Malformation of a cervical facet joint with inclusion of an arthrolith

Case report

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The authors present the case of a 49-year-old man with an isolated malformation of the left cervical facet joint at C5–6, with secondary spondylarthrotic hypertrophy of the joint leading to involvement of the C-6 nerve root. The etiology of this cervical joint malformation is discussed.

Key Words • cervical spine • facet joint • joint malformation • spondylarthrosis • arthrolith

The significance of congenital or acquired degenerative changes of the facet joints was recognized as a result of studies by Benini,1 Kirkaldy-Willis, et al.,6 and Verbiest.10 Changes of the facet joints are not often investigated in the cervical spine because they are rare and usually not significant in this region. We describe a patient with unilateral malformation of a cervical facet joint and secondary spondylarthrotic changes leading to radicular compression.

Case Report

This 49-year-old man suffered from left-sided pain and a sensory syndrome corresponding to the C-6 dermatome. His symptoms did not improve with conservative treatment.

Examination. The patient exhibited sensory disturbances in the left C-6 dermatome and reduction of cervical spine movement. Apart from that, the clinical findings were negative. Plain x-ray films of the cervical spine showed incomplete closure of the C-6 vertebral arch, irregular hypertrophy of the left C5–6 facet joint, and an intervertebral osteochondrosis at C6–7 with anterior spondylosis (Fig. 1). Computed tomography (CT) revealed hypertrophy of the C5–6 joint on the left side with interposition of a free fragment (arthrolith) inside the joint between the disorganized and thickened superior and inferior articular facets (Fig. 2). The lateral recess and the C5–6 intervertebral foramen were narrowed by this joint.

Operation. With the patient under general anesthesia and in a sitting position, the C5–6 intervertebral segment was exposed from a dorsal midline incision using the operating microscope. After hemilaminectomy of the left C-6 vertebral arch, the left C5–6 facet joint was circumferentially dissected. The dorsal facet (superior articular process of C-6) was found to be mobile and

Fig. 1. Preoperative x-ray films, lateral view (left) and anteroposterior view (right), of the cervical spine. Note the irregular hypertrophy of the left C5–6 facet joint (arrow, right), incomplete closure of the C-6 vertebral arch, and severe spondylosis of the C6–7 segment (left).
was resected. The thickened articular capsule was folded, compressing the C-6 nerve root. After resection of the articular capsule, the nerve root was decompressed. Inside the joint a round free arthrolith, totally surrounded by a cartilaginous layer, became visible; this was also resected. By the end of the operation, the dural sac and the C-6 nerve root were well decompressed. There was no significant bulge of the C5–6 disc.

Postoperative Course. The patient was discharged 5 days after surgery. His symptoms had completely disappeared and the clinical findings were normal. A postoperative CT scan showed a good dorsal decompression of the spinal canal, the lateral recess, and the intervertebral foramen (Fig. 3). At follow-up examination 4 months later, the patient was still symptom-free.

Pathological Examination. Microscopic examination of the arthrolith showed spongy bone tissue, partly surrounded by a layer of compact bone. The arthrolith was totally surrounded by a layer of hyaline cartilage, which was partly replaced by degenerative fibrous cartilage—a sign of advanced spondylarthrosis. Microscopic examination of the resected dorsal facet also showed advanced arthrotic changes.

Discussion

Etiology

This patient had a malformation of the left cervical facet joint at C5–6, which led to an isolated unilateral spondylarthrosis with nerve root compression. To understand this malformation, the embryology of the vertebral column must be considered. The vertebral column develops from a mesenchymal origin around the notochord. In the 6th embryonic week, chondrification of this mesenchymal membrane produces the cartilaginous vertebral column. Independent of the vertebral bodies, each half of the neural arch is chondrified from a center at the base of the articular and transverse processes. These processes extend ventrally into the pedicles to meet and blend with the cartilaginous vertebral bodies, and dorsally into the laminae. The articular processes, like the transverse processes, are chondrified in continuity with the neural arches. The blastemas migrate into the mesenchymal interdorsal
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membranes, meet with the corresponding articular processes, and form real diarthroses. Intervening zones of mesenchyma that do not become cartilage mark the sites of the intervertebral joints; synovial cavities appear in these later.

In the embryonic phase, an inclusion is sometimes found between the occasional anomalous articular facets, filling the gaps between the facets. The inclusion may develop into the articular menisoid fold, consisting partly of connective tissue and partly of cartilaginous tissue. Due to the complex development of the facet joints, various malformations have been described in this area. To our knowledge, the malformation in our patient has not been published previously.

We propose the following possibilities for the development of the joint malformation described here. First, it could result from a growth disturbance of the articular processes at the embryonic phase; a rather large mesenchymal inclusion in the gaping articular cavity developed, was separated from the articular capsule, became ossified, and formed an isolated arthroolith. The theory of apophyseal embryonic growth disturbance is supported by the incomplete closure of the C-6 vertebral arch in our patient. Second, in addition to the perichondrial ossification of the vertebral arches and their processes between the 10th and 14th year of spinal development, accessory centers of apophyseal ossification form the tips of the vertebral processes. If an apophyseal ossification center does not fuse with the basal articular process, the tip of the process can be divided and forms an accessory intra-articular ossicle. Free articular ossicles of this kind have been described in the lumbar spine, but not in the cervical spine.

Clinical Significance

Because of the malformation of the facet joint, a progressive spondylarthrosis of this joint occurs, so that later the spinal canal and the nerve root canal are narrowed and clinical symptoms develop. In contrast to the lumbar spine, where hypertrophy of the facets is common, isolated spondylarthrotic hypertrophy of the cervical spine facet joints is rare because, with disc degeneration, the uncinate processes come into contact with the upper vertebral bodies before increased strain is placed on the facet joints. Thus, the interaction between degeneration of the discs and facet joints is not as distinct as in the “three-joint complex” of the lumbar spine outlined by Kirkaldy-Willis, et al. Denaro and Junghans' pointed out, however, that severe cervical spondylarthrosis can occur with or without disc degeneration.

Diagnosis and Treatment

In general, each cervical spine syndrome is diagnosed on plain x-ray films and axial CT scans. It is necessary not only to examine the intervertebral discs but also to seek alterations of the facet joints. Cervical myelography or magnetic resonance imaging can also help to estimate the extent to which the spinal cord and the nerve roots are affected or compressed. If the patient's clinical symptoms cannot be treated conservatively, microneurosurgical resection of the malformed joint is required to decompress the affected nerve root.

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References