A Granular Cell Myoblastoma of the Larynx

Gill Ryoung Kim, Sun Kon Kim and Young Myung Kim

Department of Otolaryngology
Yonsei University College of Medicine, Seoul, Korea

In Joon Choi

Department of Pathology
Yonsei University College of Medicine, Seoul, Korea

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ABSTRACT

We have reported a case of granular cell myoblastoma of the larynx which is considered to be the first reported case in the Orient.

A review of the literature is given with special emphasis on the pathology, various theories of histogenesis, diagnosis, treatment and prognosis.

INTRODUCTION

Granular cell myoblastoma is a very rare tumor, especially in the larynx. The first report of a myoblastoma was Weber's lingual myoblastoma which was reported in 1864. Although there were other earlier reports by Heurtaux, Massin, Pendle, Moschowitz and Quirin, this disease entity was described precisely in 1926, by Abrikosoff who reported five myoblastoma cases in which there was one laryngeal case. To date we have gathered from the literature about 350 cases of myoblastoma throughout the entire body with 34 cases in the larynx. Since we could not find any reports from Korea, or even from the Orient, we wish to report this rare case as the first case in the Orient as well as a general review of the literature.

REPORT OF THE CASE

A 54-year-old Korean farmer suffering with progressive hoarseness and dyspnea for three years was admitted to Severance hospital on Jan. 6, 1963, via outpatient clinic with the tentative diagnosis of laryngeal polyp. His family and past history revealed nothing worthy of note except some unknown disease associated with generalized edema and jaundice at the age of 19. Mirror examination of the larynx revealed a whitish-gray, smooth, solitary mass which originated from the anterior half of the left cord. It was mostly located subglottically but would swing up with cough or forceful phonation. There was no ulceration, inflammation or erosion and the cord was not fixed.

Vital signs were: temperature, 37.6°C; pulse, 104/min.; respiration, 30/min.; blood pressure, 140/70. Chest X-ray showed increased bilateral lung markings consistent with suspected bronchitis. Urine, hematolgy and serology were within normal limits.

Operation and course: Considering the severe dyspnea and operative risk such as suffocation, we did a tracheotomy first, and subsequently tried to remove the mass under the indirect
laryngoscopy with 2 per cent pontocaine topical anesthesia. But the mass was too firm and large to remove by this method, so we had to postpone this operation. On the following day, we removed it under the suspension laryngoscopy with a laryngeal snare made just like a nasal polyp snare. The mass measured about 25×20×20 mm and was grayish white, firm and smooth. The patient was discharged on the 3rd post operative day in satisfactory condition. One year later, he was enjoying a comfortable life without hoarseness or dyspnea and on laryngeal examination there was no evidence of recurrence.

**Microscopic Finding:** As can be seen in picture 1, 2 and 3, the specimen showed an overlying layer of squamous epithelium. There was a minimal degree of superficial ulceration, and extending irregularly into the underlying tissue a form of pseudoepitheliomatous hyperplasia was noted. The underlying layer was replaced by groups of spindle, round, oval or polyhedral shaped cells of varying sizes, often having indistinct cell boundaries. The cytoplasm of these cells contained eosinophilic granules, and was sometimes vesicular and vacuolated. The nuclei were oval, round, or vesicular and centrally located. The granular cells show no definite evidence of cross striation.

**Fig. 2 and 3.** Varying sized and shaped granular tumor cell showing indistinct cell border, occasional vesicular and vacuolated cytoplasm with eosinophilic granules. (AO Spencer 430×)

**DISCUSSION**

**Regional distribution:** Murphy (1949) reported as shown in table 1. That is, the tongue is the most frequent site amounting to 34.1 per cent, followed by skin, breast, subcutaneous tissue, maxilla, muscle excluding tongue and vocal cord, in that order.

On the other hand, Bangle (1952, 1953) mentioned 43 cases with the following order of frequency tongue-21, skin-14, lip-3, breast-2, subcutaneous tissue-1, vocal cord-1, and floor of mouth-1. The percentile incidence of laryngeal myoblastoma to total granular cell myoblastoma cases is: 3.5 per cent according to Murphy (1949), 8 per cent by Klemperer, and 7.4 per cent by Paul H. Ward (1962).
Fig. 4. Age distribution of granular cell myoblastomas for 179 cases, by Murphy.

Table 1. Regional distribution of 229 cases (up to 1949 by murphy)

<table>
<thead>
<tr>
<th>Site</th>
<th>Cases</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tongue</td>
<td>78</td>
<td>34.1</td>
</tr>
<tr>
<td>Skin</td>
<td>22</td>
<td>9.6</td>
</tr>
<tr>
<td>Breast</td>
<td>18</td>
<td>7.9</td>
</tr>
<tr>
<td>Subcutis</td>
<td>16</td>
<td>7.0</td>
</tr>
<tr>
<td>Maxilla</td>
<td>16</td>
<td>7.0</td>
</tr>
<tr>
<td>Muscle, excluding tongue</td>
<td>11</td>
<td>4.8</td>
</tr>
<tr>
<td>Vocal cord</td>
<td>8</td>
<td>3.5</td>
</tr>
<tr>
<td>Thigh</td>
<td>8</td>
<td>3.5</td>
</tr>
<tr>
<td>Lips</td>
<td>6</td>
<td>2.6</td>
</tr>
<tr>
<td>Buttocks</td>
<td>5</td>
<td>2.2</td>
</tr>
<tr>
<td>Mandible</td>
<td>4</td>
<td>1.7</td>
</tr>
<tr>
<td>Mouth, excluding tongue</td>
<td>4</td>
<td>1.7</td>
</tr>
<tr>
<td>External auditory canal</td>
<td>3</td>
<td>1.3</td>
</tr>
<tr>
<td>Trachea and bronchus</td>
<td>3</td>
<td>1.3</td>
</tr>
<tr>
<td>Vulva</td>
<td>3</td>
<td>1.3</td>
</tr>
<tr>
<td>Others</td>
<td>24</td>
<td>10.5</td>
</tr>
<tr>
<td>Total</td>
<td>229</td>
<td>100.0</td>
</tr>
</tbody>
</table>

Age incidence: Murphy (1949) reported 179 cases (see Fig. 4) showing the most frequent age groups to be among the third to the fifth decades, with the peak incidence (20.5 per cent) in the fourth decade. This figure is very close to ours (see Fig. 5). As seen in Fig. 5, we could not find any laryngeal case below 23 years of age (Halperin’s case, 1964) and above 69

Fig. 5. Age distribution of laryngeal granular cell myoblastoma for 18 cases, by Kim. (Lyons, Haindel, and Blatt’s case, 1962).

Sex ratio: Murphy (1949) reported 93 males and 90 females, but it is different for laryngeal cases. In 1960, Stephen F. Balsky (1960) pointed out that the laryngeal myoblastoma shows a male predilection (male-13, female-5). Paul H. Ward (1962) reported that over 80 per cent of the laryngeal tumors are in the male. According to our review of 34 laryngeal myoblastomas, there are 27 males and 7 females, showing a male predilection.

This corresponds well with Holinger and Johnston’s (1951) report on 1,197 cases of benign laryngeal tumors which showed 70 per cent female, and also with Gill R. Kim’s (1960) report of 116 benign laryngeal tumors in Korea which shows 80 males (69 per cent) and 36 females (31 per cent).

Racial predilection: We could find no racial predilection. Among 15 cases which we could collect with racial distinction, there were 8 colored and 7 white.

Pathology

As table 2 shows, the origin of the tumors in the larynx are mostly between the posterior half of the cord and the posterior commissure excpt...
<table>
<thead>
<tr>
<th>No.</th>
<th>Author</th>
<th>Year</th>
<th>Description</th>
<th>Techniq. of treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Abrikossoff</td>
<td>1931</td>
<td>Pea-sized polyp, Rt. cord</td>
<td>snare</td>
</tr>
<tr>
<td>2.</td>
<td>Dawydow</td>
<td>1931</td>
<td>Pea-sized polyp, Rt. cord</td>
<td>biopsy</td>
</tr>
<tr>
<td></td>
<td>Derman, Golbert</td>
<td>1931</td>
<td>Pea-sized polyp, Rt. cord</td>
<td>local removal</td>
</tr>
<tr>
<td>3.</td>
<td>Geschelin</td>
<td>1934</td>
<td>Pea sized sessile mass, Rt. cord</td>
<td>laryngofissure and diathermy</td>
</tr>
<tr>
<td>4.</td>
<td>Kleinfield, also Klemperer</td>
<td>1934</td>
<td>1 cm nodular mass, Lt. cord</td>
<td>local removal</td>
</tr>
<tr>
<td>5.</td>
<td>Kernan, Cracovaner also Horn and Stout</td>
<td>1934</td>
<td>Post. 2/3 of Lt. cord</td>
<td>local removal</td>
</tr>
<tr>
<td></td>
<td>1943</td>
<td>Post. 2/3 of Lt. cord</td>
<td>recurred, local removal repeated</td>
<td></td>
</tr>
<tr>
<td>6.</td>
<td>Bobbio</td>
<td>1936</td>
<td>situated on Lt. cord</td>
<td>local removal</td>
</tr>
<tr>
<td>7.</td>
<td>Frenckner</td>
<td>1938</td>
<td>pedunculated mass, Lt. aryepiglottic fold</td>
<td>excision</td>
</tr>
<tr>
<td>8.</td>
<td>Iglauer</td>
<td>1942</td>
<td>pedunculated interarytenoid area</td>
<td>punch forceps</td>
</tr>
<tr>
<td>9.</td>
<td>Fust, Custer</td>
<td>1949</td>
<td>situated on vocal cord</td>
<td>excision</td>
</tr>
<tr>
<td>10.</td>
<td>Somers and, Farinacci</td>
<td>1953</td>
<td>a) small nodule, Lt. cord</td>
<td>forceps</td>
</tr>
<tr>
<td></td>
<td>1953</td>
<td>b) 1.5 cm plaque, Lt. cord</td>
<td>laryngofissure</td>
<td></td>
</tr>
<tr>
<td>11.</td>
<td>Maguda, Young</td>
<td>1953</td>
<td>small nodule, Lt. vocal process</td>
<td>forceps</td>
</tr>
<tr>
<td>12.</td>
<td>MacNaughtan, Fraser</td>
<td>1954</td>
<td>extensive plaque, posterior wall</td>
<td>laryngofissure, recurred irradiation and laryngectomy</td>
</tr>
<tr>
<td>13.</td>
<td>McKinlay</td>
<td>1954</td>
<td>broad-based nodule, Lt. vocal process</td>
<td>forceps</td>
</tr>
<tr>
<td>14.</td>
<td>Keohane</td>
<td>1956</td>
<td>solitary tumor, post. 1/3 of Rt. cord</td>
<td>forceps</td>
</tr>
<tr>
<td>15.</td>
<td>Hinton, Weinberger</td>
<td>1958</td>
<td>small nodule superior to Lt. false cord</td>
<td>forceps</td>
</tr>
<tr>
<td>16.</td>
<td>G. Jan Beckhuis</td>
<td>1959</td>
<td>a) ulcerative raised lesion, post. 1/3 of Lt. cord and interarytenoid area</td>
<td>forceps</td>
</tr>
<tr>
<td></td>
<td>1959</td>
<td>b) plaque, post. 1/3 of Rt. vocal cord</td>
<td>forceps</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1959</td>
<td>c) polypoid lesion, post. 1/3 ofLt. vocal cord</td>
<td>forceps</td>
<td></td>
</tr>
<tr>
<td>17.</td>
<td>Walter</td>
<td>1960</td>
<td>a) large cystic bulging, Lt. ventricle</td>
<td>tracheotomy and Lt. laryngectomy</td>
</tr>
<tr>
<td></td>
<td>1960</td>
<td>b) ant. 1/3 of Lt. vocal cord, 2.5×1.5cm</td>
<td>forceps</td>
<td></td>
</tr>
<tr>
<td>18.</td>
<td>Stephen F. Balshi</td>
<td>1960</td>
<td>a) papilloma like mass, Lt. 1/3 of vocal cord</td>
<td>laryngofissure</td>
</tr>
<tr>
<td></td>
<td>1960</td>
<td>b) small round lesion, post. edge of Lt. cord</td>
<td>excision</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1960</td>
<td>c) cystic mass, inner aspect of Rt. arytenoid</td>
<td>local removal</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1960</td>
<td>d) mass, Lt. arytenoid cartilage</td>
<td>cupped forceps</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1960</td>
<td>e) large polyp, Lt. vocal cord and post. surface of Rt. cord</td>
<td>cupped forceps</td>
<td></td>
</tr>
<tr>
<td>19.</td>
<td>Lyons, Haindel, Blatt etc.</td>
<td>1962</td>
<td>a) 3mm nodlar mass, Rt. cord</td>
<td>suspension laryngoscopy</td>
</tr>
<tr>
<td></td>
<td>1962</td>
<td>b) 2 mm polypoid lesion, Rt. vocal cord</td>
<td>?</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1962</td>
<td>c) 3 mm firm red mass, post. commissure and 5mm firm red mass, base of tongue</td>
<td>?</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1962</td>
<td>d) 1 cm sessile flat mass post. 1/3 of Lt. vocal cord</td>
<td>laryngofissure and hemilaryngectomy</td>
<td></td>
</tr>
<tr>
<td>20.</td>
<td>Ward, Oshiro</td>
<td>1962</td>
<td>pearly grayish white, 1.5 to 2 mm sessile nodule near the vocal process of Rt. cord</td>
<td>irradiation and forcep removal under direct laryngoscopy</td>
</tr>
<tr>
<td>21.</td>
<td>Kim, et al.</td>
<td>1963</td>
<td>pearly grayish white 2×2.5 cm pedunculated mass, Lt. ant. 1/2 of vocal cord</td>
<td>snare removal under the suspension laryngoscopy</td>
</tr>
<tr>
<td>22.</td>
<td>Halperin</td>
<td>1964</td>
<td>pale nodular mass measuring about 4 mm on the Rt. cord</td>
<td>forceps</td>
</tr>
</tbody>
</table>
3 cases such as one on the left aryepiglottic fold, one on the left false cord, and the other one on the left ventricle. The size of the laryngeal granular cell myoblastoma is mostly described as a small nodule below 20 mm in size but in two cases were larger than 20 mm, as in our case. The other two cases were Walter's (1960) case of 25×15mm and Lyons, Haindel and Blatt's (1962) 30×20 mm case. Laryngoscopically they appear as benign, well circumscribed, variously infiltrated, polypoid sessile nodular or papillary lesion without ulceration, and appear pearly white, or grayish white color and homogenous nodular or lobulated mass.

**Microscopic picture:** The myoblastoma consists of clumps and strands of round, oval or polyhedral cells of varying size. Specifically they have light staining acidophilic cytoplasmic granules with clear spaces between them with in the cytoplasm. The small round or oval hypochromic nucleus is situated centrally and no mitotic figure are noted. If there is overlying epithelium, it frequently shows marked pseudopitheliomatous hyperplasia, and on section one may see epithelial cell nests or keratin pearls because the strand of cells project in to the surrounding tissue with finger-like projections. For these reasons the less experienced and unwary pathologist may interpret it as a squamous cell carcinoma. Chioldi suggested that a substance may be secreted by the tumor which stimulates epithelial activity and may cause pseudopitheliomatous hyperplasia. Willis feels this is an irritative hyperplasia associated with the possible trauma inflammation, or degeneration. As to the frequency of this hyperplasia, Beckhuis (1960) has reported 12 out of 19 cases and among them 5 cases were initially misdiagnosed as squamous cell carcinoma.

**Histological classification:** There are two types of classification.

A. Abrikosoff's classification (Gouveia, 1960).

1. Typical myoblastenmyome; round oval shaped, large cell with eosinophytic cytoplasmic granules without striation.

2. Matured myoblastenmyome; composed of the first group, showing some longitudinal and transverse cytoplasmic striations indicative of maturation.

3. Hypertrophic myoblastenmyome; composed of large cells measuring 40 to 60 micron in diameter, the majority being multinucleated.

4. Malignant myoblastenmyome or sarcoma; composed of cells without granules and with numerous atypical mitosis characterizing a polymorphonuclear cell sarcoma.


1. Uniform type; composed of sheets or cords of the large granular cells with a small amount of intercellular connective tissue, Thus, as Cappel and Montgomery (1937) had pointed out, most tumors described as Abrikosoff's type 1, 2 and 3 would seem to fall into this group.

2. Pleomorphic type; composed of spindle cell stroma with scattered giant cells which are round, oval, strap or teardrop in shape, with a relatively large amount of granular cytoplasm and more central nuclei, and there are many mitotic figures. Thus Abrikosoff's type 4, falls into this group.

**Benign and malignancy:** There are many controversial opinions concerning the criteria of malignancy in granular cell myoblastoma. All granular cell myoblastoma of the larynx, thus far reported, have been histologically benign though there have been two cases with local recurrence.

Malignant granular cell myoblastoma of other regions of the body have been reported by Gamboa (1955). Azzoparodi (Ward, 1962) considered all tumors reported as malignant to be in actual fact rhabdomyosarcomas or spindle cell sarcoma. On the other hand, Ravich and Stout (1960) feel that the malignant form of myoblastoma represent a polymorphous sarcoma.
According to Howe and Warren (Balshi, 1960), a myoblastoma should be considered malignant if the following criteria are met:

1. Atypism of cells.
2. Excessive number of mitotic nuclei.
3. Spindle cell sarcomatous pattern.

On the other hand, Ross, Miller and Foote (1952) proposed that the diagnosis of malignant granular cell myoblastoma should be restricted to cases that have a clear resemblance to the structural qualities described for tumors in Abrikossoff's first three groups in at least some portion of its primary, recurrent, or metastatic components, otherwise it must be a dubious candidate for inclusion.

**Histogenesis**

The biological nature of the granular cell myoblastoma is controversial. There is even no agreement as to it's being a neoplasm. The non-neoplastic processes considered have included congenital malformation, degeneration and repair, inflammation, and storage disease. Tissues that should be considered in its histogenesis and the related theories are as follows.

**A. Muscle origin theory:** In 1929, Abrikossoff initially propounded the theory that they arose from striated muscle fibers which had undergone post-traumatic degenerative change and were in the process of regeneration. Diss reached a similar conclusion independently, and he and Abrikossoff were supported by later writers. But Klinge, in 1928, reporting the first case of a myoblastoma in the skin where no striated muscle is normally found, proposed a theory of origin from heterotrophic rests of embryonal myoblastoma (dysontogenic). Subsequently, 1931, reporting 7 more cases of myoblastomas, Abrikossoff was forced to accept Klinge's theory of origin for the myoblastomas arising where no striated muscle is normally found. But retained his belief in a degeneration-regeneration process for those found in skeletal muscle.

A number of subsequent observers followed Abrikossoff's explanation, given in 1931, accepting both the dysontogenetic and the degeneration-regeneration origin theory. In particular, the three cases of tissue culture study of Murry, in 1951, seems to bear this theory out. But Murray (1951) does not agree with Abrikossoff's degeneration-regeneration theory and agrees with Khanolkar (Murray, 1951) who says: "The close resemblance between the cellular constituents of these tumors and the forms encountered during the regeneration of voluntary muscle does not necessarily imply an absence of neoplastic growth, but may indicate the various phases during the course of a differentiation of tumor cells". Murray (1951) also cited Ravich and Stout's metastatic malignant case as a support of the neoplastic origin theory.

Those who follow the Abrikossoff's skeletal muscle origin theory point out the following points as confirmatory evidences (Murphy, 1949):

1. The tumor cells have a granular cytoplasm similar to that of early myoblasts.
2. An arrangement of the cytoplasmic granules in rows suggestive of young myofibrils has been noted.
3. Some of the granular cells have shown actual cross and longitudinal striations.
4. Numerous instances have been reported of apparent direct transition of the muscle fibers to granular cells.
5. The limiting membrane of the granular cells frequently is absent, giving the appearance of the syncytial formation as in the normal histogenesis of striated muscle.
6. In myoblastoma of the tongue, the granular cells are found immediately beneath the surface epithelium where no muscle is ordinarily found. This supports the theory of neoplasia as against that of simple degeneration.
7. Further presumptive evidence of neoplasia is seen in the local invasiveness, lack of capsule formation, and recurrence of the tumor following apparently adequate excision.

On the other hand, many others are against the skeletal muscle origin theory for the following reasons (Bangle, 1952).

1. Morphologically and histochemically identical lesions are found in the breast and skin where striated muscle normally is absent.

2. If the tumor arises from embryonal rests of aberrant muscle, one should find cells resembling myoblasts. Such cells are not present.

3. Except for congenital rhabdomyomas of the heart, all tumors known to arise from striated muscle are highly malignant. Therefore it seems improbable that a tumor arising from undifferentiated striated muscle should as a rule be benign.

4. The demarcation between striated muscle and the tumor usually is distinct. Not only is the sarcolemma intact, but also the connective sheath about the granular fascicles. For these reasons, the term "myoblastoma" is inadequate, but until the pathogenesis of the tumor is proved, it is best to retain this term in order to avoid confusion.

B. Neural origin theory: This theory is first proposed by Fust and Custer (Gouveia, 1960) on the following evidence. In 1948, Fust and Custer examined fifty two cases of granular cell myoblastoma. Among them, thirty three cases (5 lingual and 28 from other sites) were often intimately related to the axis cylinders of peripheral nerves and they appeared to arise in the internal connective tissue (possible Schwan’s cell) of the latter structures. The lesion in the remaining 19 cases in which this neural relationship was not found were identical morphologically with those in which it was present. So they proposed that it is more correct to call it a granular cell neurofibroma than a granular cell myoblastoma.

On morphological and histological study of this tumor, Bangle (1952) pointed out the following evidences as support of the neural origin theory. Among 43 cases. histologically 40 per cent had no relation with skeletal muscle and 32 cases (74 per cent) were associated with a peripheral nerve, and morphologically could be classified in two patterns.

The first pattern has granular cells concentrically arranged in whorls of varying thickness about a core of intact myelinated nerve fibers. The perineurium of this nerve was so disrupted that it was no longer discernible as such. The interstitial between granular cells often contained irregularly fragmented strungent strands of material that was stained a bright red by the allochrome procedure. This material could represent the disrupted perineurium.

The second pattern was that presented by the appearance of granular cells within nerve fascicles. In cross section, this replacement of nerve fiber by granular cells partial or complete, whereas in longitudinal section it was partial involved in a segment of a nerve branch.

These two neural patterns were present singly or together in different portions of the same tumor. Perhaps an important observation was the presence of these patterns of neural involvement located at a distance from the body of the tumor. By studying serial sections it could be observed that the granular cells or granular synctium followed a perineural and some times intraneural course that eventually became continuous with the main tumor. Thus, what appeared to be isolated nests of granular cells at a distance from the main tumor mass were, in reality, continuous with this mass and often could be traced along the course of nerve twigs. However, there is no agreement as to the specific cells
involved, perineural and intraneural fibroblasts, Schwann cells, histiocytes, vascular endothelial cells. The granular material of the myoblastoma could be derived from the degeneration of myelin sheath and axis cylinders. That none of the granules stained as does normal myelin may be due to the complete resorption of the initial phase of which has escaped detection.

Pearse (Maguda, 1953) believes that fibroblasts, including those of the perineurium and endoneurium, undergo a granular degenerative process and thereby are transformed into the myoblastoma cells. However, fibroblasts have never been known to undergo granular degeneration or to be transformed into cell types other than chondrocytes and osteocytes. Thoren has had two cases of myoblastoma analysed chemically and pointed out that, in generally, there was a good correlation between the lipid content of the myoblastoma and that of the splenic nerves of the cow.

Causey (Burston, 1962) using the electron microscope to examine the components of a peripheral nerve within the perineurium found that 95 per cent of the cells present are certainly either Schwann cells or vascular endothelial cells. Considering this evidence and their own experience J. Burston, R. J. John and H. Spencer (1962) think that the granular cell myoblastoma cells may be derived from the Schwann cell rather than from some mesenchymal cell within the perineurium. Masson believes that in some neurofibromas the Schwann cells become granular without giving rise to tumors and that these cells are degenerative and there is a striking resemblance between these cells and those of the myoblastoma. With this evidence, he proposed the degenerative Schwann cell theory. Ashburn (1952), Rodger (1952), Gamboa (1955), Herbut and Feyster are also in agreement with this theory.

C. Histiocyte origin theory: In 1939, Leroux and Delarue proposed glycogen storage histiocyte theory and they said that the tumor was formed by a local accumulation of these histiocytes as a manifestation of general metabolic process, and they said that the storage material might be a glycogen. As to the storage material, there are many other opinions. Holle says that it is a mucous storage histiocyte; Ringertz says it contained the product of protein breakdown material; and Lauche, with a negative staining result for fat and mucin, expressed the belief that the granular materials was of protein nature and is analogous to xanthoma.

Besides the above mentioned theory, Gray and Gruenfeld proposed a glandular cell origin (degeneration of the glandular epithelium) theory; Khanolkar postulated the mesenchymal cell as the cell of origin. As Maximow and Bloom (1942) had pointed out, this totipotential mesenchymal cell is widely dispersed throughout the body in the vicinity of small blood vessels. Nicholson and Willis proposed that it is a metaplasia from primitive mesodermal elements, while others proposed a smooth muscle origin theory or an epithelial or myoepithelial origin theory.

Diagnosis

Clinically this tumor is a slow growing painless benign tumor. In the larynx, there can be hoarseness, cough, stridor, dyspnea and occasional hemoptysis as with other laryngeal tumors. Occasionally there are no symptoms that can be found on routine check or upon endoscopic examination for other respiratory tract trouble. The diagnosis is made by indirect or direct laryngoscopy and confirmed by histological examination. Clinically it has to be differentiated from other benign tumors such as polyp, papilloma and singer's nodules. The histologically uniform type must be differentiated from squamous cell carcinoma, and the pleomorphic type from rhabdomyosarcoma.
Treatment and Prognosis

Among thirty two cases in which treatment in mentioned the following were used. Radiological trial failed to have any effect. Twenty five cases were removed locally. In addition, there were three cases of laryngofissure, two cases of laryngofis-
sure and hemilaryngectomy, one case of laryngofis-
sure and laryngectomy and one case of laryngect-
omy. For the remaining two cases of Lyons, Haindel and Blatt there is no mention of treatment.
Considering that there were only two cases of recurrence after local removal, it seems to be enough to treat laryngeal granular cell myoblastoma locally. The post operative care is the same as the other benign laryngeal tumors and follow up the cases for at least three years is indicated.

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