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Left laterocervical mass in a 2 months old newborn

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Case study: a 2 months old newborn, with no significant medical history, normal birth, cephalic delivery presentation, is referred by the family doctor for the ultrasonographic examination of a tumor-like neck mass. The newborn has no high temperature and appears to be in a good general condition. Laterocervical, on the left side, above the clavicle, there is a nodular, apparently not painful, firm, palpable mass, measuring approximately 1-2 cm. The head of the newborn is in a vicious position, being tilted towards the affected side, with his chin rotated and oriented on the opposite side.

Ultrasonographic findings: a fusiform, muscular structure, emerging from the sternum and left clavicle and extending oblique and upward towards the back of the ear is detected. The inferior 2/3 of the muscle are thickened (an obvious finding when the opposite, normal muscle was examined for comparison) (figure 1), the muscular structure is altered, inhomogeneous, slightly hypervascular, but with no calcifications or necrosis ar-



Fig 2. Long view of the pathologic muscle/inhomogeneous structure, with well-defined margins.

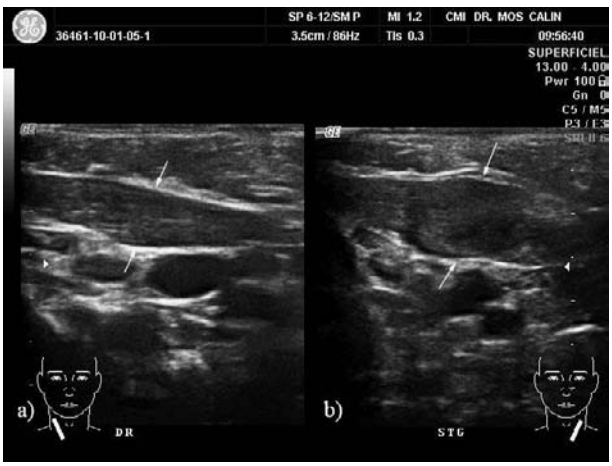


Fig 1. Long view of the normal (a) and pathologic (b) muscle. The difference between the anterior-posterior diameter of the two muscles is noticed (between the arrows).

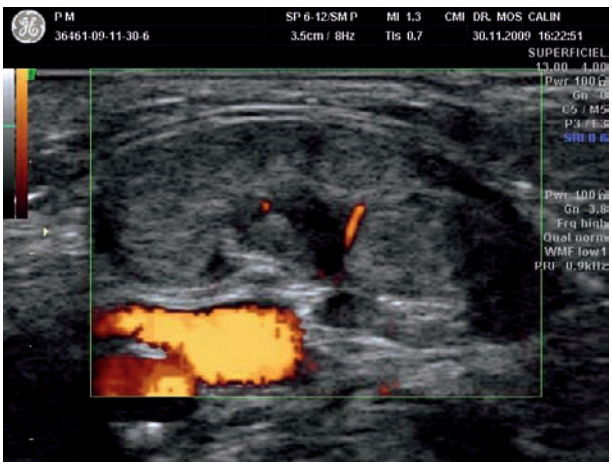


Fig 3. Transverse view and power Doppler exam of the affected muscle.

eas (figure 2, figure 3). The margins of the muscular mass are well defined and the surrounding structures show normal aspects. There were no pathological laterocervical lymph nodes present. The mass is synchronously moving with the other neck structures when the head is rotated and no signs of adherence are noticed. The same ultrasonographic aspects were found when the newborn was examined again 5 weeks later.

Questions:

1. What muscle is involved in this pathology and what is the diagnosis?
2. What are the possible ultrasonographic findings of this condition?
3. What differential diagnosis should be considered?
4. What are the treatment and the possible evolution of this condition?

Answer Quiz vol 11 no. 4

Destructive arthropathy of the shoulder in a man

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Summary of the clinical observation

A 49 year-old man with history of spinal trauma (at MRI with syringomyelia of the cervical spine) was admitted with a swollen right shoulder. Ultrasonography examination show proliferated synovia (unspecific synovitis at biopsy), multiple calcifications of the periarticular structures, tear of the rotator cuff, and irregularities of the humeral bone cortex. The bursal fluid was saguinolent, with hydroxyapatite crystals.

1. What is your diagnosis?

2. Do you find a connection between spinal injury and shoulder pathology?

The diagnosis is: **Charcot neuroarthropathy complicated with hydroxyapatite arthropathy**.

Charcot neuroarthropathy (neuropatic ostheoarthropaty) is a rare condition detailed described in 1868 by Jean-Martin Charcot as a complication of syphilis. Any condition that causes sensory or autonomic neuropathy can lead to a Charcot joint. Charcot arthropathy occurs as a complication of diabetes, syphilis, chronic alcoholism, leprosy, meningomyelocele, spinal cord injury, syringomyelia, renal dialysis, and congenital insensitivity to pain. Diabetes is considered to be the most common cause of Charcot arthropathy. It can occur at any joint; however, it occurs most commonly in the lower extremity, at the foot and ankle. Charcot arthropathy most likely results from a combination of the pathological processes: the autonomic neuropathy leads to abnormal bone formation, and the sensory neuropathy leads to an insensate joint that is susceptible to trauma. The development of abnormal bone, with no ability to protect the joint, results in gradual bone fracture and in the subluxation of the joint.

In our case syringomyelia at the cervical spine was the reason for the development of a Charcot joint in the shoulder. This rapid evolutive and destructive arthropathy was complicated in our patient with hydroxyapatite arthropathy (proved by periarticular calcifications and electronic microscopy analysis of the synovial fluid). This association was responsible of the huge swollen of the shoulder. The painless joint lesion is frequently the explanation of the delay in patient presentation (despite of the early limitation of the range of motion).