Atlantoaxial laxity in rheumatoid arthritis

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We found that 14 of 162 rheumatoid arthritis patients with chronic occipitocervical pain had anterior atlantoaxial instability in the absence of any corresponding radiographic changes in the joint cartilage or subchondral bone. Our findings suggest that ligamentous instability is a prerequisite for this type of change. At the time of the detection of the instability, the median duration of disease was 12 (6–28) years. Rheumatoid occipitocervical pain may be initially caused by facet-joint arthritis or inflammation in the ligaments, and at a later stage also by irritation of the C2 nerve roots.

Anterior atlantoaxial instability is the most common rheumatoid condition in the cervical spine. The prevalence of this condition is still unknown. We have studied whether ligamentous laxity and soft tissue involvement alone, in the absence of changes in the joint cartilage and subchondral bone, can lead to atlantoaxial instability in patients with rheumatoid arthritis.

Patients and methods

Cervical spine radiographs were taken of 162 patients treated for rheumatoid arthritis with total knee or hip replacement arthroplasty at the Rheumatism Foundation. The examination included lateral extension-flexion projections and an open-mouth anteroposterior projection (Figures 1 and 2). Anterior atlantoaxial instability was observed in 96 (59%) of all such patients. Fifty-eight (36%) had a vertical atlantoaxial subluxation according to Redlund-Johnell's (1984) method. Radiographic evaluation was done using measurements given in detail elsewhere (Santavirta et al. 1987).

We found 14 patients with recognizable anterior atlantoaxial instability with a minimum distance of 3 mm between the bone contours, but who, at the time, did not show any radiographic signs of erosion of the atlas or epistropheus (Table 1). All 14 patients were women. Their median age at the onset of the rheumatoid disease was 32 (22–64) years. When the anterior atlantoaxial instability was first recognized, the median duration of the rheumatoid disease was 12 (6–28) years. One patient had seronegative and the others seropositive rheumatoid polyarthritis. All 14 had erosion of peripheral joints, 12 of them in their hands. At the time of the diagnosis, the patients' joint score averaged 15 on a scale of 0 for normal and 24 for the worst possible condition (Kaarela 1985). All 14 patients had severe chronic occipitocervical pain. This pain is typically located in the occipital area and in the upper part of the neck corresponding to the sensory innervation area of the C2 nerve root. This pain is typically resistant to treatment with analgetics.

All the patients had used nonsteroidal anti-inflammatory medication for longer than 3 years, 12 patients had also taken slow-acting disease modifying drugs for longer than 3 years, and 11 patients had been on prednisone treatment for more than 3 years. One patient was being treated with azothioprine.

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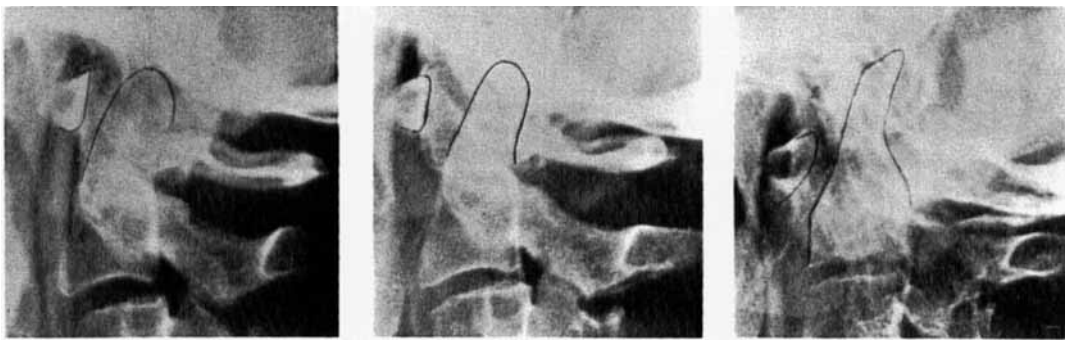
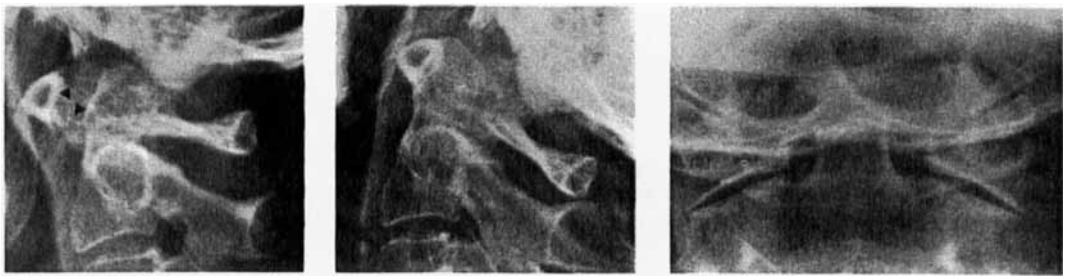


A 4-mm anterior instability after 24 years of disease. There are no signs of erosion of the dens or atlantoaxial facet joints.

Three years later, an 8-mm anterior instability with beginning of vertical subluxation; the tip of the dens protrudes 2 mm above the foramen magnum level.

Ten years after the initial radiograph, there is major vertical suhbluxation; the dens is 16 mm above the foramen level. Anterior instability is typically reduced (4 mm) as the vertical subluxation progresses.

Figure 1. A 46-year-old woman with seropositive rheumatoid arthritis and atlantoaxial instability. Lateral views in flexion.



A 9-mm anterior atlantoaxial instability (arrows) in flexion.

Instability reduced in extension.

Open-mouth anterior-posterior view shows normal atlantoaxial facet joints and dens.

Figure 2. A 53-year-old woman with a 19-year history of seropositive rheumatoid polyarthritis.

Table 1. 14 patients with anterior atlantoaxial subluxation (AAS) without any changes in the joint cartilage or subchondral bone in the occipito-atlantoaxial area

Case	Situation at the time of diagnosis of AAS					Maximum AAS	Situation at the last follow-up		
	A	B	C	D	E	(mm)	D	E	F
1	46	24	69	4	−2	11	4	+16	4
2	75	11	51	6	−3	6	6	−3	0
3	44	9	69	5	−9	13	9	−1	3
4	60	22	65	6	−8	7	7	−4	2
5	49	19	94	7	−5	7	7	−5	0
6	52	8	16	8	−5	7	7	−5	0
7	49	17	20	10	−6	12	12	−6	2
8	45	12	40	3	−7	5	5	−7	1
9	54	9	66	10	−5	13	11	+5	4
10	35	12	47	5	0	8	6	+4	1
11	34	6	22	6	−7	6	6	−7	0
12	59	28	48	10	−5	10	10	−5	1
13	57	20	65	10	−5	10	10	−5	0
14	41	10	62	3	−7	13	13	−7	0

A age.
B duration of rheumatoid disease.
C ESR (mm/h).
D AAS (mm).
E location of the tip of the dens in relation to the foramen.
F Grades of the changes in the atlantoaxial facet joints.

Results

Diagnosis of atlantoaxial instability

At the time of initial diagnosis, the median amplitude of the anterior atlantoaxial instability was 6 (3–10) mm (Table 1). The tip of the dens was located on an average 5 (0–9) mm below the level of the foramen magnum. Criticism can be raised against this method used in the assessment of upward dislocation (Redlund-Johnell 1984). Three patients showed minor subaxial (below C2) subluxations.

After 6 (1–20) years, the median amplitude of the anterior atlantoaxial instability was 8 (4–13) mm or 2–3 mm larger than at the time of first diagnosis. The dens had migrated upwards by an average of 3.2 mm.

Eight patients had developed atlantoaxial facet-joint arthrosis and five patients had erosion of the dens. The anterior atlantoaxial instability in 2 patients was later fused (Gallie 1939).

Discussion

The synovial joint between the anterior arch of the atlas and the dens and the synovia-lined bursa behind the dens and in front of the transverse ligament are focuses of inflammation that cause destruction of the transverse, alar, and apical ligaments. Synovitis around the dens can cause compression of the medulla, which can disappear after posterior occipitocervical fusion (Larsson et al. 1989). The ligaments themselves may be targets for mononuclear cell infiltration (Konttinen et al. 1987), which gradually leads to weakening and rupture of the collagen fibers and degradation of the connective tissue matrix. This in turn gradually causes ligamentous laxity and rupture. The condition may be treated with a collar (Althoff et al. 1980) or with an operation (Brattström 1981, Santavirta et al. 1988b, Slätis et al. 1989). Atlantoaxial subluxation can also occur in noninflammatory conditions, such as Down's syndrome, which is characterized by a generalized ligamentous laxity (Collacot 1987).

Instability of the cervical spine in rheumatoid arthritis represents a potential danger under general anesthesia. A preoperative radiographic examination of the cervical spine is recommended to detect the atlantoaxial instability in patients with severe long-term rheumatoid polyarthritis. The affection of the cervical spine may occasionally occur very early in the disease (Konttinen et al. 1987). However, in the present series the median interval between the onset of the rheumatoid disease and the first diagnosis of anterior atlantoaxial instability was 12 years. The majority of patients had ulnar deviation and swan-neck deformities of the fingers as diagnostic signs, which can be connected with peripheral ligamentous laxity and a tendency to subluxation. Severe occipitocervical pain, peripheral joint erosions, and laxity in patients with chronic rheumatoid disease seem to be indicative signs of possible anterior atlantoaxial instability.

The transverse ligament is the main stabilizer of the atlantoaxial joint during extension-flexion. After transecting the transverse ligament, instability of up to 5 mm may occur between the anterior arch of the atlas and the dens (Bland 1974). The apical ligament performs a minor stabilizing function, and the alar ligaments normally restrict the extent of the rotation. If both the transverse ligament and these secondary ligaments are eliminated, the anterior atlantoaxial instability can exceed 5 mm (Bland 1974). Subluxation or instability of other joints may be seen due to infiltration of ligaments without severe affection of the cartilage. Plain radiographs give very unsatisfactory information about the status of the cartilage and the soft tissues in the joints. Autopsy studies of the cervical spine may be necessary to substantiate the conclusions of the present study. Also new advances in diagnostic radiography may be used to strengthen our findings. The atlantooccipital area is well visualized on CT scanning (Raskin et al. 1983, Kaufman et al. 1983, Castor et al. 1983, Redlund-Johnell 1984). CT scanning is for practical reasons done in one position and due to technical reasons it is difficult to perform in extension-flexion projections (Laasonen et al. 1985; Toolanen et al. 1986). Actually, neurologic findings show a poor correlation with CT findings (Kaufman et al. 1983). Magnetic resonance imaging has the additional advantage of an improved soft-tissue contrast unobtainable with other imaging methods (Modic et al. 1983, Breedveld et al. 1987, Pettersson et al. 1988, Larsson et al. 1989).

The present study suggests that ligamentous laxity without involvement of the joint cartilage or subchondral bone may lead to anterior atlantoaxial instability in inflammatory rheumatic diseases. Pain may be caused by chronic ligamentous inflammation and is not necessarily a C2 nerve headPache (Santavirta et al. 1986).

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