Keywords: Sever's disease, heel pain, children

A 10 years old girl presented with history of bilateral heel pain, which started about one year back. It was gradually increasing in severity, and had worsened over one month period. Parents or child were not able to correlate the pain with any trauma and it was spontaneous in onset. It was aggravated by physical activity especially running or jumping and relieved by rest. On physical examination, child was well-nourished, with normal vitals and normal systemic examination findings. She had a limping gait with toewalking in between. Local examination of feet revealed no swelling, warmth or redness; however on palpation posterior part of heel was markedly tender, more on left side. Results of her blood investigations including complete blood count, erythrocyte sedimentation rate, C-reactive proteins, serum calcium, liver and renal function tests, anti-streptolysin O (ASO) titre and rheumatoid factor were normal. Radiograph of feet showed increased sclerosis and fragmentation within the calcaneal apophysis (Figure 1). A diagnosis of Sever's disease was made and patient was advised to take rest and to avoid activities that cause heel pain e.g. sports, jumping or running. Medication in the form of diclofenac gel for local application and oral ibuprofen for two weeks was prescribed. On follow-up after one and half months, pain was decreased significantly and now she was able to walk comfortably.

Fig 1 - X-ray ankle showing increased sclerosis and fragmentation of calcaneal apophysis

Sever’s disease or calcaneal apophysitis is a painful inflammation of the calcaneal apophysis, which is thought to be caused by overuse or repetitive microtrauma due to increased traction by achilles tendon on its insertion site i.e. secondary growth plate of the calcaneus. (1-3) The calcaneal growth centre appears at approximately seven years of age and fuses by around 13 years in girls and 15 years in boys, (4,5) hence it is more commonly seen in growing children. Sever’s disease is characterised by heel pain near lower posterior aspect of the calcaneus. Incidence or prevalence of Sever’s disease is not very well documented and according to western literatures, posterior heel pain associated with Sever’s disease comprise 2-16% of musculoskeletal injuries in children. (6) James Warren Sever first described this condition in 1912 as an inflammation of the calcaneal apophysis associated with muscle strain in immature skeleton, resulting in posterior heel pains, swelling and difficulty in walking. (7) Several different theories are proposed to explain the etiopathogenesis of posterior heel pain in Sever’s disease: 1) Rapid growth spurt during pubertal age increases relative tension in the tendoachilles/ triceps surae complex with resultant increased traction on apophysis and microfractures. (2,3,8,9) 2) Children with cavus or planus foot are more prone to calcaneal apophysitis, probably due to increased strain on the affected area secondary to harder heel strike placing. (10,11) 3) Repetitive microtrauma causing mechanical disruption is an important causative factor. (12,13) 4) Obesity and weight bearing has also been proposed as an influential factor in calcaneal apophysitis. (5,7) 5) Infection of the affected area as a causative agent was also proposed by some authors but it has been included in differential diagnosis rather than cause by most of the authors. (2,14) However, of these no single theory could gain widespread acceptance due to limitation of clinical data to support them. Recently conducted case control studies reported biomechanical malalignment and gastrocnemius equinus as possible predisposing factors. (11,15) Usual age of presentation of sever’s disease is typically adolescent period, usually between 8 and 15 years. (2,3) Diagnosis can be made on clinical examination alone. Radiographic changes seen in Sever’s disease include increased sclerosis and fragmentation within the calcaneal apophysis, which are non-specific and can be a normal feature of developing calcaneus. (16) Other causes of heel pain in children include achilles tendonitis, calcaneal stress fracture, retrocalcaneal bursitis, calcaneal cysts, osteomyelitis, and plantar fasciitis, which can usually be differentiated by thorough clinical examination and good radiological evaluation. Treatment aims at reducing the present pain and preventing recurrences. Parents must also be reassured about its self-limiting nature and that surgery is usually not required in majority of cases. Just rest and cessation of sports activity resolves symptoms with resumption of sports in 1-2 months in majority. (3) In patients with mild to moderate symptoms other options include wearing inner-shoe heel lift, padding for shock absorption, strapping of heel, monitored stretching or strengthening, pre and post-sport icing, and judicious use of anti-inflammatory agents (NSAIDs). In severe cases, immobilisation using cast in mild equinus for 2-3 wk relieves pain and surgery in the form of total or partial removal of apophysis is rarely required. (17)
symptomatic treatment. The poison she had consumed wash and her clothes were changed. She received pediatric intensive care unit (PICU) and given stomach system was within normal limits. She was shifted to air. Examination of cardiovascular and respiratory pupils were bilaterally mid-dilated sluggishly reactive were stable with very minimal oral secretions. Her vitals was irritable with dystonic movements. Her vitals before referring to our hospital. On admission she was given stomach wash and treated with atropine to have consumed organophosphorus poison and taken to a nearby hospital where she was suspected hours prior to admission to our hospital. She was first poison. She had consumed the poison about 16 with history of consumption of some insecticide with imidacloprid. She was Confidor® which contains imidacloprid. Within few hours, she went into shock and was put on ionotropic support. However she succumbed inspite of next few hours her condition deteriorated. She became stuporous with shallow breathing and her oxygen saturation was dropping even with supplemental blood gases, renal functions, blood glucose, coagulation oxygen. She was intubated and connected to ventilator. few hours her condition deteriorated. She became stuporous with shallow breathing and her oxygen saturation was dropping even with supplemental blood gases, renal functions, blood glucose, coagulation oxygen. She was intubated and connected to ventilator. Saturation was dropping even with supplemental oxygen. She was intubated and connected to ventilator.

REFERENCES