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Original article

Anterior laryngeal commissure: Histopathologic data from supracricoid partial laryngectomy



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ABSTRACT

Objectives: To analyze histopathologic invasion of the anterior laryngeal commissure on surgical specimens from patients operated on for stage-2 squamous-cell carcinoma managed by supracricoid partial laryngectomy (SCL).

Patients and methods: Twenty-five patients with previously untreated stage-2 squamous-cell carcinoma were selected. Preoperative endoscopy confirmed anterior commissure involvement; CT found no cartilage lysis. SCL was performed in all cases: 15 anterior frontal SCLs with epiglottoplasty, 8 with crico-hyoidepiglottopexy, and 2 with cricohyoidopexy. Histopathology analyzed resection margins (< 1 mm, 1–5 mm, > 5 mm), cartilage extension and vascular embolism. Mean time to observation was 18 months (range, 12–36 months).

Results: Resection margins were < 1 mm in 7 cases (28%), 1–5 mm in 9 and > 5 mm in 9 patients. Vascular emboli were found in 15 patients (60%). Twenty patients were free of medial thyroid cartilage involvement; 5 showed cartilage extension (20%), restricted to the internal cortical layer in 4 cases (stage T3) and transfixing in 1 (stage T4a). Mucosal extension appeared non-predictive of cartilage invasion. The T4a patient showed local laryngeal recurrence at 12 months.

Conclusions: In laryngeal commissure squamous-cell carcinoma, SCL enables pathologic analysis of the entire anterior commissure as organogenetically defined: medial thyroid wing, in which the three laryngeal regions are inserted. Microscopic cartilage invasion is poorly predicted by mucosal extension, and may affect 20% of initially T2 patients.

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1. Introduction

The anterior commissure of the larynx is the object of numerous controversies in laryngeal oncology: anatomic, diagnostic and therapeutic [1–3]. Twenty percent of glottic tumors show anterior commissure extension, with only 1% originating from the glottic commissure insertion [4]. Anatomic limits are imprecise: there is no universally accepted anatomic definition of the anterior commissure of the larynx [2]. Endoscopic and imaging diagnosis is hampered by the anatomic and histologic characteristics of the anterior commissure and the continuity between mucosa and cartilage, with early cartilage sclerosis, and presence of vessels and glands with potential lymphatic involvement [1,5]. Anterior commissure carcinoma with normal

laryngeal movement (T1b, T2) is classified as T3 if the internal cortical layer is involved, and as T4a if the tumor extends beyond the external cortical layer. Moreover, treatment attitudes are not consensual in anterior commissure tumor with conserved laryngeal movement, some authors recommending first-line radiation therapy, while others prefer endoscopic or external surgery [1,2].

The present retrospective study sought to describe histopathologic invasion of the anterior commissure of the larynx in specimens from patients undergoing primary supracricoid laryngectomy for squamous-cell carcinoma involving the anterior commissure.

2. Study population – Methods

Successive supracricoid laryngectomy patients were selected according to three main criteria:

- previously untreated laryngeal squamous-cell carcinoma;

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- anterior commissure of the larynx showing invasion on pathologic analysis of the diagnostic endoscopy specimen and on the surgical specimen at definitive analysis;
- patient not lost to postoperative follow-up.

Twenty-five patients were analyzed: 22 males, 3 females; mean age, 52 years (range, 32–76 years).

Twenty-three patients had perfectly normal laryngeal movement; 2 had conserved but impaired glottic movement. Four mucosal “quadrants” of tumoral extension were defined: superior epiglottic, left and right lateral ventricular, and inferior subglottic.

There was no destructive cartilage involvement on CT ahead of endoscopy.

Supracricoid laryngectomy was performed in all cases: anterior frontal laryngectomy with epiglottoplasty (n=15), supracricoid laryngectomy with cricohyoidepiglottopexy (n=8), and supracricoid laryngectomy with cricohyoidepexy (n=2). Bilateral cervical lymph-node resection of sectors IIa, IIb, III, IV and V was systematically associated. No radiation therapy was performed on the operated larynxes, but only on the cervical lymph-node areas, depending on the histopathologic analysis of adenopathy.

All histology slides were examined by the same pathologist, on a routine technique of serial sections with hematoxylin-eosin staining; resection margins were classified as < 1 mm, 1–5 mm or > 5 mm. Thyroid anterior commissure cartilage invasion was classified according to internal cortical involvement (T3) or transfixing external cortical involvement (T4a). Vascular emboli were recorded.

Tumor control was analyzed at a mean 18 months’ follow-up (range, 12–36 months).

3. Results

Twenty of the 25 patients were free of cartilage involvement (80%); 5 showed cartilage involvement (20%), non-transfixing (T3) in 4 patients and transfixing (T4a) in 1 (Figs. 1 and 2).

Fifteen patients showed vascular emboli (60%).

Resection margins were < 1 mm from the carcinomatous tumor in 7 cases (28%), 1–5 mm in 9 (36%), and > 5 mm in 9 (36%).

One patient (4%) showed local laryngeal recurrence 12 months after supracricoid laryngectomy with cricohyoidepiglottopexy; pathology found transfixing cartilage involvement (T4a), resection margins < 1 mm and vascular embolism. Salvage total laryngectomy was required to achieve control.

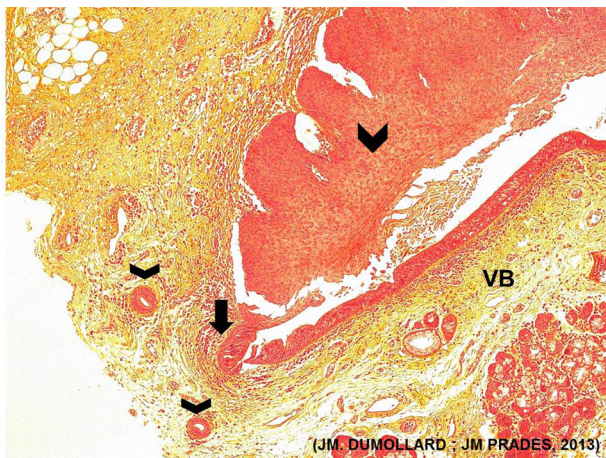


Fig. 1. Cricohyoidepiglottopexy (CHEP) specimen. Optic microscopy, hematoxylin-eosin staining (G × 25). ◀ Anterior glottic squamous-cell carcinoma; ↓ internal perichondrium infiltration; ◀ commissure vessels; VB: ventricular band.



Fig. 2. CHEP (anterior commissure): T4a cartilage invasion. Optic microscopy, hematoxylin-eosin staining (G × 50); ◀ anterior glottic squamous-cell carcinoma transfixing external cortical layer; ★ thyroid cartilage; ↓ external perichondrium.

Endoscopy found mucosal extension from the anterior glottic commissure to 4 quadrants (supra- and infraglottic and bilateral ventricular) in 2 patients with non-transfixing (T3) cartilage involvement, to 3 quadrants (supra- and infraglottic and unilateral ventricular) in 1 patient with transfixing (T4a) cartilage involvement, and to 2 quadrants (supraglottic and unilateral ventricular) in 2 patients with non-transfixing (T3) cartilage involvement. Hence, mucosal extension failed to predict cartilage involvement (Fig. 3).

Table 1 shows adenopathy status and type of surgery in the 5 patients with cartilage involvement: four showed emboli on histology, three < 1-mm margins and two 1–5-mm margins. Only one patient had reduced laryngeal movement.

	Cartilage C ⊕ (n = 5)	Cartilage C ⊖ (n = 20)
4-quadrant mucosal extension	<p>⊙ T3</p>	<p>9/20 (45%)</p>
3-quadrant mucosal extension	<p>⊙ T4a</p>	<p>4/20 (20%)</p>
2-quadrant mucosal extension	<p>⊙ T3</p>	<p>7/20 (35%)</p>

C ⊕: cartilage invasion
C ⊖: no cartilage invasion

Fig. 3. Mucosal and cartilaginous extension with type of surgery.

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