High Prevalence of Thoracic Vertebral Deformities and Discal Wedging in Ankylosing Spondylitis Patients with Hyperkyphosis

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ABSTRACT. ***Objective***. To study the prevalence of deformities of vertebrae and intervertebral discs in patients with ankylosing spondylitis (AS) in relation to fixed hyperkyphosis of the spine.

Methods. Altogether 50 patients (15 women, 35 men) with AS were studied. Hyperkyphosis was measured by the occiput to wall distance (OWD). Anterior (Ha), mid- (Hm), and posterior height (Hp) of the vertebrae and intervertebral discs were measured on lateral radiographs of the thoracic (Th5–Th12) and lumbar spine (L1–L5). Vertebral shapes were analyzed according to McCloskey, *et al*. Wedging of discs was calculated as Ha/Hp. Hyperkyphosis was defined as OWD $\geq$ 1 cm.

Results. In the thoracic spine, the prevalence of vertebral deformities was higher in patients with hyperkyphosis (n = 38) compared to patients without hyperkyphosis (n = 12) (45% vs 8%; $p = 0.01$). The prevalence of thoracic vertebral deformities in patients with hyperkyphosis differed little between men and women (39% vs 58%; $p > 0.10$) and among patients above and below the age of 45 years (50% vs 33%; $p > 0.10$). Patients with one or more deformed thoracic vertebrae had a higher mean OWD than patients without deformed vertebrae (12 ± 7 vs 7 ± 6 cm; $p < 0.01$). The total sum of deformities of the thoracic vertebrae and discs explained 43% of the variance of the age adjusted OWD ($p < 0.001$). Deformities of lumbar vertebrae and discs did not contribute to hyperkyphosis.

Conclusion. In patients with AS and hyperkyphosis, deformities of the thoracic vertebrae occur frequently and, together with wedging of the thoracic discs, contribute significantly to fixed hyperkyphosis of the spine. (J Rheumatol 2001;28:1856–61)

Key Indexing Terms:
ANKYLOSING SPONDYLITIS — VERTEBRAL DEFORMITY — DISC NARROWING
OSTEOPOROSIS — KYPHOSIS — OCCIPUT TO WALL DISTANCE

The inability to fully stand straight due to fixed hyperkyphosis of the thoracic spine is one of the potential clinical complications in ankylosing spondylitis (AS)[1-3]. The degree of hyperkyphosis is strongly related to the Stoke Ankylosing Spondylitis Spine Score, a score of radiographic damage in AS[4]. The relation of hyperkyphosis with vertebral deformities is unclear[1,5-11]. In studies reporting vertebral fractures complicated with neurological deficit, a low prevalence of vertebral deformities was found[1,6,7]. Others, using radiologists' reports[8] or morphometric evaluation[9-11], found a prevalence of 10 to 17%. The occiput to wall distance (OWD) was higher in patients with vertebral wedging compared to patients without wedged vertebrae[9,10]. However, no data are available on the prevalence of vertebral deformities in AS patients with and without hyperkyphosis. Further, the contribution of discal wedging has not been reported until now. The anatomical changes resulting in fixed hyperkyphosis in AS are thus still poorly documented and poorly understood. The clinical repercussions of vertebral deformities and discal wedging are related to the ankylosing nature of the disease[4]. In AS, wedging of vertebrae and discs results in irreversible hyperkyphosis, in contrast to postmenopausal osteoporosis, in which hyperkyphosis due to vertebral deformities can be compensated by mobility in the unaffected and mobile segments of the spine.

We compared the prevalence of deformities of the vertebrae in 38 patients with AS with hyperkyphosis to 12 patients with AS without hyperkyphosis.

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Submitted August 22, 2000; revision accepted February 15, 2001.

MATERIALS AND METHODS

A total of 50 patients with AS were studied, 12 without hyperkyphosis (OWD of 0 cm) and 38 with hyperkyphosis (OWD ≥ 1 cm, range 1–26 cm). Only patients with available radiographs of the thoracic or lumbar spine and of the sacroiliac joints were included. They were consecutively selected from daily rheumatology practice (n = 30) (PG and JV). An additional 20 patients were selected from the OASIS database to increase the number of patients with an OWD > 10 cm (n = 20)[12]. They all met the modified New York criteria for AS[13]. OWD was measured with the patient standing with heels and, if possible, the back against the wall, with the distance measured in cm from the occiput to the wall during maximal effort to touch the head to the wall, without raising the chin above its usual carrying level[14].

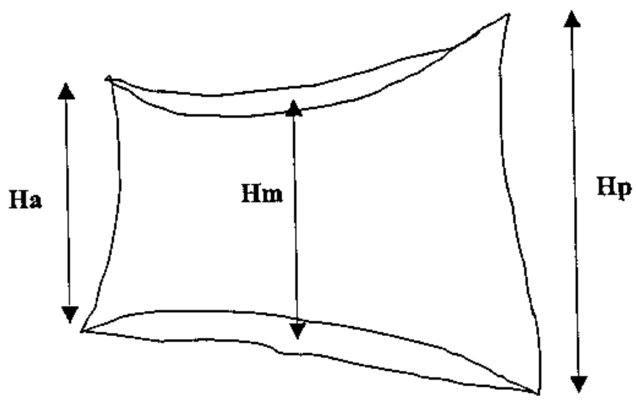
Radiographs were analyzed by one of us (JV) without knowledge of the OWD. The thoracic and lumbar spine were evaluated for the presence of syndesmophytes and squaring. Regarding height measures, anterior (Ha), mid- (Hm), and posterior height (Hp) of the vertebrae and the Ha and Hp of the discs were measured on lateral radiographs of the thoracic (Th5 to Th12) and lumbar spine (L1 to L5), using a standard caliper[15] (Figure 1). The reference points for the anterior height were taken at the crossing of the endplates with the anterior site of the vertebra. Thoracic vertebrae proximal to the level of Th5 were not measurable due to superposition of the shoulders. In some patients the vertebrae in the region Th11–L1 were not measurable due to soft tissue superposition. In 40 patients at least 6 thoracic and 4 lumbar vertebrae were measurable. In the remaining 10 patients, 5 thoracic or 3 lumbar vertebrae were measurable. The degree of vertebral deformation was evaluated according to McCloskey, *et al*[16]. Coefficient of variation for repeated measurements of Ha/Hp and Hm/Hp (2 repeated measurements on 10 radiographs) was < 2%. As fixed hyperkyphosis is the result of wedging of all vertebrae and discs, the sum of deformities of the vertebrae and wedging of the discs was also correlated with the OWD.

Statistics. Significance of differences between groups was calculated by analysis of variance and by chi-square test. The relation between OWD and the sum of deformities of vertebrae and wedging of discs was analyzed using multiple regression after adjustment for age. Statistical analysis was performed using SPSS, version 8.

RESULTS

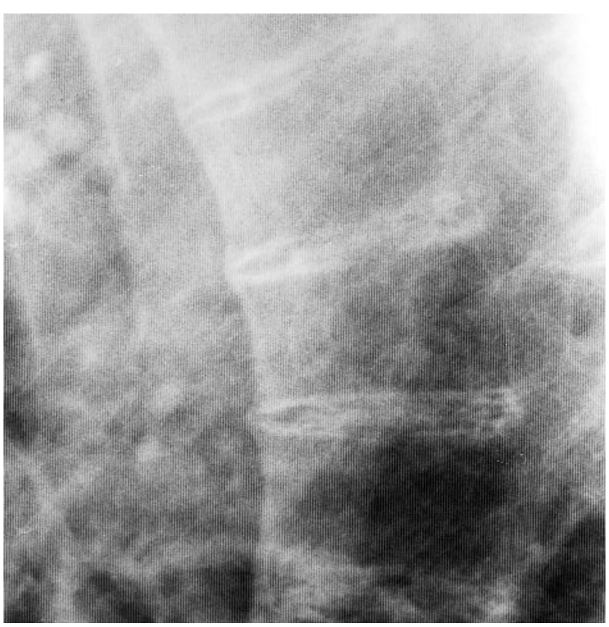
Characteristics of the patients are shown in Table 1. Patients with hyperkyphosis were older compared to those without hyperkyphosis, reflecting longer disease duration.

Thoracic spine (n = 50). In the thoracic spine, the prevalence of vertebral deformities was 8% in patients without hyperkyphosis (Table 2) and 45% in patients with hyper-



A

Figure 1. A-C. Measurement of anterior height (Ha), mid-height (Hm), and posterior height (Hp) on radiographs of vertebrae, and examples in 2 patients [standard deviations (SD) according to McCloskey[16]]. A. Measurement of Ha, Hm, and Hp on lateral radiographs of vertebrae. B. Vertebra Th9: Ha/Hp = 0.75 (–3 SD). C. Vertebra Th11: Ha/Hp = 0.65 (–4.6 SD); Hm/Hp = 0.73 (–4.4 SD).



B

C

Table 1.. Age, sex, and clinical characteristics of the patients.

	All	No Hyperkyphosis (OWD = 0 cm)	Hyperkyphosis (OWD ≥ 1 cm)	Hyperkyphosis (OWD ≥ 10 cm)
Number	50	12	38	27
Age, yrs*	50 ± 12 (30–81)	42 ± 12 (30–71)	52 ± 11 (32–81)	53 ± 11 (34–81)
Female/male (n)	15/35	3/9	12/26	8/19
OWD, cm*	8 ± 7 (0–26)	0	11 ± 6 (1–26)	14 ± 5 (10–26)
Schober index, cm*	3 ± 1 (1–5)	4 ± 1 (3–5)	3 ± 1 (1–4)	2 ± 1 (1–4)
Thoracic expansion, cm*	6 ± 3 (2–12)	8 ± 3 (4–12)	5 ± 1 (2–7)	5 ± 1 (4–7)

*Mean ± SD (range).
OWD: occiput to wall distance.

Table 2. Number (%) of patients with deformed vertebrae according to localization and occiput to wall distance (OWD).

	Thoracic Spine Th6–Th12	Lumbar Spine L1–L5	Total Spine Th6–L5
No hyperkyphosis (OWD 0 cm)	1/12(8)	1/9(11)	1/9(11)
Hyperkyphosis			
OWD ≥ 1 cm	17/38(45)*	4/30(13)	16/30(53)**
OWD ≥ 10 cm	13/27(48)*	4/20(20)	12/20(60)**

*p < 0.05, **p ≤ 0.01 vs OWD 0 cm.

kyphosis (chi-squared p = 0.01). In patients with hyperkyphosis the prevalence of thoracic vertebral deformities differed little between men and women (58% and 39%; chi-square p > 0.10) and in patients older than 45 years compared to those younger than 45 years (50% and 33%; chi-square p >0.10) (Figure 2).

Patients with one or more deformed thoracic vertebrae (n = 18) had a higher mean OWD than patients without deformed vertebrae (12 vs 7 cm, respectively; p < 0.01). The mean OWD was significantly related to the number of deformed thoracic vertebrae. It ranged from 7 cm for those without deformities to 13 cm for those who had 2 or more deformed thoracic vertebrae (p < 0.05 for trend). When only anterior wedging was considered (Ha/Hp < 0.85, n = 20), the OWD ranged from 7 to 18 cm (p < 0.01 for trend), both in women and men (Figure 3). The relative risk for OWD ≥ 1 cm according to the presence of one or more vertebral deformities in the thoracic spine was 8.9 (95% confidence interval, CI, 3.0, 26.6), and for OWD ≥ 10 cm was 10.2 (95% CI 3.4, 31.1). OWD correlated significantly with the total sum of Ha/Hp of the thoracic vertebrae (r = 0.501, p < 0.001) (Figure 4).

Lumbar spine (n = 39). The prevalence of lumbar vertebral deformities was roughly similar among patients with and without hyperkyphosis (13% vs 11%; p > 0.10). In patients with hyperkyphosis no clear differences were found in vertebral deformities between men and women (11% and 18%; p > 0.10) and between patients younger and older than 45 years (17% and 8%; p > 0.10).

Thoracic and lumbar spine combined (n = 39). In the combined thoracic and lumbar spine, the prevalence of

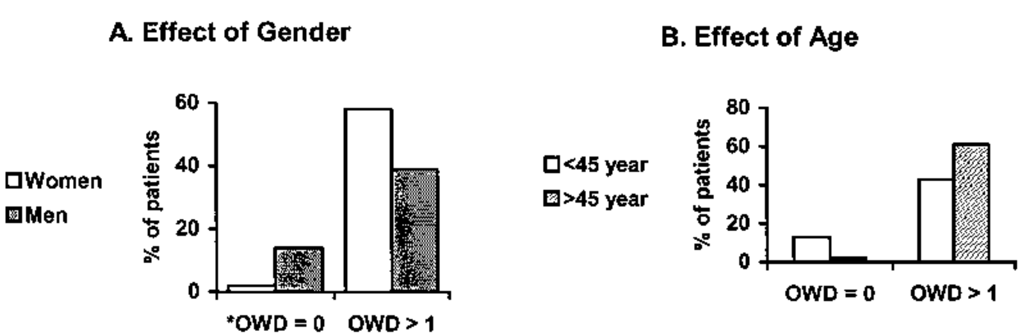


Figure 2. Prevalence of thoracic vertebral deformities according to the presence of hyperkyphosis and gender (A) or age (B). OWD: occiput to wall distance.

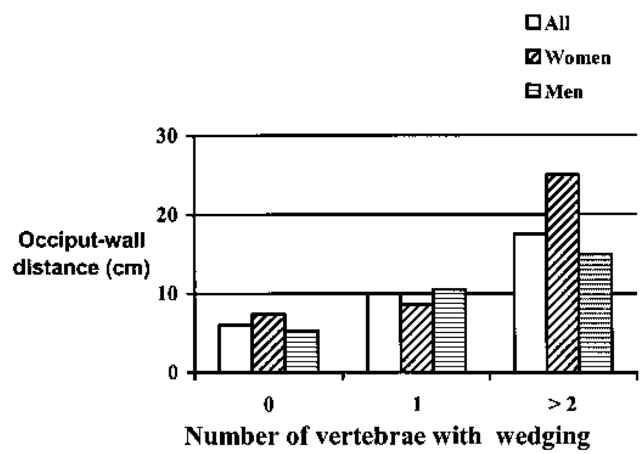


Figure 3. OWD according to the number of wedged vertebrae (Ha/Hp < 0.85) between Th6 and Th12 ($p < 0.01$ for trend) in all patients and in men and women.

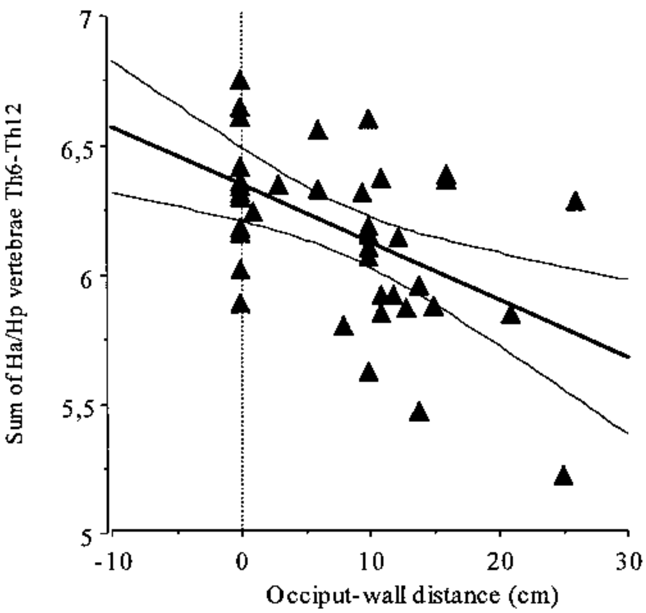


Figure 4. Regression of OWD with the sum of Ha/Hp of the thoracic vertebrae ($r = 0.501$, $p < 0.001$).

vertebral deformities was significantly higher in patients with hyperkyphosis compared to patients without hyperkyphosis (53% vs 11%; $p = 0.01$) (Table 2). In patients with OWD > 10 cm, the prevalence was 60% ($p < 0.01$ vs no hyperkyphosis).

Intervertebral discs. In patients with hyperkyphosis, 62 discs were more wedged than the lower limit of wedging in patients without hyperkyphosis (42 in the thoracic spine, 20 in the lumbar spine). OWD was significantly increased in patients with one or more discs showing a more pronounced wedging than the lower limit of wedging in patients without hyperkyphosis (10 vs 6 cm; $p < 0.05$). No correlation was found between the number of vertebral deformities and the number of discs that were more wedged than the lower limit of wedging in patients without hyperkyphosis ($r = 0.183$, $p > 0.30$).

The total sum of deformities of the thoracic vertebrae and wedging of the thoracic discs explained 43% of the age adjusted OWD ($r = 0.657$, $p < 0.001$). Summing total deformities of the lumbar vertebrae and wedging of the lumbar discs did not increase this variance.

No neurological involvement was documented in any patient.

DISCUSSION

The anatomical changes associated with fixed hyperkyphosis in AS are still poorly understood. This study indicates that the degree of fixed hyperkyphosis of the spine in AS is correlated with a combination of deformations of the vertebrae and, to a lesser degree, of wedging of the intervertebral discs in the thoracic spine that has not yet been reported.

Thoracic vertebral deformities were found in 45% of patients with an occiput to wall distance (OWD) of 1 cm. This prevalence was much higher than in patients without hyperkyphosis (mean age 42 yrs), in whom the prevalence was similar to the normal prevalence in the Western European population between 50 and 54 years of age (11%)[15,16]. The prevalence of vertebral deformities in patients with hyperkyphosis is higher in AS patients younger than 50 years (32%) compared to the normal population, indicating that deformities occur early in the disease process. A prevalence of 17% of vertebral deformities has been documented in mild AS, using analysis according to McCloskey[11]. In that study, however, no differentiation according to the degree of OWD was reported.

The prevalence of vertebral deformities differed little between men and women for all methods of analysis, indicating that in this respect factors related to AS seem more important than differences in sex. The prevalence of thoracic vertebral deformities increased significantly with age, reflecting that age and duration of disease contribute to the risk for vertebral deformities, a finding also reported by others[8-10].

In addition to vertebral deformities, wedging of the intervertebral discs contributes independently to fixed hyperkyphosis. This was reflected in the high prevalence of wedged discs in patients with hyperkyphosis compared to those without hyperkyphosis, especially at the thoracic spine. Further, the association with OWD remained after correction for age and vertebral deformities.

Our results indicate that both discal and vertebral wedging in the thoracic spine are involved in the pathogenesis of hyperkyphosis. This raises questions about the pathophysiology and prevention of hyperkyphosis. In several studies on AS it has been shown that bone resorption is increased[17,18] and that bone loss occurs early in patients with active disease[17-23]. Insofar as osteoporosis is involved in the development of deformed vertebrae, the prevention of bone loss should be a target for therapy. To what degree disc narrowing is the result of inflammation and/or insufficient effect of physiotherapy, exercise, or analgesic drug therapy is unclear. The absence of correlation between vertebral and disc narrowing indicates that different pathophysiological processes might be involved in the occurrence of vertebral and discal deformities.

In several reports neurological complications have been reported as a rare consequence of vertebral fractures[24-28]. In our rather small group of patients, no neurological deficits were found. This indicates that the clinical consequences of vertebral deformities are mainly the repercussion on the hyperkyphosis and the resulting restrictions in function.

The measurements of vertebral deformities were performed in a way similar to studies on postmenopausal osteoporosis, in which they are validated and compared with other measurements[29]. Therefore, the measurements provide a valid approach to defining vertebral deformities. Interestingly, in postmenopausal osteoporosis, intervertebral disc heights are well preserved[30], in contrast to the wedging we found in many AS patients. This is presumably related to differences in pathophysiology of osteoporosis between postmenopausal osteoporosis as a bone disease and AS, with inflammation as a possible contributor to bone loss and vertebral deformities.

This study has several shortcomings. First, it was cross sectional. Clearly, longitudinal studies are necessary to assess to which degree wedging of the discs and vertebrae is pre-existing, and at what rate it progresses in the course of the disease. Second, detailed analysis of changes in vertebral structure and discs in the whole spine is necessary, including the cervico-thoracic and thoraco-lumbar transitions that are usually not easily accessible by plain radiographs. Further, mobility should be measured separately in the cervical, thoracic, and lumbar spine to determine the clinical repercussions of deformities of vertebrae and discs. Third, it remains to be determined which would be the best method for evaluating changes in bone density in AS in relation to the development of wedging of vertebrae[31]. Indeed, squaring and syndesmophytes are frequent findings in our patients that interfere with classical measurements of bone density in the spine, such as dual energy absorptiometry[11,17–23].

We conclude that in patients with AS with fixed hyperkyphosis, vertebral deformities occur frequently and, together with discal wedging, contribute significantly to fixed and irreversible hyperkyphosis of the spine. Further investigations are needed to fully understand the anatomical changes in the whole spine that contribute to hyperkyphosis. Those studies should include the cervico-thoracic and thoraco-lumbar transitions. These findings might open new perspectives for treatment of AS, which should not only aim at prevention of inflammation, but also at prevention of the associated vertebral deformities and discal wedging.

ACKNOWLEDGMENT

The authors thank J. Mertens and H. Leroi for their devoted administrative support.

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