factors as hypogonadism, chromosomal alterations (mosaicism) and Short stature HOMEBOx (SHOX)-related haploinsufficiency disorders can affect bone mass and geometry in TS girls. Recent studies have confirmed an intrinsic oestrogen-independent defect of bone mass and geometry mainly in skeletal sites with a higher content of cortical bone, such as the femoral neck and long bones. Conversely, it was observed that TS patients receiving appropriate oestrogen treatment usually showed normal trabecular BMD after adjusting for body size. In addition to Hormonal Replacement Therapy (HRT), Growth Hormone (GH) administration is considered standard treatment for TS girls, but the role of GH in promoting bone accrual has not yet been completely defined.

Several studies reported in TS patients an increase in fracture prevalence and risk that was higher during childhood and after the age of 45. Fractures occurred mainly in skeletal sites consisting predominantly of cortical bone, often after a high or medium force trauma. However, the most recently published guidelines for the care of girls and women with TS do not recommend BMD assessment during childhood and/or adolescence. It must be stressed that there is, as yet, no definition of when the defect in bone mass acquisition starts and whether early recognition could improve bone health. Quantitative ultrasound (QUS) is a densitometric technique of particular interest during paediatric age because it is easy to perform, non-invasive and radiation-free. At present, data on bone mineral status in TS girls assessed by QUS are lacking.

The aim of this study was to evaluate bone mineral status in adolescent girls with TS by two different densitometric techniques, DXA and phalangeal QUS, and to assess whether a reduced bone status was related to the history of fracture.

MATERIALS AND METHODS

Patients

Twenty-four Caucasian Italian girls with TS aged 17.1±3.1 years (range 8.7-19.9) were referred as outpatients to the Division of Paediatric Endocrinology of the University of Pisa, Italy. The diagnosis of TS was made by karyotyping (45X n=13; deletion n=3; mosaic n=8). Clinical details are summarized in Table 1.

| Table 1. Clinical findings in TS girls and in controls |
| Turner syndrome girls | n | Mean ± SD (range) |
| Age at diagnosis, years | 24 | 8.9 ± 3.1 (0.1-14.1) |
| Age at start of GH therapy, years | 24 | 9.3 ± 2.9 (2.0-14.2) |
| Age at start of HRT, years | 21 | 13.4 ± 0.8 (12.0-15.0) |
| Duration of GH therapy, years | 24* | 6.9 ± 2.8 (2.4-12.0) |
| Duration of HRT, years | 21 | 4.6 ± 1.9 (0.9-7.2) |
| Stature, Z-score | 24 | -1.8 ± 0.8 (-3.3;-0.3) |
| BMI, Z-score | 24 | 0.9 ± 0.9 (-0.6;3.2) |

* 15 girls had stopped GH therapy from 1.4 ± 0.7 years (range 0.1-2.5)
GH: growth hormone; HRT: hormone replacement therapy; BMI: body mass index.

None of the patients was affected by any other disease known to be associated with abnormal bone mineral status. Two patients, affected by autoimmune thyroiditis, had normal blood levels of thyroid hormones. There were no contraindications to patients taking part in non-competitive sport activities. Human recombinant GH was administered at a dosage of 0.330-0.375 mg/Kg/week. Pubertal development was induced in 21 patients with conjugate oestrogens at a dosage of 0.330-0.375 mg/Kg/week. Pubertal development was induced in 21 patients with conjugate oestrogens at a dosage of 0.3 mg daily for 6 months, followed by 0.625 mg daily. From the second year, cyclic HRT was started (conjugate oestrogens 0.625 mg daily for 21 days, adding progesterone for 10 days/cycle). The remaining three girls were too young to induce puberty.

Study design

In all the girls we evaluated lumbar and femoral BMD by DXA and phalangeal amplitude-dependent speed of sound (AD-SoS) and bone transmission time (BTT) by QUS to assess bone mineral status.

Lumbar and femoral BMD of patients were compared with appropriate age-reference values for the girls, which were obtained with the same DXA apparatus and software. Phalangeal AD-SoS and BTT results were compared with our own reference data for QUS.

Standing height and body weight of all the patients were measured with a wall-mounted stadiometer and a mechanical balance. In each patient, both length and weight were the mean of three measurements. Body Mass Index (BMI) was calculated using the formula weight (Kg)/height (m)². Height, weight and