Sabah Ahmed Hussein[1], **Khaled Mahmoud Kamel**[1], **Safy Zahid Kaddah**[1],
Emad Efat Abd El-Hamid[2], **Marwa Moawad Shaban**[1]

[1]Chest Department, Faculty of Medicine, Cairo University, Egypt
[2]Shebin Elkom Chest Hospital, Egypt

Role of ultrasonography in assessment of anatomic upper airway changes in patients with obstructive sleep apnea

Abstract

Introduction: Obstructive sleep apnea is a common disorder, characterized by recurrent narrowing and closure of the upper airway accompanied by intermittent oxyhemoglobin desaturation and sympathetic activation. Ultrasound imaging of the airways has advantages of being safe, quick, repeatable, portable and widely available. Airway ultrasound can visualize and assess the mouth and tongue, oropharynx, hypopharynx, epiglottis, larynx, vocal cords, cricothyroid membrane, cricoid cartilage, trachea, and cervical esophagus.

Material and methods: This study assessed the role of ultrasonography in detecting the level and degree of obstruction of airway passages in patients with obstructive sleep apnea (OSA) and its relation to OSA severity. It included thirty-three patients diagnosed as OSA, and ten healthy subjects as a control group. All participants were $\geq$ 18 years and were subjected to full medical history, Epworth sleepiness score (ESS), thorough clinical examination, complete overnight polysomnography and neck ultrasonography.

Results: Ultrasonography findings showed a statistically significant increase in lateral parapharyngeal wall thickness (LPWT) (P < 0.001) and a significant increase in distance between lingual arteries (DLA) (P < 0.01) among OSA patients. Moreover, there was a significant statistical decrease in the retropalatal pharynx transverse diameter (RPD) (P < 0.05) in the OSA group compared to those without OSA. LPWT and DLA are parameters that can be used to predict the severity of OSA. Combination of LPWT and RPD can achieve a 100% sensitivity and specificity.

Conclusions: Ultrasound is more objective and convenient than the questionnaire because it doesn't require overnight time consumption. It is also more relevant than pulse oximetry for examining pharyngeal airspace. Also, this study demonstrated that submental ultrasonography is sufficiently sensitive for differentiating OSA severity.

Key words: obstructive sleep apnea, neck ultrasonography, upper airway anatomy

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Introduction

Obstructive sleep apnea is a common disorder, characterized by recurrent narrowing and closure of the upper airway accompanied by intermittent oxyhemoglobin desaturation and sympathetic activation. Diagnosis of OSA can be indicated by symptomatology and the presence of known risk factors such as increasing age, obesity and large neck circumference, although OSA can occur in individuals with none of these risk factors [1]. A number of tools and methods are available for the assessment of sleep health as self-reported questionnaire instruments. However, the gold standard for diagnosis of OSA is the overnight polysomnography [2].

Due to convenience, inexpensiveness, no radiation exposure and office-based procedure, an increasing number of physicians uses ultrasonography (US) to examine the neck. Several studies also demonstrate the role of US in detecting anatomic risk factors for OSA, including parapharyngeal pad and the tongue [3]. Airway ultrasound can visualize and assess the mouth

and tongue, oropharynx, hypopharynx, epiglottis, larynx, vocal cords, cricothyroid membrane, cricoid cartilage, trachea and cervical esophagus. Neck ultrasound can measure coronal mid-tongue base thickness (CTBT), sagittal mid-tongue base thickness (STBT), retropalatal pharynx transverse diameter (RPD), distance between lingual arteries (DLA), lateral parapharyngeal wall thickness (LPWT). It can detect both the anatomic characteristics, the dynamic changes of pharyngeal airspace and discriminate OSA severity [4]. Different treatment options are now available for effective management of OSA. Positive airway pressure (PAP) provides pneumatic splinting of the upper airway and is effective in reducing apnea–hypopnea index (AHI). PAP may be delivered in continuous (CPAP), bi-level (Bi-PAP), or auto-titrating (APAP) modes [5].

This study investigated the role of ultrasonography in detection of the level and degree of obstruction of the airway passages in patients with OSA and its relation to OSA severity.

Material and methods

This is an analytical case/control study that was performed as a collaborative work between the sleep unit and chest ultrasonography unit, Chest Department, Faculty of Medicine, Cairo University, during the period between January 2017 and October 2018. The study included thirty-three patients diagnosed as OSA and ten healthy subjects as a control group. All patients met the following eligibility criteria: physician confirmed diagnosis of OSA, age ≥ 18 years, and on a regular medication regimen for OSA. All persons with tracheostomy, evident underlying cause of any sleep disorders such as alcohol intake or endocrinal disorders such as hypothyroidism or acromegaly, patients with abnormalities in the soft palate or upper airway and those with underlying heart disease (e.g., organic valvular heart diseases, dysrhythmias, cardiac tamponade and pericardial effusion) were excluded from the study. The study was ethically approved by the institutional research ethics committee. Informed consent was obtained from all participants. All subjects underwent detailed clinical evaluation, including history taking, clinical examination, Epworth sleepiness score, Electrocardiogram, echocardiography. Complete overnight polysomnography was performed over a whole night (8-hour sleep). The study started at 10 P.M. until 6 A.M. using SOMNOscreen™ plus Polysomnography with digital IR-Videometry (Germany).

Data obtained included:
— Apnea: complete cessation of airflow breathing at the nostrils and mouth for at least 10 seconds or longer.
— Hypopnea: decrease in rate and depth of breathing by 50% for 10 seconds or longer.
— Apnea-hypopnea index (AHI): average number of apnea and hypopnea per hour of sleep. Persons with AHI < 5 are not considered to have OSA. In contrast, AHI ≥ 5 and < 15, AHI ≥ 15 and < 30, and AHI ≥ 30 are classified as mild, moderate, and severe, respectively [6].
— Other measured variables are total sleep time, sleep efficiency, sleep stage percentage, sleep stage latency, arousals, respiratory disturbance index (RDI), snoring, body position, oxyhemoglobin saturation, limb movements and arrhythmias.
— The system records sleep stages by electroencephalography (EEG), electrooculography (EOG) and electromyography (EMG). EEG was used to monitor sleep stages and identify sleep latency and arousal. EOG was applied to monitor both horizontal and vertical eye movements to document the onset of REM and slow rolling movements accompanying the onset of sleep. EMG records atonia during REM or lack of atonia in REM-related parasomnia.

Neck ultrasonography was done using Hitachi EUB-7000 in the ultrasound unit, Chest Department, Kasr Al-Ainy Hospital. All cases were examined by B-mode and Doppler scan with curvilinear transducer (2–5 MHz). Ultrasound examination was done on the day after overnight polysomnography. All sonographic measurements were made by the same well-trained operator in ultrasonography who was blinded to the polysomnographic data. The study subjects laid supine on the examination couch. The neck of the patient was slightly extended with the infraorbital meatal baseline (the line joining infraorbital margin and the ear tragus) perpendicular to the scanning table.

Measurements obtained included:
— Retropalatal pharynx transverse diameter (RPD): ultrasonography scanning from the hyoid bone to the external auditory meatus at the level of oral pharynx, then the probe is tilted downward to locate the retropalatal pharynx which is defined as air column at the highest plane near the uvula. RPD is determined by the outer margin of the air column (RPD) (Figure 1A).
— Distance between lingual arteries (DLA): in the same previous ultrasonography scanning position, the lingual arteries were observed
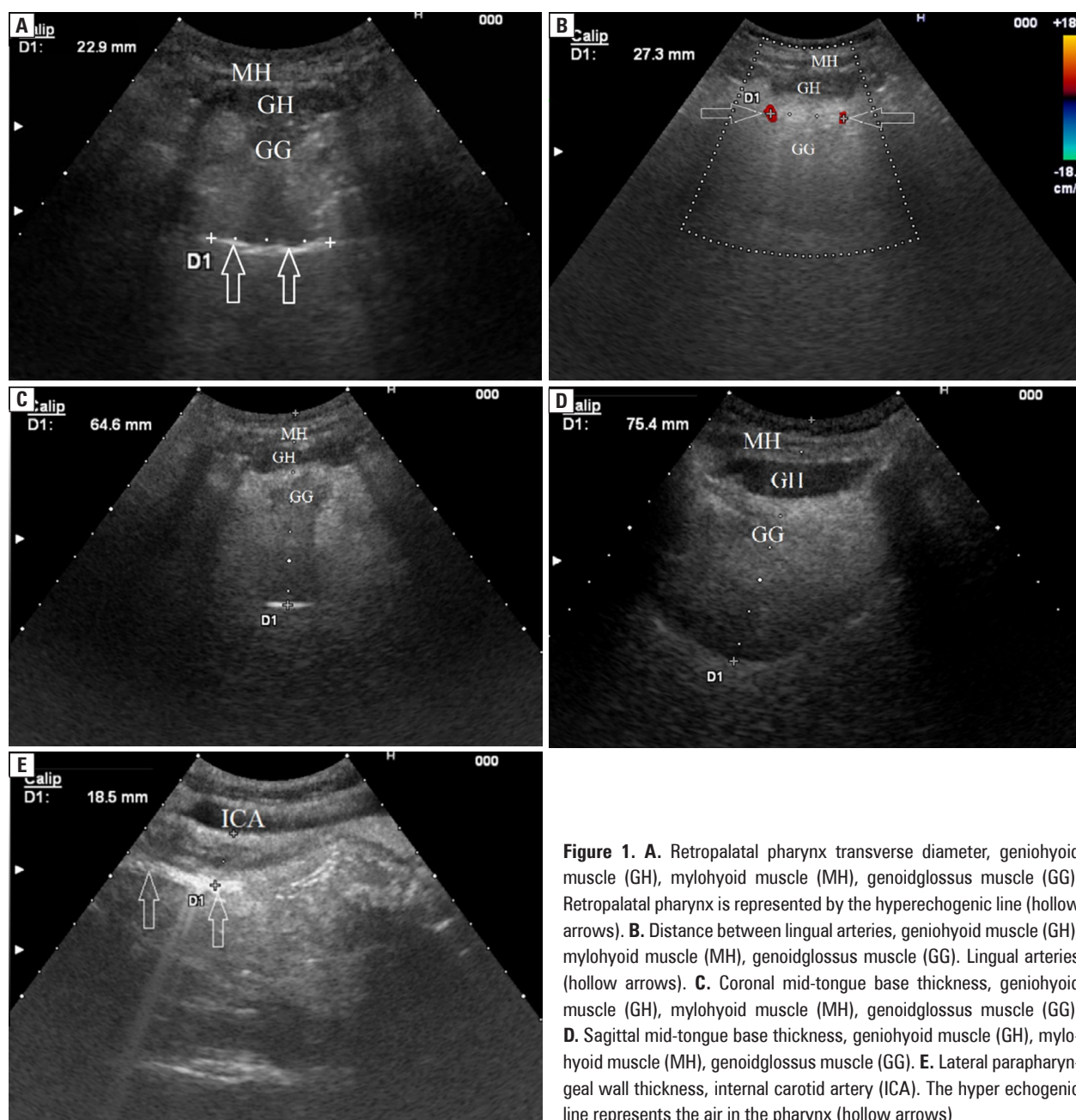
Figure 1. A. Retropalatal pharynx transverse diameter, geniohyoid muscle (GH), mylohyoid muscle (MH), genoidglossus muscle (GG). Retropalatal pharynx is represented by the hyperechogenic line (hollow arrows). **B.** Distance between lingual arteries, geniohyoid muscle (GH), mylohyoid muscle (MH), genoidglossus muscle (GG). Lingual arteries (hollow arrows). **C.** Coronal mid-tongue base thickness, geniohyoid muscle (GH), mylohyoid muscle (MH), genoidglossus muscle (GG). **D.** Sagittal mid-tongue base thickness, geniohyoid muscle (GH), mylohyoid muscle (MH), genoidglossus muscle (GG). **E.** Lateral parapharyngeal wall thickness, internal carotid artery (ICA). The hyper echogenic line represents the air in the pharynx (hollow arrows)

by power Doppler scan on both sides of the lower lateral border of the tongue base. The distance between lingual arteries (DLA) was measured (Figure 1B).
— Coronal mid-tongue base thickness (CTBT): in the same previous coronal ultrasonography scanning position, the mid-tongue base thickness (CTBT) was measured (Figure 1C).
— Sagittal mid-tongue base thickness (STBT): from the previous coronal ultrasonography scanning position the probe was rotated 90° into the sagittal position, then the sagittal mid-tongue base thickness (STBT) was measured (Figure 1D).

— Lateral parapharyngeal wall thickness (LPWT): the oblique coronal plane of the parapharyngeal space was scanned with the transducer longitudinally placed on the lateral side of the neck, just underneath the lateral border of the occipital bone. The long axis of the ipsilateral internal carotid artery was identified with color application. The lateral wall of the pharynx appeared as an echogenic line on real-time ultrasound, whereas the lumen of the pharynx was completely obscured by gas shadowing. Vibration artifacts occasionally occurred when the subjects swallowed, which also helped to confirm the location of the

pharynx. The distance between the internal carotid artery and the echogenic surface of the pharynx represented the LPW thickness in an oblique coronal plane. All the measurements were recorded on frozen images when the lateral wall of the pharynx moved farthest away from the transducer (Figure 1E).

Statistical methods

Data were statistically described in terms of mean ± standard deviation (± SD), range, frequencies (number of cases) and/or percentages when appropriate. Correlation coefficient was used to find the strength of association between numerical variables — that is, anthropometric measurements, submental US measurements, and PSG parameters. Receiver operating characteristics (ROC) analysis was used to determine the optimal cutoff values of the individual anatomical US indices and individual anthropometric indices for predicting and screening for OSA using computer-generated randomization of the OSA patients with complete data, multivariate analysis of the patient group. Pearson correlation (r) was used to detect association between quantitative variables. Accuracy was represented using the terms sensitivity, specificity, +ve predictive value, –ve predictive value, and overall accuracy. Multivariate logistic regression analysis models were used to test for the preferential effect of the independent variable(s) on the occurrence of OSA. Probability (P-value) is considered significant if < 0.05 level. All statistical calculations were performed manually as well as using computer program SPSS (Statistical Package for the Social Science; SPSS Inc., Chicago, IL, USA) version 22 for Microsoft Windows.

Results

This study comprised two groups. The group 1 included OSA patients (n = 33): 15 males (45.5%) and 18 females (54.5%), their age range was 39–75 years with mean ± SD of 57.76 ± 7.84 years, their body mass index (BMI) ranged from 28.10 to 66.10 kg/m^2 with mean ± SD of 43.30 ± 10.11 kg/m^2. They were subdivided according to polysomnography results into mild OSA (5 ≤ AHI < 15) patients (9/33, 27.2%), Moderate OSA (15 ≤ AHI < 30) patients (12/33, 36.4%) and Severe OSA (AHI ≥ 30) patients (12/33, 36.4%). The group 2 included 10 healthy individuals as a control group that comprised 5 males (50%) and 5 females (50%), their age range was 35–58 years with mean ± SD of 48.00 ± 7.85 years, their BMI

range was 25–45.80 kg/m^2 with mean ± SD of 32.08 ± 6.67 kg/m^2. The most common sleep-related symptoms among OSA patients were excessive daytime sleepiness (EDS) 28/33 (84.8%), nocturia 27/33 (81.8%), snoring 26/33 (78.8%) and morning headache 25/33 (75.8%), and the difference between the cases and controls was statistically significant. There was a statistically significant difference between OSA cases and the controls regarding mean age (57.76 ± 7.84 *vs* 48.0 ± 7.85; P < 0.01), BMI (mean ± SD = 43.30 ± 10.11 *vs* 32.08 ± 6.67; P < 0.001), neck circumference (mean ± SD = 43.24 ± 2.69 *vs* 38.20 ± 1.23; P < 0.001) and ESS (mean ± SD = 16.15 ± 3.12 *vs* 3.50 ± 2.84; P < 0.001). No statistically significant difference between the cases and controls regarding diabetes mellitus and hypertension was found. But the difference in incidence of ischemic heart disease between OSA cases (19/33, 42.4%) and the controls (1/10, 10%) was statistically significant (P < 0.05).

Polysomnography and neck ultrasonography data of the studied groups was presented in Table 1, and it revealed a statistically significant difference between OSA cases and the controls regarding A+H/h in supine position, respiratory-related leg movements (Resp-LMs) index, minimal and average oxygen saturation (%), SpO$_2$ Time < 90%, oxygen desaturation index, apnea index, hypopnea index, AHI and arrhythmia index. In regard to ultrasonography findings, anatomical parameters showed a statistically significant increase in LPWT (P < 0.001) and DLA (P < 0.01) among OSA patients in comparison to the controls. Moreover, there was a significant decrease or shortening in the RPD (P < 0.05) in OSA patients than among the controls. There was a significant positive correlation between LPWT and neck circumference [correlation coefficient (r) = 0.571, P < 0.001) and BMI (r = 0.328, P < 0.05) and a significant positive correlation between DLA and neck circumference (r = 0.384, P < 0.05). In addition, there was a significant negative correlation between RPD in all the studied groups and neck circumference (r = -0.407, P < 0.01).

The correlation between neck ultrasonography findings and polysomnography parameters among all studied groups showed that LPWT is negatively correlated with baseline oxygen saturation (r = -0.320, P < 0.05) and average oxygen saturation (r = -0.483, P < 0.001), while it was positively correlated with SpO$_2$ time < 90% (r = 0.445, P < 0.01), oxygen desaturation index (r = 0.545, P < 0.001), apnea index (r = 0.393, P < 0.01), hypopnea index (r = 0.550, P < 0.001), AHI (r = 0.586, P < 0.001), A+H/h in supine position

Table 1. Polysomnography and ultrasonography data of the studied groups

	The studied groups		P value
	OSA patients (N = 33)	Control (N = 10)	
Polysomnography data			
Total sleep time (TST) [min] (mean ± SD)	292.85±90.42	345.70±59.07	> 0.05
Total sleep period (SPT) [min] mean ± SD	339.21±78.00	377.50±39.51	> 0.05
Sleep efficiency (mean ± SD)	85.63±20.20	84.7±94.45	> 0.05
Wake index (mean ± SD)	4.92±5.36	7.08±8.55	> 0.05
Flow limitation index % total (mean ± SD)	3.01±8.29	13.67±12.32	> 0.05
% of sleep in supine position (mean ± SD)	75.95±28.86	81.48±22.70	>0.05
A+H/h in supine position (mean ± SD)	28.00±21.98	1.49±1.54	**< 0.001**
Isolated LMs index (mean ± SD)	16.15±16.74	16.35±13.18	> 0.05
Resp-LMs index (mean ± SD)	29.94±39.80	0.40 ±0.55	**< 0.001**
Minimal oxygen saturation [%] (mean ± SD)	68.09±12.63	83.40 ±10.12	**< 0.001**
Baseline oxygen saturation [%] (mean ± SD)	90.58±5.88	92.90 ±4.28	> 0.05
Average oxygen saturation [%] (mean ± SD)	87.41±6.94	92.50 ±4.74	**< 0.05**
SpO_2 time < 90% [%] (mean ± SD)	48.63±32.26	20.54 ±32.41	**< 0.01**
Oxygen desaturation index (mean ± SD)	44.51±26.33	5.25±5.96	**< 0.001**
Apnea index (mean ± SD)	13.75±15.81	5.25±5.96	**< 0.001**
Hypopnea index (mean ± SD)	17.66±13.90	1.39±1.30	**< 0.001**
AHI (/h) (mean ± SD	32.05±22.79	1.63±1.64	**< 0.001**
Snore index (mean ± SD)	158.51±172.41	128.24±151.15	> 0.05
Arrhythmia index (mean ± SD)	66.12±129.02	0.41±0.49	**< 0.001**
Neck ultrasonography data			
RPD [cm] (mean ± SD)	1.61±0.82	2.32±0.28	**<0.05**
DLA [cm] (mean ± SD)	2.63±0.47	2.07±0.48	**< 0.01**
CTBT [cm] (mean ± SD)	6.56±0.67	6.72±0.77	> 0.05
STBT [cm] (mean ± SD)	7.26±0.73	7.11±0.70	> 0.05
LPWT [cm] mean ± SD	4.59±1.01	3.41±0.71	**< 0.001**

CTBT — coronal mid-tongue base thickness; DLA — distance between lingual arteries; LPWT — lateral parapharyngeal wall thickness; N — number; Resp-LMs — respiratory-related leg movements; RPD — retropalatal pharynx transverse diameter; SD — standard deviation; STBT — sagittal mid-tongue base thickness. P value ≤ 0.05 is significant

(r = 0.542, P < 0.001) and arrhythmia index (r = 0.354, P < 0.05). DLA is negatively correlated with minimal oxygen saturation (r = -0.501, P < 0.001) but positively correlated with SpO_2 time < 90% (r = 0.402, P < 0.01), oxygen desaturation index (r = 0.368, P < 0.05), Apnea index (r = 0.317, P < 0.05) and AHI (r = 0.328, P < 0.05). Also, there was a significant positive correlation between Resp-LMs index and LPWT (r = 0.326, P < 0.05), DLA (r = 0.353, P < 0.05) and a negative correlation with RPD (r = -0.320, P < 0.05).

From receiver operating characteristic (ROC) curve for prediction of OSA using neck ultrasonography parameters, a cutoff value of 3.47cm for LPWT showed sensitivity of 100% and specific-ity of 60%, and the area under the curve (AUC) was 0.855, with high statistical significance (P < 0.001), and it was the most predictive for the presence of OSA, followed by DLA with a cutoff value of 2.105 cm (AUC = 0.785, sensitivity: 90.9%, specificity: 60%, P < 0.01), then RPD with a cutoff value of 1.65 cm as the least predictive diagnostic parameter of OSA (AUC = 0.750, sensitivity: 54.5%, specificity: 100%, P < 0.05) (Figure 2). So, the combination of LPWT and RPD results can achieve a 100% sensitivity and a 100% specificity. Also, LPWT was found to be useful in predicting OSA severity as there was a statistically significant difference between its value in mild and moderate OSA cases (mean ± SD =

Table 2. Collective comparison of P value between neck ultrasound parameters in the four study groups

	Normal *vs* mild OSA	Normal *vs* moderate OSA	Normal *vs* severe OSA	Mild OSA *vs* moderate OSA	Mild OSA *vs* severe OSA	Moderate OSA *vs* severe OSA
RPD	**0.004**	0.123	0.107	0.219	0.148	1.000
DLA	0.095	**0.006**	**0.036**	0.247	0.554	0.347
CTBT	0.968	0.674	0.974	0.917	0.972	0.630
STBT	0.497	0.314	0.628	0.651	1.000	0.977
LPWT	0.065	**0.002**	**0.001**	**0.034**	**0.018**	0.514

RPD — retropalatal pharynx transverse diameter; DLA — distance between lingual arteries; CTBT — coronal mid-tongue base thickness; STBT — sagittal mid-tongue base thickness; LPWT — lateral parapharyngeal wall thickness. P value ≤ 0.05 is significant

3.99 ± 0.39 *vs* 5.08 ± 1.43; $P < 0.05$) and between mild and severe OSA cases (mean $\pm$ SD = 3.99 ± 0.39 *vs* 4.55 ± 0.50; $P < 0.05$). LPWT was also significantly lower in the normal controls (mean $\pm$ SD = 3.41 ± 0.71) than moderate and severe OSA patients ($P < 0.01$ and 0.001 respectively). Also, DLA was significantly lower in normal controls (mean $\pm$ SD = 2.07 ± 0.48) than moderate (mean $\pm$ SD = 2.75 ± 0.50) and severe (mean $\pm$ SD = 2.60 ± 0.46) OSA cases ($P < 0.01$ and 0.05, respectively). In addition, RPD was significantly lower in mild OSA cases than normal controls (mean $\pm$ SD = 29.43 ± 17.13 *vs* mean $\pm$ SD = 34.61 ± 14.11; $P < 0.001$). Collective comparison of P value between neck ultrasound parameters in the four study subgroups was shown in Table 2.

Discussion

Obstructive sleep apnea is considered the most severe form of sleep-related breathing disorders. It can result in cardiovascular diseases, metabolic dysregulation, and neuro-cognitive dysfunction. Early diagnosis and intervention with continuous positive airway pressure (CPAP) can effectively improve airway patency, daytime wakefulness, blood pressure, metabolic abnormalities, and the quality of life [7]. Sub-mental US can detect both the anatomic characteristics, the dynamic changes of pharyngeal airspace and discriminate OSA severity [4]. The study investigated the role of ultrasonography in detecting anatomic upper airway changes in OSA patients and was trying to assess the value of these anatomical findings in prediction of OSA.

The mean age of the studied OSA patients was significantly higher than the controls'. This was consistent with Waterman *et al.* [8] who found that age is one of the most significant risk factors for OSA with mean $\pm$ SD value of $67.87 \pm$ 5.92 years, as age is a well-known risk factor associated with changes in the upper airway tone, pharyngeal fat distribution and the development of comorbid diseases. La Grotte *et al.* [9], Chen *et al.* [10], Jehan *et al.* [11] and Gray *et al.* [12] have shown a correlation between the prevalence of OSA syndrome and obesity (with BMI of 25–29.9 kg/m^2, 25–29 kg/m^2, 28–31 kg/m^2 and more than 30 kg/m^2, respectively). In a longitudinal analysis of a subset (n = 690) of the Wisconsin cohort with a 4-year follow-up, Peppard *et al.* [13] have shown that a 10% increase in weight is associated with a 6-fold greater risk of developing OSA among persons initially free of OSA. These findings were in harmony with our results.

The neck circumference of the study patients ranged from 38 to 49 cm with a mean $\pm$ SD of 43.24 ± 2.69 cm (17 ± 1 inches). The current study agreed with several studies done by Medeiros *et al* [14] and Simpson *et al.* [15] who found that a large neck circumference was predictive of OSA in the general population, and it is considered a significant marker for central obesity and has been associated not only with OSA but also with increased cardiovascular risk and insulin levels in OSA patients. Neck circumference cut-points predicting OSA risk are ≥ 17 inches (43.2 cm) and 16 inches (40.6 cm) for males and females, respectively. This study agreed with Christensen *et al.* [16], who evaluated the validity of ESS in diagnosing SDB and found a significant positive correlation between ESS and AHI. They concluded that if ESS > 10, SDB should be suspected. Sleep apnea increases the risk of hypertension, cardiovascular disease, and cerebrovascular disease [17]. In agreement with our findings, Shah *et al.* [18] and Garcia-Rio *et al.* [19] reported that mild-severe OSA is an independent risk factor for MI and a risk of recurrent MI and revascularization was lower in OSA patients who
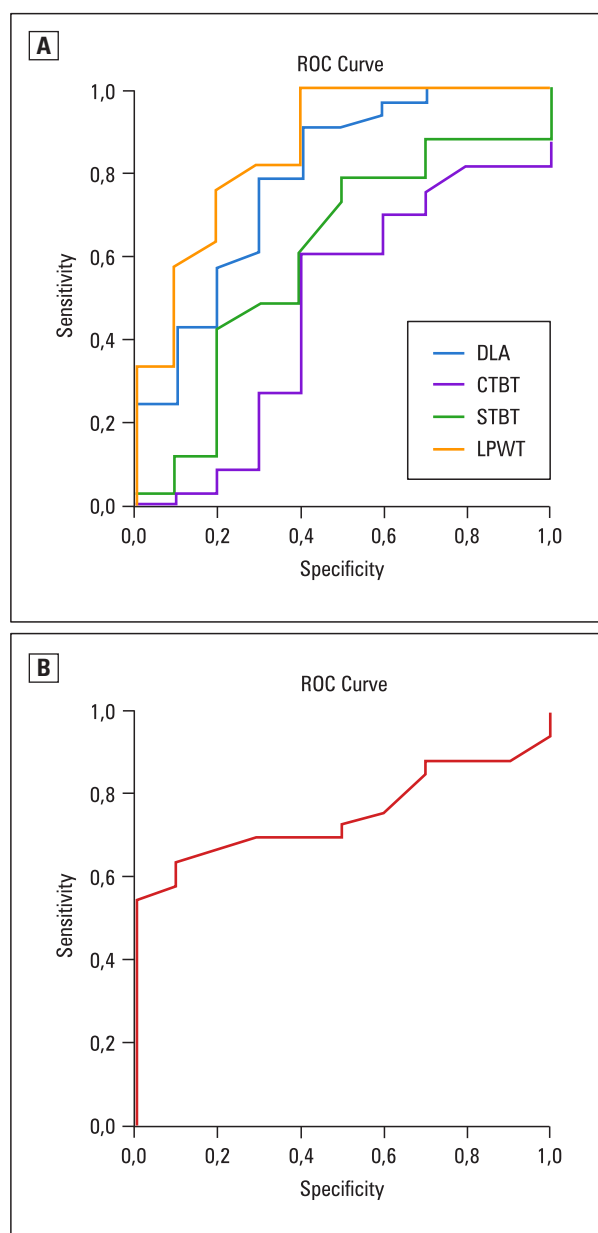
Figure 2. A. Receiver operating characteristics (ROC) curve for prediction of obstructive sleep apnea (OSA) using neck ultrasound parameters. **B.** ROC curve for prediction of OSA using retropalatal pharynx diameter. DLA — distance between lingual arteries; CTBT — coronal mid-tongue base thickness; STBT — sagittal mid-tongue base thickness; LPWT — lateral parapharyngeal wall thickness

tolerated CPAP. This study revealed a statistically significant difference between OSA patients and controls regarding EDS (P < 0.001), nocturia (P < 0.001), snoring (P < 0.01) and morning headache (P < 0.05). This was consistent with Xie *et al.* [20] who reported that EDS is a chief clinical consequence among patients with SDB and is directly related to the severity of sleep apnea.

Regarding the comparison between OSA patients and controls in polysomnography and neck ultrasonography data, our results were consistent with Bidarian-Moniri *et al.* [21], who found altered AHI depending on the position and were in accordance with Yousef and Alkhiary [22] who reported significantly higher oxygen desaturation index and significantly lower minimal oxygen saturation in OSA patients than controls. As expected, AHI demonstrated significantly higher values in OSA patients than controls in this study (mean ± SD = 32.05 ± 22.79; *vs* mean ± SD = 1.63 ± 1.64; P < 0.001). Spicuzza *et al.* [23] mentioned that snoring, male gender, middle age, menopause in women, obesity and a variety of craniofacial and oropharyngeal features such as a large neck circumference, retro- or micrognazia, nasal obstruction, enlarged tonsils/adenoids, macroglossia and low-lying soft palate are all important factors in pathogenesis of OSA which could explain a higher AHI among these groups. The prevalence of arrhythmia in OSA patients was significantly higher than in controls (arrythmia index: mean ± SD = 66.12 ± 129.02 *vs* mean ± SD = 0.41 ± 0.49; P < 0.001). Hypoxemia, reoxygenation, hypercapnia, negative intrathoracic pressure, arousal, sleep deprivation and hypertension that are associated with OSA affect electrical stability and increase the risk of arrhythmias. Likewise, Almeneessier *et al.* [24] found that arrhythmia in OSA patients was higher than in controls (26.9% *vs* 11.5%; P < 0.001) with higher incidence of premature atrial contraction, premature ventricular contraction (PVC) and atrial fibrillation in OSA patients.

Regarding correlations between neck US measurements and polysomnography parameters, our results agreed with Shu *et al.* [4] and Liu *et al.* [25] who found that enlargement of soft tissue structures, particularly the LPW, is associated with an increased likelihood of OSA. In addition, enhancement of LPWT is the principal cause of OSA among patients presenting to sleep disorders centers. Again, Lan *et al.* [26] reported that increased LPWT was the main cause of airway collapse, and this was significantly associated with higher oxygen desaturation index, higher AHI, higher apnea index, lower minimal SaO₂, and higher SpO₂ time < 90%. This study revealed a highly significant positive correlation between LPWT and A+H/h in supine position, and this was confirmed by Yalciner *et al.* [27] who found that supine position makes the upper airway collapsible by the tongue and mandible, which is thought to be responsible for the worsening of OSA compared with non-supine position. Also, LPWT and DLA were significantly lower among this study normal subgroup than in moderate and
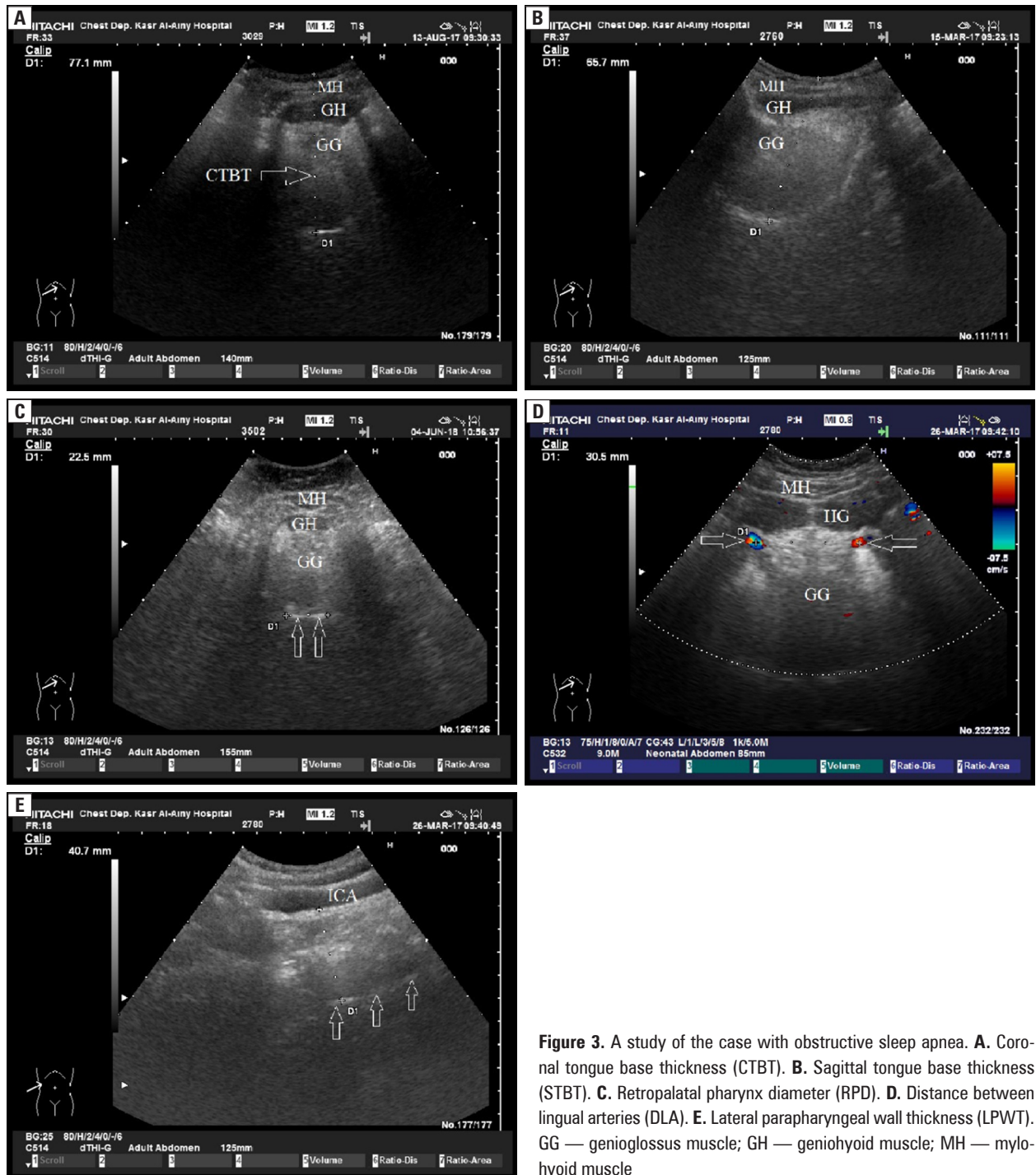
Figure 3. A study of the case with obstructive sleep apnea. **A.** Coronal tongue base thickness (CTBT). **B.** Sagittal tongue base thickness (STBT). **C.** Retropalatal pharynx diameter (RPD). **D.** Distance between lingual arteries (DLA). **E.** Lateral parapharyngeal wall thickness (LPWT). GG — genioglossus muscle; GH — geniohyoid muscle; MH — mylohyoid muscle

severe OSA subgroups, and this was in agreement with Bilici *et al.* [7] who stated that submental US can be used to precisely measure tongue and lateral pharyngeal wall dimensions by integrating the information. The LPWT and DLA correlate with severity of OSA as well. Lahav *et al.* [28], Yonatan *et al.* [29] and Li *et al.* [30] concluded that, when DLA exceeded 30 mm, the risk for OSA was significantly increased and reported that, with cutoff point of 30 mm for DLA, the

sensitivity, specificity, and accuracy for screening and diagnosis of OSA were 71.3, 74.7, and 63.6%, respectively, which was very close to our results. This study demonstrated that DLA is negatively correlated with minimal oxygen saturation and positively correlated with SpO_2 time < 90%, oxygen desaturation index, AHI and apnea index. This was in agreement with a study done by Ahn *et al.* [31] who found that absolute tongue volume showed stronger associations with

lowest O_2 saturation during sleep than with the severity of AHI. Retropalatal pharynx transverse diameter was significantly lower in the mild OSA group than in the normal group in the present study. This finding was in agreement with Ali and Muhammad [32] who found a highly significant decrease in RPD in OSA patients compared to the controls. In addition, they observed significant progressive shortening and narrowing for RPD with progression of OSA severity and AHI. Also, Zhang *et al.* [33] reported that shorter retro-palatal distance was associated with higher AHI.

In the present study, STBT and CTBT were not significantly different between OSA patients and the control groups. Similar to our findings, Okubo *et al.* [34], Iida-Kondo *et al.* [35], Shigeta *et al.* [36] and Ahn *et al.* [31] found that tongue volume was not significantly different between OSA patients and the controls, and tongue volume did not correlate with BMI, AHI or RDI. Also, this study showed a significant positive correlation between Resp-LMs index and LPWT (r = 0.326, P < 0.05), DLA (r = 0.353, P < 0.05) and a negative correlation with RPD (r = -0.320, P < 0.05). In addition, a significant positive correlation was found between STBT and isolated LMs index. This was explained by Manconi *et al.* [37] who mentioned that respiratory-related leg movements are provoked by respiratory-related arousals and because of the periodic nature of obstructive respiratory events, they "mimic" periodic leg movements. Thus, they are considered unique from periodic leg movements that occur as part of the phenotypic spectrum of restless legs syndrome.

The main limitation of the present study is that the cases and the control group are not matched in respect to age, BMI or neck circumference, which has definitely affected the ultrasound parameters, so we tried to divide OSA patients into grades and correlating these grades with ultrasound measurements for more accurate comparisons. In conclusion, sleep-related breathing disorders represent a spectrum of abnormalities ranging from simple snoring to upper airway resistance syndrome to sleep apnea. This study tried to highlight the role of ultrasound in OSA as a more objective tool than the questionnaire, more convenient because it does not require overnight time consumption and more relevant than pulse oximetry for examining the pharyngeal airspace. This is supported by the very good sensitivity in the present study, which demonstrates that submental US is sufficiently sensitive for differentiating OSA severity.

Conflict of interest

None declared.

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