Charcot’s Knee Joints

A 48 years male farmer presented with history of progressively increasing painless swelling left knee for 3 years followed by instability in knee joint and inability to walk without support for last 4 months. There was also history of progressively increasing painless swelling right knee for last 3 months. There was history suggestive of lightning pains in lower limbs. There was no history of bowel or bladder involvement. The patient was a social drinker. Local examination of left knee revealed diffuse swelling all around knee variable in consistency, non-tender with normal local temperature (Fig. 1). The joint stability was lost. Right knee also had a diffuse globular swelling, non-tender, firm to hard consistency (Fig. 1). In the CNS examination higher mental functions, cranial nerves and speech were normal. Motor system examination revealed wasting of left lower limb. Tendon reflex at knee and ankle were absent with mute plantars. Sensory examination revealed impaired vibration and position in lower limbs. Rest of the examination and review of other systems was normal. Investigations revealed normal hemogram and blood biochemistry. Fasting and postprandial blood sugar estimation was normal. Blood VDRL test was reactive (Titre=16). CSF biochemistry examination showed proteins 65 mg% and sugar 44 mg%. The cytology had 6 WBCs/ cmm with majority of lymphocytes. CSF VDRL test was reactive. Serum TPHA was positive (1:320 dilution). X-ray left knee joint revealed 6 D’S i.e destruction, dislocation, disorganization, distention, debris and increased density (Fig. 2). X-ray right knee also revealed destruction, disorganization, distention, debris and increased density (Fig. 3). In view of above clinical features and investigations patient was diagnosed to have Charcot’s knee joints with tabetic neurosyphilis (tabes dorsalis).

Charcot’s joints or neuropathic joint disease first described by Jean-Martin Charcot a French Neurologist in tabes dorsalis in 1868 is a progressive degenerative arthropathy associated with decreased sensory innervation of the involved joints. Neuropathic arthropathies are considered to be an accelerated osteoarthritis that is precipitated by trauma to a joint not protected by its proprioceptive or nociceptive reflexes. This process continues until destruction of the joint occurs. The common causes of Charcot joints includes diabetic neuropathy (most common cause nowadays), tabes dorsalis and syringomyelia.1 In tabes dorsalis Charcot joint is seen in 4-10% patients and weight-bearing knee joint is predominantly involved.2 The general consensus is that the loss of proprioception and deep sensation leads to recurrent trauma, the damage goes unnoticed by the neuropathic patient and ultimately leads to progressive destruction, degeneration, and disorganization of the joint. Another theory postulates that neurally mediated vascular reflex results in hyperemia, which leads to increased osteoclastic bone resorption. Both of these theories probably play a role in the development of a Charcot joint.

S Raina*, SS Kaushal**, D Gupta***, A Goyal#, V Sood#
*Senior Resident; **Professor and Head; ***Associate Professor; #P G Student; Department of Medicine, Indira Gandhi Medical College, Shimla – 171 001 (H.P.).
Received : 13.2.2007; Accepted : 4.10.2007

REFERENCES