Microvascular True Vocal Fold Lesions

Khalid Hassan Al-Malki, M.D., Ph.D.
Associated Professor
Consultant of Communication Disorders
Communication and Swallowing Disorders Unit (CSDU)
Department of ORL
King Abdul Aziz University Hospital
Riyadh, Saudi Arabia

Correspondence address:
Khalid H. Al-Malki
P.O Box 23008
Riyadh, 11426
Saudi Arabia
kmalky@yahoo.com
Fax No. (01) 4775682
Abstract

Objective: There is little discussion in literature about microvascular true vocal fold lesions. The objective of this study was to present a review article about these benign true vocal fold lesions.

Material and Method: Literature review was conducted using PubMed (MEDLINE) for English articles, from January 1980 to February 2007. The following keywords were used: microvascular AND vocal AND fold, ectasia AND vocal AND fold, varix OR varices AND vocal fold, hemorrhage AND vocal AND fold, hematoma AND vocal AND fold, and ultrastructure AND vocal AND fold.

Results: Microvascular true vocal fold lesions are more prevalent in female professional voice users. Such patients seek medical evaluation earlier because of the negative effect of these lesions on their career. Microvascular true vocal fold lesions are managed by voice therapy and/or phonosurgery.

Conclusion: In the asymptomatic patient, such lesions may create a management dilemma due to the risk of future hemorrhage versus the immediate risks of management.

Key words: Ectasia, varices, microvascular, voice, larynx.
Definitions and terminology

Microvascular lesions found within the true vocal fold may threaten the career of a professional vocalist because of recurrent submucous hemorrhage or scar formation. Vocal professionals seek evaluation much sooner and for more subtle vocal changes than the average individual does. In the asymptomatic patient, such lesions may create a management dilemma due to the risk of future hemorrhage versus the immediate risks of intervention.

There is little discussion in the literature regarding the nomenclature for subepithelial microvascular vocal fold malformations, which are commonly referred to as ectasias and varices (E's and V's). Varix is a Latin term and is defined as a dilated vein, while ectasia is a Greek term and is defined as a dilated tubular structure [1].

Due to the lack of well-accepted terminology, Hochman et al. [2] adopted the following classification for convention: (1) varix; (2) papillary ectasia, and (3) spider telangiectasia. In their work, varices (Figure 1) were defined as enlarged veins or acutely tortuous vessels. Papillary ectasia (Figure 2) resembles spheroid-appearing coalescent hemangioma. Spider telangiectasia resembles the dermatologic disorder and demonstrates a fine network of inappropriately oriented vessels. In
their opinion, Bouchayer and Cornut [3] prefer the term "vascular corditis".
Epidemiology:

Vocal fold E's and V's are more prevalent in female professional voice users, particularly singers who “push their larynges to the limit” [4, 5, 6], but such lesions are not rare in males [6].

The retrospective review of Postma et al. [6] on 800 dysphonic patients showed that 25 patients (3.1%) had isolated true vocal fold E’s or V’s, 76 % of them (19 patients) were females, and 88 % of the them were professional voice users. In their sample, this includes professional singers, teachers, and a newscaster. The prevalence of E's and V's was 4.5% among all females, and 1.6% among all males. Nine of the 25 patients (about 1/3) needed surgery to treat their dysphonia.

In their study, Hochman et al. [2] operated on 42 patients with microvascular vocal fold lesions. Thirty four patients (81%) were females, and 39 patients (93%) were singers. Ten patients (24%) had isolated lesions, and the remaining 32 patients (76%) had microvascular vocal fold lesions in association with other benign vocal fold lesions, such as vocal fold nodules, polyps, or cysts.
Etiology and pathogenesis:

Standard histopathologic and electron microscopic analysis of the microvascular anatomy of the normal true vocal fold revealed that the blood vessels in the superficial lamina propria (SLP) course in a longitudinal direction with frequent arteriovenous anastomoses [7, 8]. This vascular pattern is believed to facilitate continuous blood flow through the microcirculatory system of the SLP during high-pressure phonation. There is no direct communication between the microvasculature of the SLP and the vocalis muscle, which allows for maximal flexibility of the mucosal cover over the core [8]. Therefore, vessel patency is maintained despite the aerodynamically induced shearing stresses that are placed on the microvasculature in the SLP during vocal fold oscillation.

Postma et al. [6] mentioned that many authors believe that these microvascular vocal fold lesions may be secondary to: (1) vocal abuse and misuse; (2) hormonal variations, because of preponderance of these lesions in female professional voice users; or (3) repeated inflammation.

Vocal trauma is a major contributing factor in the development of microvascular vocal fold lesions [9, 10, 11]. It has been postulated that increased blood flow or a reactive hyperemia after intensive vocal usage,
similar to upper extremity vascular engorgement after vigorous exercise, may lead to varix formation [8, 12, 6].

The reported female predominance of E's and V's supports the fact that these vascular malformations may be hormonally related, as previously suggested [13, 14]. However, this hypothesis has not been proven [6, 2]. It is also possible that the gender difference is related to differences in vocal fold anatomy and associated mechanics of sound production [2].

It has been reported that the patient with E's and V's may suddenly become aphonic due to a submucous hemorrhage (Figure 3), apparently due to a ruptured varix [5]. Based on our review of literature, we summarized the natural history of a microvascular vocal fold lesion in figure 4.
Clinical picture:

The clinical presentation of vocal fold microvascular lesions is highly variable. These lesions can be asymptomatic or may cause: (1) dysodia, which is a change or failure of some aspects of the singing voice, although the speaking voice is intact [15]; (2) dysphonia; and/or (3) phonasthenic symptoms. The latter include sensation of throat dryness or tightness; frequent throat clearing; sensation of sticky secretions in the throat that are difficult to swallow; and inability to continue speaking during vocally stressful situations [15]. In women, the appearance of the lesion may depend on the stage of the menstrual cycle; therefore, periodic examinations may be required to accurately establish severity.
Laryngeal endoscopy:

Endoscopic true vocal fold examination may show varices, ectasias, and/or spider telangiectasias. The additional importance of the laryngoscopic examination is to detect any underlying benign vocal fold lesions of which the E's and V's are but a surface manifestation. Kleinsasser [5] mentioned that 50% of varices affect one vocal fold only, and the rest affect both folds.

Hochman et al. [2] reported that E’s and V’s are most commonly located in the middle third of the membranous part of the true vocal fold. They called this area the *striking zone* because of the predominance of phonatory-induced traumatic lesions in this part. They reported also that in two thirds of their cases, these lesions are located on the superior surface of the true vocal folds. They believed that deceleration forces involved in vocal fold oscillation create a whiplash-type injury to the microvasculature at this site. They also mentioned that in the other one third of cases, these lesions are found on the medial surface of the true vocal folds. According to them, collision forces probably make a major contribution to the formation of these lesions on the medial surface of the vocal folds.
Laryngostroboscopy:

These lesions may alter the vibratory characteristics or closure of the true vocal fold. This may be secondary to a differential mass effect on vocal fold vibration or physical interposition of the lesion between the vocal folds edges [6]. This may hinder the movement of the "cover" layer upon the vocal fold “core”, and produces greater stiffness of the vocal fold. On the other hand, some reports mentioned that ectasias of their patients did not greatly interfere with stroboscopic vibration [3]. It seems that the variation in the number and size of E's and V's contribute to the variable stroboscopic findings.
**Treatment:**

E's and V's can be treated by: (1) behavioral readjustment voice therapy, which involves vocal hygiene advice and vocal training exercises. Postma et al. [6] reported that two thirds of their patients improved by this line of treatment only; (2) adjuvant antireflux therapy [2]; and/or (3) phonomicrosurgery. This involves cauterizing, vaporizing, or excising the dilated vessels or masses. The excision of these delicate vessels is not detrimental to the blood supply of the true vocal folds, due to the presence of a rich network of anastomosing vessels [8, 16].

The timing of surgery is critical. Some female patients have a significant increase in the size of the varix in the premenstrual and early menstrual period. Surgery during this time or immediately preceding this enlargement could result in postoperative submucosal hemorrhage with potentially increased scarring. Serial examination of the varix allows the surgeon to determine which patients have varices that change in size with menstruation, and to schedule the procedure a week or so after menses in those patients [6].

The indications for phonomicrosurgery are: recurrent vocal fold hemorrhage, large or enlarging varices, development of vocal fold
masses in conjunction of the varix or ectasia, or unacceptable dysphonia after maximal voice therapy [6].
**Phonomicrosurgical options:**

(1) Electromicrocautary:

Local application of cautery to stop the vocal fold hemorrhage was a common practice from the beginning of laryngology [2]. Bouchayer and Cornut [3] described their surgical intervention as follows. A series of point cauterizations are made along the length of the vessel with a needlepoint microcautery. It is always worth palpating to check for any area of induration that might indicate an intrafolder lesion. When in doubt, exploratory cordotomy may be performed. The intervention very often ends with an intrafolder hydrocortisone injection. It must be borne in mind that cauterized vessels take several weeks to disappear entirely. The final outcome is generally very good, both anatomically and functionally, on condition that pre- and postoperative voice therapy is conducted. Even so, there are occasional cases of relapse [3].

(2) Epithelial cordotomy and excision:

Kleinsasser [5] and Zeitels [17] suggested the use of cold instruments to dissect E' and V's. Hochman et al. [2] prefer cold instruments over CO2 laser because of fear of scarring and stiffness. In their procedure, an epithelial cordotomy is performed directly over the site of the vascular lesion regardless of its location. A sharp angled pick or needle is then used to dissect the lesion from the superficial lamina.
propriate and microforceps and/or microscissors are used to excise it. Bleeding is typically controlled with a topical solution of saline with 1:1,000 to 10,000 epinephrine. It is not uncommon for patients to have 2 to 4 separate incisions in one vocal fold involving both the medial and superior surfaces. The epithelial cordotomy is then allowed to heal primarily. Patients are placed on complete voice rest subsequent to the procedure for 7 to 14 days, and limited voice use for the next 10 to 14 days, depending on the rate of the patient's healing. Most patients are given antireflux treatment, and all of them received voice therapy. The same authors operated on 42 patients with microvascular vocal fold lesions. Cold instruments were used in 23 patients, and CO2 laser surgery was used in the remaining 19 patients. They reported generally better results in the cold instruments patients [2].

(3) Laser surgery:

Abitbol [18] and Postma et la. [6] used CO2 laser to treat unacceptable or recurrent dysphonia resulting from E's and V's. Postma et al. [6] described their surgical intervention as follows. The patient undergoing laser surgery is orally intubated with a small laser-safe endotracheal tube or is jet-ventilated and given intravenous dexamethasone. The lesion is palpated with a blunt probe under magnification, and the vessels supplying and draining the lesion are
identified by occluding the vessels sequentially and observing patterns of refilling. If present, the endotracheal tube cuff is protected with saline pledgets, and the vocal fold is covered with an iced epinephrine (1:10,000) pledget. A carbon dioxide laser with a micromanipulator is set at 1 to 2 W of power in single pulse mode of 0.1-second duration. The laser is defocused to 300 to 400 µm, and the vessels feeding the lesion are vaporized. This low power density lessens the likelihood of significant scarring of the superficial layer of lamina propria or vocal ligament damage. The draining vessels are then photocoagulated. The patient is briefly taken out of suspension to ensure that no refilling of the feeding vessels occurs. The main lesion itself is then photocoagulated or excised. The patient is, then, placed on voice rest for 7 to 14 days, and then he/she begins voice therapy.

In a retrospective review [6], CO2 laser surgery was performed on 9 patients out of 25 patients with vocal fold microvascular lesion. Eight of the 9 patients were able to return to full unrestricted vocal activity within an average of three and a half months. None of the 9 surgical patients has had a recurrent varix or further vocal fold hemorrhage. The follow up period ranged from 1 to 4.5 years.
Pulsed angiolytic lasers that emit radiation at high absorbance peaks of oxyhemoglobin cause intravascular photocoagulation or photoangiolyis of the subepithelial microcirculation [19]. Such lasers include the 585-nm pulsed dye laser (PDL) and 532-nm pulsed potassium titanyl phosphate (KTP) laser.

Hsiung et al. [20] introduced the 532-nm pulsed KTP laser in the treatment of microvascular vocal fold lesions. The efficiency of KTP laser operation in the continuous mode with a 0.4- or 0.6-mm beam (1- to 2-W aim for 3 to 7 seconds delivering a total energy of 3 to 7 J) was studied retrospectively in 14 patients with microvascular lesions of the vocal fold. They reported that KTP laser operation was a useful, cost-effective, and time-saving procedure and can be considered as an option in management of patients with microvascular lesions of the vocal fold, particularly those with repeated hemorrhages.

Zeitels et al. [19] conducted a prospective study on 39 patients (40 procedures in 54 vocal folds) with E's and V's to evaluate the effectiveness of a 585-nm pulsed dye laser (PDL; 25 cases) and a 532-nm pulsed KTP laser (15 cases). They demonstrated that both the 585-nm PDL and the 532-nm pulsed KTP lasers were found to be efficacious and relatively safe treatment modalities for vascular abnormalities of the
vocal folds in singers. However, the pulsed KTP laser was substantially easier to use because of its enhanced hemostasis due to its longer pulse width. Vessel wall rupture was common during the use of the 585-nm PDL, but rarely occurred during photoangiolysis with the 532-nm pulsed KTP laser.

Based on their study, Hirano et al. [21] showed that KTP laser photocoagulation is a relatively simple and safe procedure for treating microvascular lesions of the vocal fold. But, it is not recommended for photocoagulation of hemorrhagic polyps or hematomas, because such lesions have little blood flow inside and thus photocoagulation is usually impossible or requires too much laser energy. However, photocoagulation of perimeter or feeding vessels of such disorders may facilitate the following procedure by avoiding unnecessary bleeding, as well as preventing recurrence of hemorrhagic lesions.

(4) Combined approach:

Kleinsasser [5] advised that large varices are excised with scissors after dividing the epithelium, whereas smaller lesions should be coagulated carefully with a needle. Such operation should not be carried out during the acute phase of bleeding into the vocal fold but should be delayed until the blood has been resorbed [5, 6]. Submucus vocal fold
hemorrhages are managed by absolute voice rest, increased oral hydration, and intramuscular dexamethasone [6]. However, CO2 laser can be used to vaporize supero-lateral lesions, and cold instruments to dissect lesions near or on the vibratory margin [2].
References


Correspondence address:

Khalid H. Al-Malki
P.O Box 23008
Riyadh, 11426
Saudi Arabia

kmalky@yahoo.com
Legends of figures:

Figure 1. Vocal fold varix on the right true vocal fold.

Figure 2. Vocal fold ectasia on the left true vocal fold.

Figure 3. Right true vocal fold hemorrhagic polyp with submucous hemorrhage.

Figure 4. The natural history of microvascular vocal fold lesions.