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Amphotericin-B-liposomal/azathioprine/prednisolone

Fungal central skull-base osteomyelitis secondary to the nasopharyngeal mucormycosis and progressive leucopenia: case report

A woman [exact age at the reaction onset not stated] was described, who developed fungal central skull-base osteomyelitis secondary to the nasopharyngeal mucormycosis during treatment with prednisolone for sarcoidosis. Additionally, she developed progressive leucopenia during treatment with amphotericin-B-liposomal for nasopharyngeal mucormycosis and azathioprine for sarcoidosis [not all dosages and route stated].

The woman was presented to the medicine department with complaints of loss of appetite, generalised weakness, occasional difficulty in breathing and decreased sleep for duration of 4 months. Based on haematological and radiological findings, a diagnosis of sarcoidosis was made. Therefore, she was scheduled to receive treatment with oral prednisolone 40mg for 8 months. Her medical history was significant for type 2 diabetes mellitus and hypothyroidism for 10 years, which were well controlled on metformin, vildagliptin, gliclazide and levothyroxine sodium [thyroxine; Thyronorm]. She was admitted to the emergency department of a hospital in India with left eye ptosis, left lateral gaze rectus palsy and decreased sensation over the left cheek for the past one month. The contrast-enhanced MRI of the head and neck revealed nasopharyngeal abscess with heterogenous enhancement in the left cavernous sinus and loss of T2 flow voids in the left internal carotid artery. Additionally, destruction of the left pterygoid plate, the floor of the middle cranial fossa with involvement of the left carotid canal was noted. The positron emission tomography-CT scan revealed necrotic lesion in the nasopharyngeal wall with involvement of fossa of Rosenmuller and the left carotid canal. Additionally, multiple supraclavicular, mediastinal, paratracheal, precarinal and bilateral hilar lymph nodes were also noted. The CT angiography showed complete thrombosis of the left internal carotid artery and involvement directly in the necrotic lesion. Therefore, she was treated with enoxaparin sodium [enoxaparin]. Additionally, she was managed on insulin to control increase blood sugar level, and her levothyroxine sodium therapy was continued for hypothyroidism. Thereafter, emergency endoscopic debridement was done, which showed a necrotic lesion in the nasopharynx with black crusts destroying sphenoid bone, pterygoids and prevertebral body. The potassium hydroxide mount demonstrated broad aseptate hyphae, which was suggestive of mucormycosis. Based on the clinical presentation and laboratory findings, fungal central skull-base osteomyelitis secondary to the nasopharyngeal mucormycosis was considered, which was related to the prednisolone therapy.

The woman started receiving amphotericin-B-liposomal [liposomal amphotericin-B] injection, and her therapy with prednisolone was stopped. Thereafter, she started receiving azathioprine for sarcoidosis. However, she had no neurological deficit and angiography revealed good cross circulation. During the course of treatment (amphotericin-B-liposomal and azathioprine), she developed progressive leucopenia (total leucocyte count of 1100 /µL). After medical consultation, leucopenia was considered to be related to the amphotericin-B-liposomal and azathioprine therapy. Therefore, her therapy with azathioprine was stopped; however, amphotericin-B-liposomal therapy was continued. She then underwent bone marrow biopsy for establishing diagnosis, which showed megaloblastic picture only. Thereafter, she received a cumulative dose of amphotericin-B-liposomal 4g injection. However, no improvement was noted in leucopenia and again medicine referral was done. Thereafter, her therapy with amphotericin-B-liposomal was stopped, and she was treated with granulocyte macrophage colony stimulating factor due to persistent leucopenia. She then started receiving posaconazole. As a result, total leucocyte count improved to 9500/µL. Additionally, ptosis and gaze palsy gradually improved in the left eye. There were no complaints of decreased vision or diplopia, and she was discharged in a haemodynamically stable condition on posaconazole therapy. On discharge, monthly follow-up visits was advised.

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