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Retrospective evaluation of cases of CNS toxoplasmosis in patients with AIDS hospitalized in the Department of Hospital for Infectious Diseases, Warsaw

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Purpose of the study

The aim of the study was to characterize retrospectively the group of patients (pts) with central nervous system toxoplasmosis and AIDS.

Methods

Data were collected according to basic pts' profile, including immunological status, ARV treatment, clinical presentation of disease, treatment pattern and outcome: clinical state and neuro-imaging results were taken into consideration mainly. Autopsy examination, if performed, was evaluated as well.

Summary of results

The examined group consisted of 40 pts hospitalized in 4th Department of Regional Hospital for Infectious Diseases in Warsaw, between 1994 and 2007; 33 men and 7 women were included. Age ranged from 24 to 49 (average 34) years. 73% of patients had severe immunological deficit (CD4 + < 100 cells/mm3) when CNS toxoplasmosis was diagnosed. Focal neurological deficiency, fever and disturbances of attention and concentration were the most frequent clinical signs of the disease (respectively, 71%, 68%, 65%). Neuro-imaging revealed single (56% in CT) or multiple (70.6% in MRI) typical focal lesion/s with peripheral enhancement with contrast (35% in CT, 77% in MRI) and brain edema (63% in CT and 65% in MRI) in most cases. Majority of pts (63%) were treated with sulfadiazine, pyrimetamine and folinic acid. In 14/27 (52%) pts treated with sulfadiazine, adverse events complicated therapy: allergic reaction 64%, nephrotoxicity 21%, neutropenia 7%. Complete recovery was achieved in 38% of pts; 18% pts died, the rest had some neurological complications as remnant: epilepsy 18%, hemiparesis 13%, cerebellar ataxia 5%. In the group of pts that died, toxoplasmosis of CNS often co-existed with other neuropathology as CMV infection of CNS (42%) or HIV encephalitis (34%) which were revealed in autopsy examinations. Most of pts that died of CNS toxoplasmosis (57,9%) did not receive ARV treatment, in opposition to all survivors. The secondary prophylaxis of CNS toxoplasmosis was broken by the relapse of the disease in two (9%) cases. Six cases of CNS toxoplasmosis were developed despite the primary prophylaxis.

Conclusion

Most of our pts with CNS toxoplasmosis had severe immunodeficiency. Pts in our group presented various clinical signs of the disease. Neuro-imaging of CNS toxoplasmosis revealed mainly single in CT but multiple in MRI focal lesions. Therapy with sulfadiazine was often associated with side-effects. The prophylaxis of CNS toxoplasmosis can be insufficient in some cases.