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Paper Poster Session I

Clinical and diagnostic parasitology

Intracerebral toxoplasmal and cryptococcal co-infection in immunocompromised patient with systemic lupus erythematosus

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Objectives: Despite a significant increase in the survival rate of patients with systemic lupus erythematosus (SLE), opportunistic infections represent a significant cause of morbidity and mortality. Risk factors include immunosuppressive theraphies as well as some manifestations of active SLE itself. Clinicians need to be aware about the possibility of polymicrobial infections which may cause diagnostic and therapeutic delay.

Methods: We report a case of intracerebral coinfection with *Cryptococcus neoformans* and *Toxoplasma gondii* in a 29-years-old man with SLE and congenital IgA deficit, under treatment with steroids. The patient, suffering from one week of fever, vomit and diarrhea, was admitted to our hospital for a syncopal episod following which did not regain consciousness. A neurological consultancy took over stiffness in all four limbs and lockjaw, coma with decortication reaction to painful stimuli, mydriatic pupils. Brain MRI with gadolinium contrast showed the presence of interhemispheric multiple focal lesions enhancement.

Results: Cerebrospinal fluid (CSF) examinations revealed 24 white blood cells/µl, glucose 1 mg/dl and protein 266 mg/dl. An India-ink preparation of CSF disclosed mucinous capsule of Cryptococcus as a translucent halo surrounding budding yeast. A latex agglutination test was positive for cryptococcal antigen at a diluition of 1:4096 and CSF culture grew *C. neoformans.* HIV serology was negative but lymphocyte and CD4 lymphocyte count were 308/µl and 190/µl, respectively. CD4/CD8 ratio was 2.00. He was treated with liposomal amphotericin B (2 mg/kg/die) and dexamethasone (32 mg/die). Because of severe immunosuprression, other opportunistic pathogens were investigated. A qualitative in house PCR resulted positive for *T. gondii* DNA (B1 gene) in CSF, peripheral blood mononuclear cells and serum. Anamnestic *T. gondii* serology was negative. The therapeutic regimen was promptly strengthened with cotrimoxazole (20mg/kg/die+100mg/Kg/die), but a new CT scan demonstrated a diffuse cerebral swelling. Because of severe general impairment, the patient died two days later.

Conclusions: Patients with severe SLE under immunosuppressive treatment with steroids may undergo acute infections including those from virus**,** common bacteria and *Mycobacteria, fungi (i.e C. neoformans)* and parasites (i.e *T. gondii)* which can occur alone or rarely in combination. In this regard, only one case of intracerebral coinfection with *Burkholderia pseudomalley* and *C. neoformans* has been recently described. To our knowledge, the presented case is the first describing an intracerebral coinfection with both *C. neoformans* and *T. gondii* in a patient with SLE. Monitoring CD4 lymphocyte count is highly recommended in patients with SLE under immunosuppressive treatment; if lower than 200/µl, they should assume cotrimoxazole prophylaxis, as HIV+ patients. In case of detection of brain lesions, rapid molecular diagnosis for more than one opportunistic infection should be taken into account as failure to timely recognition of coinfections may lead to insufficient treatment and affect outcome.