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Dilation of ascending aorta in Turner syndrome - short-term follow-up

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Background

Aortic dissection causes excess mortality in Turner syndrome. Cross-sectional studies show increased prevalence of aortic dilatation, the principal surrogate risk marker for the aortic event. Increased risk has been linked with congenital cardiovascular abnormalities, karyotype, and blood pressure

Purpose

To study the previously uninvestigated natural cause of the aortopathy in Turner syndrome over time, and possible predictors of increasing aortic size

Methods

Prospective follow-up study comparing Turner syndrome (N = 102, examined twice) with healthy age-matched female controls (*N* = 65, examined once). Non-contrast, respiratory-navigated, ECG-gated, 3D SSFP MRI analyzed for thoracic aortic dimensions at eight positions. Bland-Altman reproducibility testing determined the limits of agreement. Echocardiography assessed aortic valve morphology. Ambulatory blood pressures were performed

Results

Of 102 women enrolled at baseline, 11 were lost to follow-up, and 80 had technically successful magnetic resonance imaging scans at both baseline and follow-up. Aortic diameters were comparable in Turner syndrome (baseline) and controls, except from the distal aortic arch and the aortic isthmus (classical site of coarctation) (Table 1). The prevalence of aortic dilation was significantly higher in Turner syndrome both before and after indexing for body surface area. In Turner syndrome, bicuspid aortic valve, aortic coarctation, and elongated transverse aortic arch were present in 28%, 11%, and 38%. 45, × karyotype was seen in 61%. Blood pressures and heart rates were significantly elevated in Turner syndrome; 23% took antihypertensive medication. The mean follow-up time was 2.4 ± 0.4 years (range: 1.4 to 3.5 years). The sinutubular (P = 0.02) and mid-ascending (P = 0.009) aortic diameters increased with dilatation rates of 0.16 and 0.23 mm/year, respectively. The remaining thorax aortic diameters showed no statistically significant changes. None of the currently identified factors of risk for aortic dilatation or dissection predicted the increase. Overall, twenty-two of 80 (30%) Turner syndrome patients had an aortic diameter increment above the limits of agreement, ranging from 0.7 to 2.7 mm/year (highest for the mid-ascending aorta). No congenital anomaly or karyotype was more prevalent in this group, where daytime and 24-hour heart rates, but not blood pressure, were significantly higher (P < 0.03). Antihypertensive treatment did not predict reduced increases in aortic diameter over time

Table 1: Maximum aortic diameter (mm) in Turner syndrome at inclusion and after an average follow-up time of 2.4 years compared with age-matched healthy controls (examined once).

	Turner syndrome (*N* = 80)			*P*† (Turner syndrome at baseline versus follow-up)	Controls (*N* = 65)	*P*‡ (Controls versus Turner syndrome at baseline)
	Baseline	Follow-up	Change during follow-up			
Sinotubular junction	25.3 ± 4.3	25.7 ± 4.0	0.4 (0.1;0.7)	0.02	25.6 ± 2.7	0.7
Ascending aorta	27.5 ± 5.0	28.0 ± 5.1	0.6 (0.2;0.9)	0.009	26.5 ± 3.4	0.2
Distal ascending aorta	25.3 ± 3.6	25.2 ± 3.6	-0.1 (-0.3;0.2)	0.8	25.3 ± 2.9	0.9
Proximal aortic arch	23.4 ± 3.6	23.4 ± 3.4	0.1 (-0.1;0.4)	0.4	24.1 ± 2.8	0.2
Distal aortic arch	20.5 ± 2.7	20.5 ± 2.6	0.1 (-0.1;0.3)	0.5	22.8 ± 2.4	<0.0001
Aortic isthmus	19.3 ± 2.3	19.4 ± 2.3	0.1 (-0.1;0.3)	0.3	21.4 ± 2.3	<0.0001
Descending aorta	19.5 ± 2.8	19.4 ± 2.7	-0.05 (-0.2;0.1)	0.6	19.7 ± 2.3	0.7
Distal descending aorta	18.2 ± 2.2	18.1 ± 2.2	-0.1 (-0.2;0.1)	0.2	18.3 ± 2.3	0.7

†: Paired Student's t-test, equal variances assumed
‡: Independent Students t-test, equal variances assumed

Conclusion

An ascending aortopathy in adult Turner syndrome was indicated by a general increment of sinutubular and mid-ascending aortic size. Furthermore, a seemingly accelerated dilatory aortopathy was evident in a third of the cohort where the previously indices of risk did not clearly predict the increase in aortic size.