Aseptic necrosis or osteonecrosis is a condition that occurs when the blood supply to the bone is disrupted. This leads to death of osteocytes, dead tissue reabsorption and overall osseous tissue weakening, which can lead to subchondral fractures and collapse.

The pathophysiological mechanism of the development of osteonecrosis involves multiple factors. Increased differentiation of bone marrow cells into adipocytes, abnormality of lipid metabolism and clotting events, decreased angiogenesis and elevated vasoconstriction, oxidation injuries and finally a genetic predisposition; are the factors involved in the development of osteonecrosis.

OBJECTIVES

- To present a case of iatrogenic induced multiple osteonecrosis from high methylprednisolone treatment, the second case at the Pediatric Hospital, ’Mother Teresa’ University Hospital Center.
- To present the clinical state of the patient and the evolution.
- To address the preventing measures and management approach, since corticosteroids are widely used in clinical prac-

CLINICAL PRESENTATION

The patient A.N. is diagnosed with Craniofaryngioma at the age of four years old and undergoes three neurosurgical interventions because of the tumor’s recidivism. The third intervention is at the age of six years old.

The patient presents at our clinic at the age of five and a half years old, after his second neurosurgical intervention when he presents with left hemiconvulsions. AED therapy with Carbamazepine therapy is started and is continued for 5 years. During this time the child remains seizure free and the CBZ is terminated. Six months after the AED termination the child presents generalized seizures refractory to treatment. Valproic acid is started followed by Lamotrigine. The seizures stop.

One month later, at the age of fifteen years old the patient manifests a hypersensitivity skin reaction with erythema multiforme and generalized desquamation of the skin. Diagnosis of Lamotrigine induced Stevens-Johnson syndrome is made and the child is inappropriately admitted at the adult dermatology clinic where he is treated with IV Methylprednisolone, 150mg/day for 4 weeks. During the last days of treatment, the boy feels severe back pain while walking, falls in the ground on his knees and is unable to walk anymore. At this moment he gets admitted at our clinic.

At the moment of admission, the condition and overall state of the patient were dramatic and severe. Marked Cushingoid appearance is noticed. The skin features hyper pigmented maps and striae all over the body, with a predominance on the lower extremities. Knee joints and talo-crural joints were severely painful and edematous, dexter-nilser. Palpation of abdomen showed hepatomegaly, 4.5 cm under the rib cage. Neurological exam showed bilateral tremor and left pyramidal syndrome.

A multidisciplinary team is gathered to evaluate the situation and a panel of laboratory assays and imaging studies are ordered, with results and short comments as follow:

- CBC, Biochemical panel, Blood Electrolytes, Coagulation tests: without significant changes.
- Hormonal panel and pituitary axis show normal levels of TSH, ACTH and Cortisol.
- Adrenal and Heart ultrasound: normal.
- Bone marrow examination shows normal marrow activity.

TREATMENT AND MANAGEMENT

- Anti resorptive therapy is initiated with bisphosphonates. Pamidronic Acid 60mg over the course of 3 months. Calcium supplementation + Vit D3 3600 UI/day
- Pain management: Ibuprofen, up to 1200mg/day
- Gastric protection: Omeprazol 40mg/day
- Immunoglobulin therapy: IVig Pentaglobin, (one cycle).
- AED therapy with Valproate and Clobazam.
- Bed rest.
- The patient gets transferred to a specialized center in Germany for orthopedic treatment.

DISCUSSION

The literature is poor regarding prevention of osteonecrosis. Multiple agents and methods have been tried in animals with promising results but controlled human studies are absent. The agents and methods used in animal studies include: Lipid lowering agent on the prevention of steroid induced osteonecrosis. Conclusions of the steroid-induced osteonecrosis study in rabbits by intra bone marrow injection of autolo-