Abnormally prominent coracoid process is a rare finding. The only published case with grossly prominent coracoid was reported in 1936 as a congenital condition called os coraco-sternale vestigiale. Comparative findings are present in few other congenital or traumatic conditions as a part of bony or joint pathology. In contrast, abnormally prominent coracoids in a young tetraplegic brings attention to an entirely different aetiology.

A 15-year-old malnourished boy was admitted as a case of traumatic spinal cord injury (SCI) at C5 level with a body mass index of 11.4 kg/m². His injury was ten months old and he had been totally bed ridden since then. He was observed to have conical prominences at both shoulders anteriorly which were not there before the trauma. The possibility of birth related injury or congenital deformity was excluded based upon history. A shoulder deformity was suspected related to trauma. These abnormal prominences were identified as coracoid processes. Shoulder pathology was ruled out by performing detailed examination and further imaging was normal. Radiographs and CT imaging did not show any overgrowth, elongation or angulation of coracoids. On the left side, both pectorals were wasted while on the right side, pectoralis minor was intact and seen through wasted pectoralis major. Normally this muscle is neither seen nor felt. This muscle atrophy was due to SCI with patchy unequal paralysis between sides and malnutrition, making the coracoids abnormally prominent (Figure I).

Muscle atrophy in SCI patients can be multi-factorial. It can be due to denervation atrophy, disuse atrophy and fatigue in paralyzed muscles. Denervation atrophy results from injury to the motor neurons in the spinal cord or to the motor nerves in the ventral roots through which they exit. On the other hand, disuse atrophy occurs as a result of loss of muscle activation due to disruption to the central and segmental synaptic drive onto the surviving spinal motor neurons. After an acute SCI, a hyper-metabolic, catabolic cascade is also initiated which results in nitrogen losses, loss of lean muscle mass and deterioration of nutritional status, all of which occur within few weeks of injury.

In view of such complications, early rehabilitation interventions are important. These include physical therapy, occupational therapy and use of assistive technology. Use of functional electric stimulation, tendon transfers and muscle reinnervation are other options to prevent atrophy and restore functional movements. Additionally, nutritional support to meet caloric and dietary needs may reduce the deleterious effects of the catabolic process which occurs after acute SCI.

REFERENCES