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Elastic Properties of the Aorta Are Affected in Young Patients with Sleep Bruxism

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ABSTRACT

Background and aim: This study aimed to evaluate the effects of sleep bruxism (SB) on aortic elastic properties, which predict cardiovascular morbidity and mortality.

Materials and methods: Twenty-eight patients (25 female, 3 male) with SB admitted to maxillofacial surgery outpatient clinic were enrolled in the study. The control group consisted of age and sex-matched 44 volunteers (9 female, 5 male) with no Bruxism complaints and normal orofacial examination findings. The two groups were compared in terms of aortic stiffness as measured by pulse wave velocity (PWV), aortic strain, and aortic distensibility.

Results: Aortic strain and aortic distensibility were statistically significantly decreased in SB patients compared to the control group (p=0,006 and p=0,028 respectively), while PWV was increased in SB patients compared to the control group with a limited statistical significance (p=0.064). Correlation analysis revealed statistically significant positive associations between PWV and CRP level (r=0.281, p=0.017) and negative associations between aortic strain and distensibility with CRP level (r=-0.347, p=0.003 and r=-0.277, p=0.019, respectively). The diastolic function parameters, E/A, E/é mean, and E wave deceleration time, were within normal limits in SB patients, and we did not observe statistically significant differences among diastolic function parameters between two groups.

Conclusion: Our study illustrated that aortic strain and aortic distensibility were decreased while PWV was increased in patients with SB. Further studies are required to further elucidate cardiovascular system manifestations in SB patients.

1. Introduction

Bruxism is a movement disorder characterized by involuntary and undesirable rhythmic contractions of the masticatory muscles. If it occurs while awake, it is called awake Bruxism, and if it occurs during sleep, it is called sleep bruxism (SB).[1] SB patients usually complain of tooth grinding, gnashing, and jaw clenching. Excessive daytime sleepiness, headache, jaw pain, facial pain, abnormal tooth wear, and tinnitus are symptoms of SB. With wide variations in definition and diagnosis criteria used in the associated studies, the approximate prevalence of SB in the adult population is reported at about 13%. At the same time, Bruxism, in general, is a more common condition with a reported prevalence of up to 31,4%. SB is more common in the younger population, and its prevalence decreases with age such that it is reported only 3% among people over 60 years old.[1] Although there are various speculations about the pathophysiology of SB, it is known that individuals with depression, anxiety, and emotional stress are more likely to develop SB.[2] Stressful conditions may cause both bruxism and cardiovascular diseases. SB has been shown to develop secondary to microarousals during sleep, and the microarousals are associated with an increase in heart rate and muscle tone, blood pressure, and cortisol levels.[3] Also, SB is associated with blood pressure fluctuations during sleep.[4] Chronic stress is generally accompanied by increased sympathetic activity. Results show elevated catecholamine levels in the urine of SB patients and show the relation between SB and autonomic dysregulation and increased sympathetic activity.[5, 6] The role of the sympathetic drive over vascular aging and arterial stiffness is recently identified.[7]

Aortic stiffness, a negative indicator of aortic elasticity, is the main determinant of subclinical atherosclerosis and a major predictor of future cardiovascular events and all-cause mortality.[8, 9] Owing to their elastic properties, the aorta and proximal large arteries push forward some amount of blood contained in them to the peripheral circulation during diastole. Thus they provide continuity of the peripheral blood flow. As the aorta stiffens, it loses the ability to absorb the pressure generated by cardiac contraction, and the pressure is transmitted to smaller vessels which contribute to the pathogenesis of end-organ damage in hypertension. Aortic stiffness is usually

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assessed noninvasively by measuring the pulse wave velocity (PWV) along the aorta.[10] The aortic strain, which is assessed through volume changes and aortic distensibility, calculated by the ratio of volume changes to pulse pressure, is commonly used to assess aortic compliance.[8] SB is a common health condition beyond assumptions, and the data about its relation to the cardiovascular system is very limited. SB's increased sympathetic activity and muscle tone may predispose these patients to vascular changes earlier than expected. In this study, we aimed to define the elastic properties of the aorta in SB patients.

2. Materials and methods

It is a case-control study carried out between January and June 2019. Twenty-eight patients admitted to maxillofacial surgery outpatient clinic with complaints of Bruxism and diagnosed as SB was assigned as a patient group. Age and sex-matched 44 patients with no Bruxism complaints and normal orofacial examination findings were assigned as healthy control groups. Exclusion criteria of the study were specified as refusal to provide informed consent, diagnosis of moderate to severe congenital or acquired valvular heart disease, bicuspid aortic valve, coronary artery disease, cardiac arrhythmias, hypertension, renal failure, diabetes, and use of medications for these reasons. Inadequate echocardiographic windows and structural heart disease revealed by echo were also excluded. Smoking status was defined as positive only for current smokers. All procedures were carried out in accordance with the responsible committee's ethical standards and the Declaration of Helsinki's 2008 revision. The study was approved by a local clinical research and ethics committee (approval number and date resp; 2019/3-1, 16.04.2019) and all participants provided written informed consent.

Physical examination

Patients referred with SB complaints were evaluated by taking comprehensive anamnesis, orofacial evaluation, and echocardiography. SB was diagnosed based on the medical histories and clinical criteria of the patients. Patients who ground and clenched their teeth at least five times per week for six months were diagnosed with SB. If possible, the patient's bedtime friend or family members were asked about the presence of grinding and clenching sounds while sleeping. Signs of masseter muscular hypertrophy and palpation evoked masseter pain, tooth wears, and glare in the dental restoration were checked during orofacial examination by an experienced orofacial surgeon for both patient and control groups. The participants' height and weight were measured, and their body mass indexes (BMI) were calculated by dividing their weight in kilograms by their height in meters squared. Brachial blood pressure measurements were taken twice using an automated device (Omron 705 IT electronic blood pressure monitor), and the averages were calculated. After at least 10 minutes of rest, measurements were performed while the participant sat in a recumbent position. The difference between systolic and diastolic blood pressures (SBP and DBP) was used to calculate peripheral pulse pressure (PP).

Biochemical analysis

Following an 8-hour fast, each patient had an 8- to 12-ml fasting blood sample obtained through the superficial veins of the forearm. Blood samples were centrifuged at 3000 rpm for 10 minutes to separate sera. The tests performed were complete blood count (CBC), thyroid-stimulating hormone (TSH), fasting blood glucose, serum electrolytes, urea, creatinine, and lipid panel.

Echocardiography and doppler

Echocardiographic and Doppler studies were performed using Philips EPIQ 7 device (Philips Healthcare, Andover, MA, USA) equipped with 2.5 MHz and 3.5 MHz transducers. Echocardiographic measurements were made following the European Society of Cardiovascular Imaging protocols.[11] A minimum of three cardiac cycles were recorded for each analysis. 2D images from the parasternal long-axis view at the level of mitral leaflet tips were used to measure LV end-systolic and end-diastolic dimensions and wall thicknesses. LV mass index was calculated by using the formula:

$$(0.8\{1.04[([LVEDD + IVSd + PWd]^3 - LVEDD^3)]\} + 0.6) / \sqrt{\frac{(\text{height (cm)} \times \text{weight(kg)}}{3600}}$$

LVEDD, IVSd, and PWD are the end-diastolic thicknesses of the left ventricle, interventricular septum, and posterior wall. USING A MODIFIED SIMPSON'S RULE, the LV ejection fraction was calculated using 2D images from the apical four- and two-chamber views. Pulsed wave (PW) Doppler and PW tissue Doppler imaging (TDI) applied in an apical four-chamber view were used to evaluate diastolic function. By placing a 2 to 5 mm Doppler sample volume between the tips of the mitral leaflets, the early (E) and late (A) transmitral flow velocities, the E/A ratio, and the E deceleration time (EDT) were measured. With PW TDI and a low pass filter, the early diastolic velocity (e') of the mitral annulus was measured at the lateral border of the mitral annulus. E/e' was calculated from the E and e.'

The measurements of ascending aorta were made from the parasternal long-axis view. For optimal visualization of ascending aorta, a little counter-clockwise rotation of the echo probe and moving it to a higher intercostal space was applied. Systolic and diastolic diameters of ascending aorta were obtained at a level 3 cm above the aortic valve by using the leading-edge to leading technique. The following formulas were used to calculate aortic strain (AS) and aortic distensibility (AD)Using the mean of the three measurements for each parameter:

$$AS\ (\%) = 100 \times ((\text{systolic diameter of ascending aorta})^2 - (\text{diastolic diameter of ascending aorta})^2) / (\text{diastolic diameter of ascending aorta})^2$$

$$AD\ (10^{-3} \times mmHg^{-1}) = 10 \times AS / \text{pulse pressure}$$

The PWV was measured as previously described 12 by using the formula PWV=ΔL/ΔT, where ΔL is the distance between the distal aortic arch and left distal external iliac artery, ΔT is the time it takes for the arterial pulse to pass between these two sites. The PW Doppler sample volume's axial length was set at 3 to 5 mm, and the low-velocity filter was reduced to highlight the waveform's beginning. We recorded ten consecutive waveforms at a sweep speed of 100 mm/sec, first in the distal aortic arch and the left distal external iliac artery. The distal aortic arch was visualized from the SSN (suprasternal notch) long-axis view, while the patient was positioned supine and the head was tilted backward. Doppler flow images of the external iliac artery were taken by placing the probe over the left inguinal crease in the longitudinal plane, with the patient in the supine position. The difference between the time from the R wave to the onset of the EIA Doppler waveform and the time from the R wave to the distal aortic arch Doppler waveform was used to calculate the transition time. The analysis was based on the average of ten consecutive cycles.

The distance between the two Doppler recording points was measured with a tape measure on the body surface. The distance from the suprasternal notch to the umbilicus and the distance from the umbilicus to the left femoral pulse was added, and the sample volume depth between the suprasternal notch and the distal aortic arch was subtracted from the summation. PWV was calculated by dividing the transition time by the distance. The PWV of each participant was calculated using the arithmetic mean of ten consecutive cardiac cycles at a regular heart rate.

Statistics

IBM Statistical Package for the Social Sciences (SPSS) v.22 was used for statistical analysis. While analyzing data, numbers, percentages, mean, and standard deviation values were used as descriptive statistics. Data were tested for normality using a histogram and the Kolmogorov-Smirnov test. The Student's T-Test or analysis of variance (ANOVA) was used to compare normally distributed continuous data between the two groups. The Mann-Whitney U-test was used To analyze non-normally distributed data. The Chi-square test was used to analyze the qualitative variables statistically. Pearson's correlation analysis was used to determine the relationship between Aortic stiffness parameters and CRP levels. At the level of $p \leqslant 0.05$, statistical significance was assumed.

3. Results

Table 1 lists the general characteristics of the participants. Female patients made up the majority of the patients (64 female vs. 8 male). The two groups were similar means of age. All subjects were normotensive, nonobese (body mass index [BMI] <30 kg/m^2), and free of diagnosis of any chronic diseases. Only CRP was higher in the SB group compared to the control (p=0.045) among laboratory measures.

Table 1. Comparison of the general characteristics of Bruxism (+) and control subjects.

Variables	Bruxism (+) (n=28)	Control (n=44)	p
Age(years)	27.68 ± 8.899	29.55 ± 6.811	0.318*
Female (n / (%))	25 / (89.3%)	37 / (84.1%)	0.534‡
Height (cm)	164.82 ± 5.221	166.27 ± 5.892	0.291*
Weight (kg)	60.79 ± 9.130	64.27 ± 7.666	0.085*
BMI (kg/m^2)	22.3807 ± 0.22886	23.1936 ± 1.84739	0.178*
Smoker (n / (%))	4 / (14.3%)	9 / (20.5%)	0.507‡
SBP (mm Hg)	115.89 ± 11.200	113.64 ± 10.205	0.382*
DBP (mm Hg)	74.79 ± 7.244	71.91 ± 6.361	0.081*
PP (mm Hg)	41.11 ± 7.099	41.73 ± 7.164	0.720*
Heart rate (bpm)	74.75 ± 10.790	72.89 ± 9.211	0.436*
Fasting Blood Glucose (mg/dL)	93.86 ± 10.295	97.16 ± 10.982	0.207*
Total Cholesterol (mg/dL)	188.96 ± 26.898	176.14 ± 32.810	0.088*
Triglyceride (mg/dL)	146.75 ± 50.061	139.07 ± 53.339	0.544*
LDL (mg/dL)	106.97 ± 22.622	100.10 ± 29.401	0.296*
HDL (mg/dL)	52.643 ± 9.8214	48.227 ± 10.0784	0.071*
Creatinine (mg/dL)	0.8179 ± 0.15586	0.8355 ± 0.22153	0.715*
TSH (mIU/L)	1.375 (0.200-4.800)	1.678 (0.340-6.430)	0.304†
Calcium (mg/dL)	9.3750 ± 0.49712	9.4341 ± 0.55487	0.648*
CRP (mg/dL)	0.616 ± 0.484	0.402± 0.397	0.045*

BMI: body mass index; CRP: C-reactive protein; DBP: diastolic blood pressure; HDL: high-density lipoprotein; LDL: low-density lipoprotein; PP: pulse pressure; SBP: systolic blood pressure; TSH: Thyroid-stimulating hormone. ‡; analyzed by qi-square test.

*; In cases where the data were normally distributed, the mean ± SD were calculated and analyzed using the student's t-test.

†; In cases where the data was not normally distributed, median (minimum-maximum) values were presented and analyzed using the Mann-Whitney U test.

Echocardiographic and aortic stiffness parameters of SB and control subjects are compared in table 2. Although PWV measurements were higher in SB patients, this difference was statistically limited (p=0.064). Aortic strain and Aortic distensibility were statistically significantly lower in SB patients (p=0,006 and p=0,028, respectively) (Fig. 1). Of the diastolic function parameters, E/A, E/é mean, and EDT were all within normal limits in SB patients, and there was no statistically significant difference among diastolic function parameters, just like among all other echocardiographic measurements.

Table 2. Comparison of the echocardiographic and aortic stiffness parameters between Bruxism (+) and control subjects.

Variables	Bruxism (+) (n=28)	Control (n=44)	p
LVEF (%)	61.25 ± 5.454	61.87 ± 5.484	0.640 *
LA (mm)	3.175 ± 0.229	3.107 ± 0.243	0.240 *
IVSd (mm)	0.80 (0.60-1.10)	0.90 (0.60-1.00)	0.080†
PWd (mm)	0.80 (0.50-1.00)	0.80 (0.70-1.00)	0.261†
LVEDD (mm)	4.507 ± 0.252	4.439 ± 0.244	0.256 *
LVESD (mm)	3.096 ± 0.238	3.066 ± 0.184	0.543 *
LVMI (g/m^2)	71.947 ± 15.564	72.565 ± 12.475	0.853 *
E (m/s)	88.21 ± 20.92	94.32 ± 18.73	0.202 *
A (m/s)	63.39 ± 13.34	69.55 ± 15.24	0.084 *
E/A	1.424 ± 0.369	1.378 ± 0.218	0.511 *
é septal (m/s)	14.61 ± 2.11	15.14 ± 1.91	0.276 *
é lateral (m/s)	10.57 ± 1.45	11.32 ± 1.64	0.053 *
é mean (m/s)	12.59 ± 1.68	13.23 ± 1.57	0.107 *
E/é mean	6.96 (3.81-10.91)	7.12 (4.55-9.17)	0.844†
EDT (ms)	196.107 ± 25.701	198.614 ± 29.566	0.714†
Systolic AA diameter (cm)	2.818 ± 0.251	2.898 ± 0.158	0.101 *
Diastolic AA diameter (cm)	2.550 ± 0.219	2.566 ± 0.161	0.724 *
T1-Ao (ms)	102.714 ± 11.932	104.364 ± 10.033	0.530†
T2-E iliac (ms)	170.571 ± 14.444	175.136 ± 11.953	0.150†
ΔT (ms)	67.86 ± 8.627	70.77 ± 5.660	0.087 *
Aorto-iliac distance (cm)	46.29 ± 1.652	46.55 ± 1.910	0.556 *
Aortic Strain (%)	22.349 ± 8.396	27.888 ± 7.747	0.006 *
Distensibility (10−3 x mmHg-1) at AA level	5.60 ± 2.43	7.05 ± 2.80	0.028 *
PWV (m/s)	6.923 ± 0.884	6.612 ± 0.518	0.064 *

A: late transmitral flow velocity; AA: Ascending Aorta; E: early transmitral flow velocity; é: mitral annular early diastolic velocity; EDT: E wave deceleration time; IVSd: interventricular septum thickness; LA: left atrium diameter; LVEDD: left ventricular end-diastolic diameter; LVESD: left ventricular end-systolic diameter; LVEF: left ventricular ejection fraction; LVMI: left ventricle mass index; PWD: posterior wall thickness; PWV: pulse wave velocity; T1-Ao: time from the R wave to the onset of Doppler waveform on distal aortic arch; T2-E iliac: time from the R wave to the onset of Doppler waveform on external iliac artery; ΔT: (T2-E iliac)-(T1-Ao).

*; In cases where the data were normally distributed, the mean ± SD were calculated and analyzed using the student's t-test.

†; In cases where the data was not normally distributed, median (minimum-maximum) values were presented and analyzed using the Mann-Whitney U test.

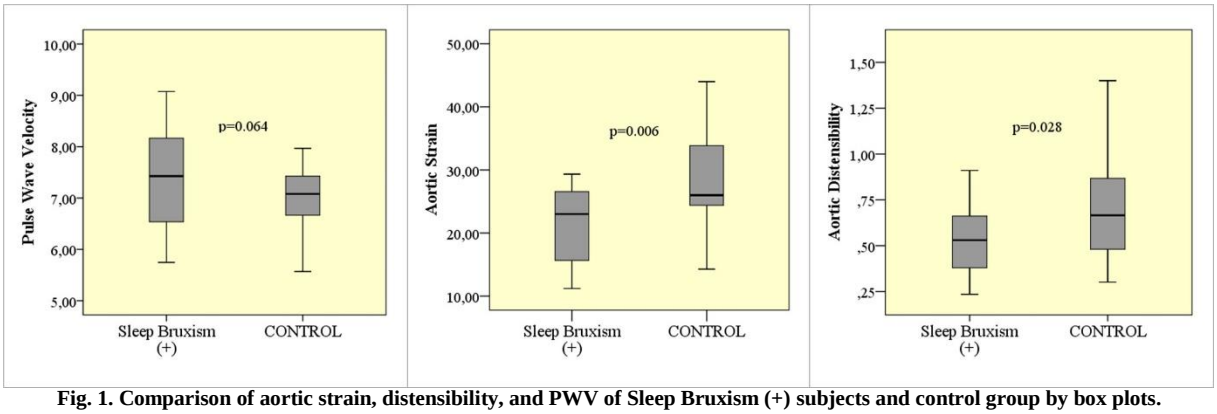

Fig. 1. Comparison of aortic strain, distensibility, and PWV of Sleep Bruxism (+) subjects and control group by box plots.

As shown in table 3 and Fig. 2, correlation analysis revealed statistically significant positive associations between PWV and CRP level (r=0.281, p=0.017) and statistically significant negative associations between aortic strain and distensibility with CRP level (r=-0.347, p=0.003 and r=-0.277, p=0.019, respectively).

Table 3. Pearson's correlation coefficient and p-value (lower value) for the linear correlation between the aortic stiffness indices and CRP level.

Variables	r value	p
Aortic Strain (%)	-0.347	**0.003**
Distensibility (10−3 x mmHg-1) at AA level	-0.277	**0.019**
PWV	0.281	**0.017**

CRP, C-reactive protein; PWV: pulse wave velocity; r value: Pearson's correlation coefficient.
CRP: C reactive protein; PWV: pulse wave velocity.

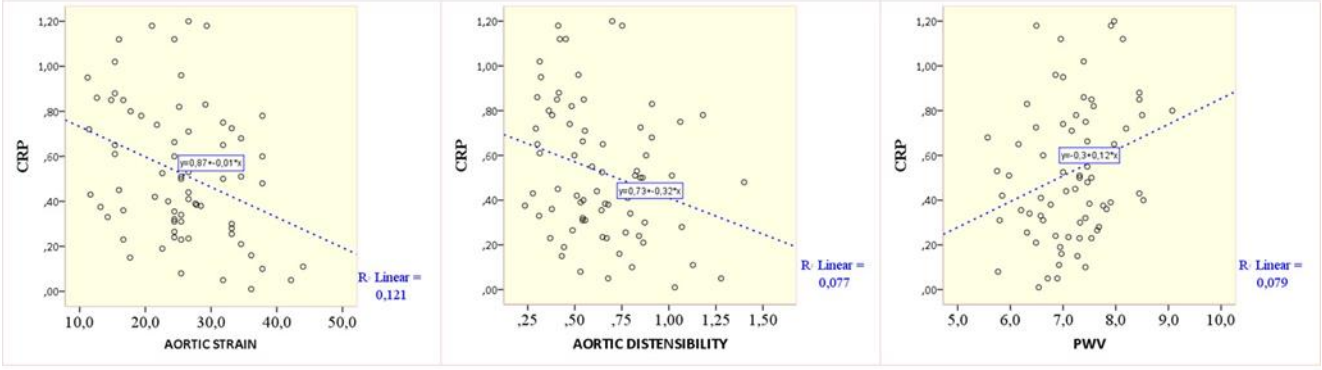

Fig. 2. Correlation of aortic stiffness indices with CRP level by Scatterplot (bi-variate) analysis.

4. Discussion

The elastic properties of the aorta in patients with SB were evaluated in the current study. When comparing SB patients to the control group, we found that aortic strain and aortic distensibility were decreased while PWV was increased. However, the decrease in strain and distensibility were much more statistically significant than the increase in PWV as the indicative of the aortic elasticity, aortic distensibility, strain, and stiffness are deemed predictive for subclinical atherosclerosis, future cardiovascular events, and all-cause mortality.[9, 13] Therefore, our results may depict an increased risk of atherosclerosis and cardiovascular events in SB patients who do not have a verified diagnosis of any CVD. Both emotional and oxidative stress have been revealed to be increased in the presence of SB, and both play an important role in the etio-pathophysiology of SB.[14-16] Moreover, metabolic and hormonal disorders related to inflammation and stress have been reported in SB patients in the literature.[16, 17] Autonomic dysregulation and increased sympathetic activity are also related to SB.[6, 18] In addition to sympathetic overdrive and impaired cardiac autonomic nerve innervation, jaw clenching and accompanying hypertonic contractions of the masseter and temporal muscles might contribute to the increased afterload, resulting in increased aortic wall smooth muscle size, i.e., the wall hypertrophy. Park S. et al.[19] have clarified the importance of inflammation on the pathogenesis of aortic stiffness. In line with previous reports, we have found significant correlations between CRP and aortic stiffness parameters. CRP is an inflammatory marker utilized to ascertain the intensity of chronic low-grade systemic inflammation and is noted as an independent predictor of adverse cardiovascular events.[20] Additionally, cumulative exposure to CRP is associated with decreased arterial stiffness even if the inflammation was limited.[21] We think that both

stress and inflammation, which are approved to be contributing and triggering factors for the decrement in the elasticity of the aorta, might be featured in the explanation of our results in SB patients. Bruxism has been defined as working as a kind of an eternal motion machine, as augmenting symptoms arising from the abnormal functioning of the organism increase perception of being stressed, and consequently resulting in an increased muscle tone and teeth clenching.[22] In addition to stress and inflammation, increased skeletal muscle tone in SB could eventually give rise to a loss of elasticity in the aorta. It has been shown that prolonged exposure to resistance exercise leads to an increase in the content of smooth muscle cells in the vascular wall and changes in the load-bearing characteristics of elastin and collagen.[23] Besides, elevated afterload might stiffen the aorta by amplifying vascular resistance due to increased skeletal muscle tone in SB. It would be a plausible pathophysiologic pathway that might contribute to elucidating the mechanism of arterial stiffness increase in our results. One of the most extensively used methods of measuring is arterial stiffness in aortic PWV. Even though these methods are considered indirect arterial stiffness measurements, a recent consensus identified PWV as the best available technique for assessing arterial stiffness. PWV can be measured using both invasive and non-invasive procedures.[24] Compared with invasive measurements, Doppler measurements are compatible with invasive measurements and reproducible. Styczynski G previously et al.[12] used the method we used in calculating PWV in this study, and the results are compatible with invasive measurements done with catheterization. Also, several studies have found that increased PWV predicts cardiovascular outcomes independently of blood pressure, including all-cause mortality in the general population and risk for stroke, coronary artery disease, and heart failure.[13, 25, 26] In the control group, we excluded features that might affect elastic parameters in PWV evaluation and included age- and gender-matched individuals strictly. As a result, the difference between the study and control groups may be attributed to the presence of SB. These outcomes strengthen the evidence of macrovascular involvement in SB patients. Great arteries, mainly the aorta, are rich in elastic fibres that ensure blood flow continuity to the periphery in the diastole. While the capacity of an artery to expand in systole shows its compliance, on the contrary, its failure to expand indicates its stiffness. Aging, various traumas, and inflammation commonly engender deterioration of the elastin/collagen balance in the vessel wall in favour of collagen, resulting in a decline in vascular elasticity. Besides, thickening of the smooth muscle layers, increased fragmentation of elastin, an increment in the amount of collagen, and calcification in the arterial media give rise to an increase in arterial stiffness, diminished compliance, and finally, the aorta becomes dilated. Consequently, these age-associated pathophysiological changes, independent of atherosclerosis, are represented in clinical practice as decreased aortic distensibility and strain and increased PWV. However, Aortic arch distensibility in younger people and aortic arch pulse wave velocity in older individuals were the best markers of subclinical large artery stiffness compared to other aortic function measures.[8] The average age of our patient population is substantially lower than that of previous studies. It may explain the marked decrease in aortic strain and distensibility while our research slightly increases aortic stiffness. On the other hand, we believe that our PWV readings are more reliable since the measurement errors are due to pathologies of the abdominal aorta (e.g., tortuosity, aortoiliac occlusion, or aneurysm) are expected to be less in such a young population. Numerous studies in the literature indicate a significant relationship between decreased arterial elasticity, i.e., increased stiffness and cardiovascular morbidity, yet we have come across few articles investigating the consequences of SB on the cardiovascular system. Nashed et al.[4] noted that SB and blood pressure fluctuations during sleep were associated. According to them, individuals with chronic stress are prone to increase sympathetic activity via adrenocortical and adrenomedullary responses, which might explain the relationship between hypertension and SB. In another report,[27] carotid intima-media wall thickness predicting preclinical atherosclerosis is increased in SB patients. Though it is inconvenient to make a head-to-head comparison, in the course of SB, some mutual pathways might be affected in reduced aortic elasticity sharing similar pathophysiological mechanisms with all the pathologies mentioned above. We have not observed diastolic dysfunction in SB though it has been recognized that there has been a positive association between aortic stiffness and diastolic dysfunction. it might be because it is too early to detect any diastolic dysfunction in a young study population.[28, 29] Attenuation of the elastic properties of the aorta influences end-systolic cardiac wall stress secondary to increased afterload, and subsequently, a relative reduction in coronary perfusion promotes LV remodeling and delayed LV relaxation.[30, 31] Also, alterations in sympathetic cardiac innervation have been associated with diastolic dysfunction.[32] Thus, increased aortic stiffness might result in diastolic dysfunction. Nonetheless, for the subjects included in this study, the time required to develop diastolic dysfunction due to increased aortic stiffness might not have been attained yet. Moreover, Komnenova et al.[33] have reported that withdrawal of the responsible factor had reversed aortic stiffness in rats within three weeks, independent of diastolic dysfunction. In this respect, needing further studies, prevention of diastolic dysfunction would be another incentive to treat bruxism punctually.

Limitations

During patient selection, we followed up-to-date diagnosis criteria for SB, including questionnaires and oral examinations that are practical and frequently used. Studies with SB patients of whom the diagnosis was confirmed with polysomnography would be of extra value. The relatively small number of the study population was another weakness.

5. Conclusion

In this study, we depicted a tendency to decline in the elasticity of the aorta in young individuals with SB. More comprehensive randomized studies are needed for definitive judgment. Since increased arterial stiffness is a predictor and risk factor for many cardiovascular pathologies, especially hypertension, SB is a clinical entity that should not be neglected in terms of cardiovascular system involvement. We present an adjunctive reason why individuals with this complaint are treated properly on time.

Conflict of Interest

The authors declared that there is no conflict of interest.

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