infections, or more seldom endogenous ones. In such cases it comes to activating delitescent chronic viral infection in conditions of reduced cellular immunity. The viruses often locate themselves in OUN neural ganglion, specially trifacial nerve. Then, the disease may start with neurological disorders, usually focal ones, which are sometimes joined by meningeal symptoms. In about 20% of patients, imperceptible meningeal symptoms, or an absolute lack of them were noticed. Cases like these are caused by among others, viruses from group Herpes Simplex, type 1 (HSV 1). In the case of herpes simplex encephalitis (HSE), diagnostic difficulties are caused by psychical disorders which mostly resemble paranoid or maniac complex without precursory symptoms indicating general viral infection (2,5). At first, patients are hospitalized at psychiatric wards, and only the appearing neurological symptoms indicating organic cause of the disease turn doctors' attention to encephalitis. The course of HSE depends on the kind of pathogen (HSV1 or HSV2), Seriousness of the clinical syndrome, the patient's general state and the efficiency of the treatment. In Lin's et al investigations, it was found that some genetic inclinations are responsible for the appearance of the HSE symptoms (4). Probably the gene of apolipoprotein E (allel epsilon 2) is a risk factor of HSE. Immunity decrease resulting from various reasons may activate the herpes virus in the organism.

The prognosis is very serious because HSV virus brings about quick necrotic-hemorrhagic changes of cerebrospinal meningitis and brain, especially in the area of temporal and limbic cortex (1,5). Since acyclovir was used, the death rate has decreased from 70% to 30%. Sometimes the disease finishes with the patients' recovery, some times there remain permanent consequences in the form of hemiparesis, epilepsy, psychoorganic changes as cognitive and affective disturbances (3). There also appear relapses after finishing the treatment.

CASE REPORT

A 25-year old patient, unmarried woman with technical education, unemployed, living with her family was brought to district hospital because of serious aggravation of her psychical state: the patient used to escape from home, demolished her house, uttered delusions, she had hallucinations, was frightened, stimulated and aggressive. She had been under psychiatric ambulatory treatment for about 4 months because she changed: she withdrew into herself, avoided contacts with other people, revealed anxiety, suspiciousness, at night sleeplessness, she

lost her appetite. The paranoid syndrome was diagnosed. As it appears from family anamnesis, the course of pregnancy and delivery and also the patient's upgrowth in early childhood were normal. At the proper time she went to primary school, she did not repeat any classes. She left Elementary Technical School 7 years ago, she did not take up a job anywhere; for all the time she lived with her parents. She had not been seriously somatically ill. There had not been any psychic diseases or alcoholism in the family.

In the ward, she was at first stimulated psychomotorially, she did not come to contact, in evident fear, anxiety; she gave the impression of hallucinating auditorily. Because of the patient's aggressive behavior she was immobilized mechanically. Treatment with neuroleptics was applied and because of bronchitis an antibiotic was given. In the neurological examination there were no symptoms of OUN damages.

After about one and a half day, the patient's general state got worse: high temperature of 39°C, consciousness disturbances, fine trembling of the whole body, incitement in the limits of bed. The patient was sent to the Neurology Ward. Except consciousness intensity disturbances, the examination found involuntary movements in the form of grimacing in the limits of the face, lower extremities trembling, involuntary upper extremities movements of 'pinching' character, positive symptoms of neck stiffness and Kernig symptom eyeballs in divergent strabismus, more shallow nasal-labial fold, muscle tension aggravation in all extremities. Eyegrounds and examined cerebromedullary liquid were correct. Because of breath disturbances: Cheyne-Stokes breath and serious general state, temperature of 41°C, the patient was brought to the Intensive Therapy Ward. At the ward, the cerebromedullary liquid was examined once more and it was proper. Bacteriological inoculation showed the presence of saprophytic bacteria: *Streptococcus viridans*. Tomography examinations of the patient's head were executed twice at a 3-week interval.

The first result of head CT, showed the existing inflammatory hypodense areas of temporal lobes and slight reactive changes in the subarachnoid area of frontal lobes of both cerebral hemispheres. In the second CT examination of the patient's head, they found a slight extension of the fourth ventricle and great cistern (about 15×26 mm), the third ventricle was vestigially widened. The lateral ventricles were of proper size, without ventricular system displacement. There was decreased density of white substance of diffuse character, especially next to lateral ventricles symmetrically, including the sphere callosum. In the CT, there appeared oozing mastoiditis symptoms (more visible in the right side). A consulting specialist on infectious diseases confirmed the diagnosis of cerebritis.

The patient was hospitalized totally for 41 days, her breathing was controlled by respirator, the basic vital functions were monitored. She was sedated with Dormicum, all the time the general serious state remained. The treatment was with antibiotics, antiviral medicine and steroids. In spite of intensive treatment, the patient died of circulatory-respiratory insufficiency, not regaining consciousness. In the autopsy they found macroscopically: brain congestion and swelling; histopathologically: congestion, micro-embolisms, ischaemia foci and brain congestion. The congestion of internal organs tissue was discovered. Herpes simplex encephalitis was diagnosed.

DISCUSSION

Herpes simplex encephalitis is a serious disease burdened with a high death rate; that is why caution is necessary. The disease lasts for a few to twenty days. It sometimes starts subacutely as anxiety, depression, sluggishness, vegetative disorders, which is caused by affecting limbic system (5). In the described case, in the beginning the patient had psychical disorders during four months before the appearance of acute symptoms of cerebritis. Whether they were prodromal symptoms resulting from slow virus HSV-1 activity in the brain, or there

came to the overlap of infecting the brain with endogenous virus to the beginning of schizophrenic process, we are not completely sure.

CONCLUSIONS

1. For psychiatrists: a) necessity of regarding the possibility of organic, in it neurological backgrounds of psychotic disorders, b) duty of using of a wide range of diagnosis in the case of having an inkling of an inflammatory state of brain in spite of proper results of cerebromedullary liquid examination, c) quick pharmacological intervention with the use of antiviral drugs is necessary in case of temperature increase and consciousness disturbances.

2. For neurologists: a) need of taking into account a contingency of coexistence of psychic state disorders and organic brain diseases, especially the situation in so called neurologically 'silent' parts of brain as frontal lobes, or limbic system, b) lack of focal neurological symptoms does not bespeak of a total lack of developing disease.

REFERENCES

1. Andreula C. et al.: Viral encephalitis, Rivista Neuroradiol., 10, 3, 357, 1997.
2. Fisher C.M.: Hypomanic symptoms caused by herpes simplex encephalitis, Neurol., 47, 6, 1374, 1996.
3. Hokkanen L., Launes J.: Cognitive recovery instead of decline after acute encephalitis: A prospective follow up study. J. Neurol. Neurosurg. Psych., 63, 2, 222, 1997.
4. Lin W.R., Woźniak M.A., Esiri M.M. et al.: Herpes simplex encephalitis: Involvement of apolipoproteine E genotype. J. Neur. Neurosurg. Psych., 70, 1, 117, 2001.
5. Wald I., Członkowska A.: Neurologia kliniczna. PZWL, Warszawa 1987.

SUMMARY

Patients with viral infection of the central nervous system are often difficult to establish the correct diagnosis. This paper presents our knowledge of herpes simplex encephalitis and a case report of 25-year-old woman. In this case paranoid syndrome preceded viral encephalitis. We established that the presence of mental abnormalities could go before encephalitis or could dominate in symptomatology of encephalitis.

Syndrom paranoidalny a wirusowe zapalenie mózgu – przypadek czy powiązanie?

Trudno jest postawić prawidłową diagnozę w przypadku nietypowych objawów wirusowego zapalenia opon mózgowo-rdzeniowych i mózgu. W niniejszej pracy przedstawiono na przykładzie 25-letniej chorej problemy diagnostyczne występujące w sytuacji zainfekowania wirusem *Herpes simplex* ośrodkowego układu nerwowego. Występujące zaburzenia psychotyczne w okresie prodromalnym infekcji jak i w czasie ostrych objawów mogą być jedynym przejawem opryszczkowego zapalenia mózgu i są obarczone znaczną śmiertelnością.