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(CASE REPORT)

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Granular cell tumor of the esophagus: Case report and a brief review of the literature

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Abstract

Granular cell tumors (GCTs) of the esophagus are rare, typically benign neoplasms of Schwann cells that are often overlooked in the differentiation of submucosal lesions of the esophagus. They can have a clinical presentation similar to more common structural or malignant causes of dysphagia, making their diagnosis and treatment delayed. We present a case of a 36-year-old woman with progressive symptoms of dysphagia and weight loss. The symptoms lasted for several months and were ultimately diagnosed as a benign GCT of the lower esophagus. Multimodal imaging studies suspected a benign submucosal lesion, which was biopsied and later excised. The histopathology with immunohistochemistry (IHC) assistance confirmed the diagnosis. The case emphasizes the importance of a broad differential in the workup of submucosal masses of the esophagus in younger patients with atypical or nonspecific symptoms. Early GCT diagnosis and minimally invasive management can be provided by considering the GCTs early. It is crucial to keep reporting such cases to increase the awareness of clinicians and improve the diagnosis of this rare entity.

Keywords: Granular cell tumor; Benign; Differential diagnosis; Dysphagia; Esophagus

1. Introduction

Granular cell tumors (GCTs) of the esophagus are uncommon benign neoplasms that are thought to arise from Schwann cells. [1] [2] Although GCTs may develop in any region of the body, their location in the esophagus is uncommon, and thus, may present a diagnostic challenge because of their rarity and nonspecific clinical manifestation. [3] [4] Most of the GCTs of the esophagus are asymptomatic and are diagnosed incidentally during an endoscopic procedure done to investigate other conditions. [5] When they present with complaints, the typical symptoms are vague retrosternal pain or discomfort and, at times, dysphagia, a feature of the slow-growing character of these submucosal lesions. [6] [7]

Imaging tests, including barium swallow and computed tomography (CT), are usually used in the diagnostic procedure of esophageal GCTs and can show a submucosal mass. [8] Endoscopic ultrasound (EUS) has become central in further defining these lesions, giving information on their size, location, depth in the wall of the esophagus, and in directing acquisition of tissue for tissue diagnosis. [9] Histopathological evaluation of biopsy specimens is, however, definitive in diagnosing the disease because it shows characteristic polygonal cells with excessive granular eosinophilic cytoplasm and a small nucleus. [10] Confirmation is important, and immunohistochemistry (IHC) is essential; GCTs are strongly positive for S-100 protein and CD68 and are distinguished by these markers, compared to other possible submucosal lesions. [11]

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The main method of treatment of esophageal GCTs is through local excision. Endoscopic mucosal resection (EMR) and endoscopic submucosal dissection (ESD) represent minimally invasive endoscopic methods, which have become the treatment of choice for most benign lesions due to their capacity to achieve effective removal with good outcomes and low recurrence. [11] [12]. Surgery is normally applied in larger, more complicated, or suspected malignant cases. [4] Since GCTs are rare and sometimes may be subtle, clinicians' and pathologists' awareness is critical to achieve timely and accurate diagnosis, which will enable proper management and better outcomes. The case report is expected to add further to the existing knowledge of esophageal GCTs with a brief literature review.

2. Case presentation

A 36-year-old female presented to the outpatient clinic with a several-month history of progressive worsening of dysphagia. Her first issue was that solid food could not go down her throat, and this was followed by liquids. She described it as food getting stuck in her chest, and she would frequently have to cut her bites smaller, and certain food textures were bothersome. Nonspecific retrosternal pain was also reported by her, especially during and after meals, but she denied heartburn, odynophagia, or regurgitation. She noticed unintentional weight loss in the last few months, leading to further medical evaluation.

Her physical examination was unremarkable. Complete blood count, comprehensive metabolic panel, and liver function tests done periodically were normal on several occasions, which made her and her primary physician feel at ease that there was nothing to worry about. The primary physician started more investigations with the persistence and ever-increasing symptoms. A barium swallow showed a smooth, well-defined filling defect in the lower esophagus, showing a napkin-ring filling defect, causing suspicion for an esophageal submucosal mass.

EUS showed a 1.3 cm homogeneous, hypoechoic submucosal mass. The retained layer structure and clear margins of the mass were in favor of a benign lesion. These results were confirmed by contrast-enhanced CT imaging, which showed a well-defined, moderately enhancing mass, isodense to the adjacent muscles, without invasion or regional lymphadenopathy. Based on the size and imaging features of the lesion, it was decided to perform an endoscopic biopsy to get a definite histological diagnosis. Endoscopy revealed a lower esophagus submucosal lesion with an intact overlying mucosa. Tissue sampling was obtained. The biopsy had histopathological changes of pseudoepitheliomatous hyperplasia of the overlying squamous mucosa and a well-defined proliferation of large, solid sheets of polygonal cells. The tumor cells showed a large eosinophilic and granular cytoplasm. The nuclei were small, oval to round, central, and minimally pleomorphic. Mitotic figures were low, less than 1-2 per 10 high-power fields. No necrosis or cellular atypia was noted. (Figure 1A, B)

The differential diagnosis included a variety of granular cytoplasmic entities. Alveolar soft part sarcoma, despite cytological similarity, was not considered because it usually appears in younger patients and has an alveolar structure. Rhabdomyoma and adult rhabdomyosarcoma were also ruled out due to the lack of cross-striation and myogenic markers. Oncocytomas and oncocytic carcinomas were also ruled out because the granularity present in such tumors is due to the presence of mitochondria and not lysosomes. Amelanotic Melanoma was also ruled out based on immunohistochemical analysis. A diffuse, strong positivity of S-100 protein by IHC was noted, compatible with Schwann cell origin. The diagnosis was supported by additional positivity of CD68, inhibin, and neuron-specific enolase (NSE). It was useful that the tumor was negative for Melan-A, HMB-45 (excluding Melanoma), and myogenic markers (desmin, myogenin, MyoD1) (excluding rhabdomyosarcoma). Pan-cytokeratin was negative as well, and not in support of carcinomatous origin.

The results revealed the presence of a GCT of the esophagus, which was benign and did not exhibit any malignancy. The 1.3 cm mass was endoscopically removed entirely and with negative margins. The pathology of the excised mass confirmed the biopsy results, and no atypia, necrosis, or high mitotic activity was observed. The patient experienced no postoperative complications, and gradually all symptoms disappeared. At the 3-year follow-up visit after surgery, the patient had no complaints, and there was no evidence of recurrence of complications.

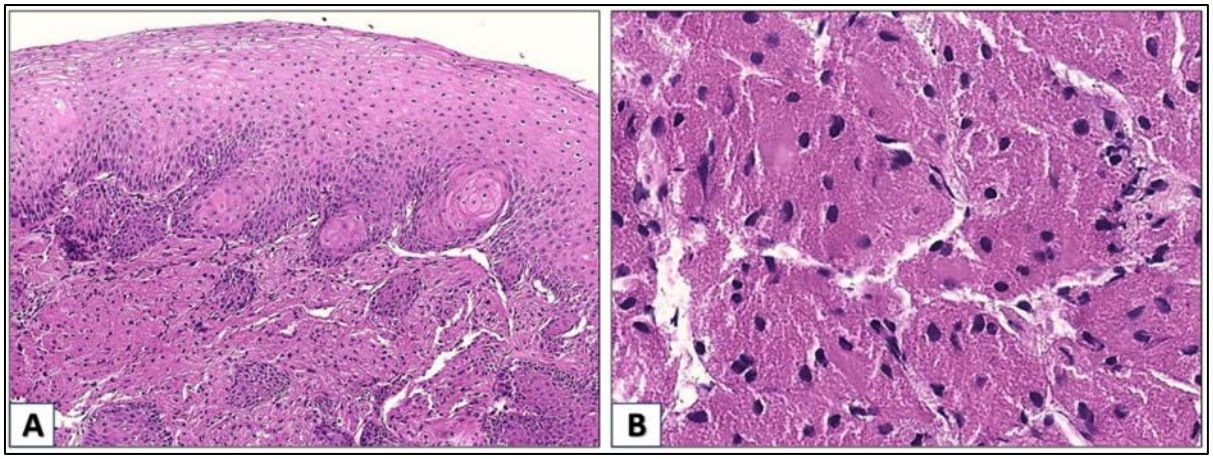

Figure 1 Histomorphology of esophageal granular cell tumor (GCT)

- **1A** Low power view showing intact squamous mucosa with basal cell hyperplasia and submucosal solid sheets of granular cell tumor (H&E stain X20)
- **1B:** High-power view showing individual cells of granular cell tumor. The cells show a large eosinophilic and granular cytoplasm. The nuclei are small, oval to round, and central, and are minimally pleomorphic (H&E stain X60)

3. Discussion

3.1. History, epidemiology, risk factors, and WHO classification

Esophageal granular cell tumors (GCTs) are uncommon, neurogenic neoplasms of Schwann cell origin from the submucosal neuronal plexus. These tumors were initially reported on the tongue by Abrikossoff in 1926 and on the esophagus in 1931. [2] The lesions are most often diagnosed in the fourth to sixth decades of life, with a minor female predominance (female-to-male ratio: 1.8–2.4:1). [13] The reported median and mean ages are 43 and 44.00 ± 3.48 years, respectively, however, they can occur in all age groups. Demographic patterns vary by population. [14] Historically, two-thirds of benign GCTs have been documented in African-American patients, whereas malignant cutaneous cases predominantly affect Caucasians. [2]

Typically, GCTs are solitary, up to 29% present with multiple lesions, with approximately 30% involving the subcutaneous tissues or skin. [11] Distribution patterns show 45–65% in the head and neck, 15% in the breast, 30% in cutis/subcutis, 10% in the respiratory tract, and 5–11% in the gastrointestinal tract. [10] Malignancy is reported in only 1–2% of cases. [13] The World Health Organization (WHO) classifies GCTs as benign soft tissue tumors without a site-specific category for esophageal lesions. Even though they are usually indolent, some of them are atypical histologically or frankly malignant. [1]

3.2. Clinical presentation and imaging findings

Granular cell tumors (GCTs) of the esophagus are not very common, and their presentation may be highly variable, which causes delays in the definitive diagnosis. Traditionally, most esophageal GCTs lack symptoms and are detected accidentally during an endoscopy that was done due to unrelated reasons. [5] [7] [15] When the symptoms appear, they are usually nonspecific and insidious in their development, which indicates the low rate of growth of these benign lesions. [1] [Benchekroun 11] Retrosternal discomfort or pain. [7] [11] is the most reported symptom and is often vague and continuous. The other common complaint is dysphagia or the inability to swallow, but it is not as prevalent as pain. [7] Patients might report the feeling that food is stuck in the chest, making them change their eating behavior, such as eating smaller bites or not consuming certain food textures. Epigastric pain, nausea, or globus sensation may also be present in other less typical cases. [1] [11]

Imaging is important in the early diagnosis and characterization of esophageal GCTs, but final diagnosis usually needs histopathological confirmation. A smooth and well-defined filling defect of the esophageal lumen, which can appear as a napkin-ring or apple-core and is suspicious of a submucosal mass, and may be demonstrated by barium swallow studies. [8] Nevertheless, such a finding is nonspecific and should be studied further. EUS can be regarded as the most

useful imaging study in assessing esophageal GCTs. [11] Usually, EUS will show a homogenous hypoechoic lesion located in the submucosal or muscularis propria layers [11]. The significant characteristics that may indicate a benign GCT in EUS are an intact layered structure of the esophageal wall and intact margins, implying the non-invasive character [8]. These can be further confirmed using contrast-enhanced computed tomography (CT) scans in which a well-defined, moderately enhancing mass with no evidence of invasion into surrounding structures or regional lymphadenopathy is usually noted to be isodense with the surrounding muscle [8]. Although imaging can highly indicate the existence of a benign submucosal tumor, the confirmation of an esophageal GCT is based on the biopsy and a consequent histopathological analysis with IHC studies.

3.3. Pathogenesis

GCT is neuronal in origin and may be confirmed with positive IHC markers, including S-100 protein, SOX10, neuron-specific enolase, CD68, calretinin, inhibin, and nestin. The presence of eosinophilic granular cytoplasm in tumor cells also characterizes the tumor. This patient's neoplasm was positive for S-100, CD68, inhibin, and neuron-specific enolase. Both the American Society for Gastrointestinal Endoscopy and the American Gastroenterological Association recognize that most Granular cell tumors are typically benign and have a Schwann cell origin, marking the importance of histopathologic and immunohistochemical confirmation for diagnosis. [17] [18] The pathophysiology of Granular cell tumors involves the formation of a submucosal mass, which can be found anywhere along the GI tract, but is most commonly in the middle or distal esophagus. The tumor is typically firm, well-defined, and covered with normal-looking mucosa when it is detected by endoscopy**.** [14] [17] Malignancy was generally reported in lesions that were larger than 2-4 cm. [17] [19] An association has been found between esophageal granular cell tumors, Crohn's disease, and eosinophilic esophagitis. This may suggest that a chronic state of esophageal inflammation can be a potential link between the two; however, the causal relationship is still unclear. [20] [21]

3.4. Laboratory Findings, pathology findings, immunohistochemistry, and molecular studies

The common laboratory results in patients with esophageal GCTs are not specific and typically within the normal range since these tumors are typically benign with no systemic effects that would be reflected in a routine blood test. [15] [8] As an example, the complete blood counts, comprehensive metabolic panels, and liver function tests are mostly unremarkable. The presence of any abnormalities, including mild anemia or weight loss, tends to be secondary to such symptoms as dysphagia, rather than the direct effects of the tumor itself. [10] Thus, the main role of laboratory research is to exclude other systemic pathologies or complications instead of direct diagnosis of GCT.

The clinical course of esophageal GCTs usually starts with endoscopic examination, typically indicated by nonspecific symptoms or an accidental finding. Although endoscopy can detect a submucosal lesion, usually yellowish and firm with intact overlying mucosa, it is not conclusive in the diagnosis of a GCT. [2] [14] The subsequent important procedure is EUS, which gives important details of the size, location, and depth of origin of the lesion in the esophageal wall. [2] [13] EUS can also be used to direct biopsies. The submucosal location of GCTs may make traditional endoscopic biopsies difficult, and the result may be only overlying normal mucosa or insufficient tissue. [14] Thus, more invasive biopsy methods, e.g., EUS-guided fine-needle aspiration (FNA) or mucosal incision-assisted biopsy (MIAB), are frequently required to obtain adequate tissue to make a histopathological diagnosis. [2] [13]

Esophageal GCTs have typical microscopic features. They can be made up of nests or sheets of large, polygonal cells that contain much eosinophilic, granular cytoplasm and have small, frequently pyknotic, oval to round nuclei. [8] [11] Benign GCTs usually lack mitotic figures or have only a few of them, and no necrosis or substantial cellular atypia. [10] The granular look of the cytoplasm is owed to the many lysosomes. A frequent accompaniment is overlying pseudoepitheliomatous hyperplasia of the squamous mucosa that may be confused with squamous cell carcinoma unless it is critically assessed. [10]

IHC studies are essential to differentiate GCT from other mimics. Because they originate from Schwann cells, GCTs are diffusely positive for the S-100 protein. [11] [22] They are also positive for CD68 (lysosomal marker), beta-enolase, and inhibin-alpha. [11] [22] Furthermore, GCTs are negative with cytokeratin (epithelial), melan-A, HMB-45 (melanocytic), desmin, myogenin, and MyoD1(muscle markers), differentiating them from other tumors such as carcinoma, Melanoma, or rhabdomyosarcoma. [10] [11] [22]

Diagnosis of benign esophageal GCTs is often made on typical histopathological and immunohistochemical findings, so the use of molecular tests for their diagnosis or treatment is less frequent. [13] Given that esophageal GCTs are usually benign and curable by excision, extensive molecular workup of anatomic pathology is generally not required.

3.5. Treatment and outcome

Treatment of esophageal GCTs is mainly based on complete local excision, as they are usually benign and of low malignancy potential. The treatment modality is determined by the size, location, and endoscopic features of the tumor, the presence of symptoms, and whether it is to be treated with surgery or not. Endoscopic methods are preferred to surgery since they are less morbid and highly effective in completely resecting most benign esophageal GCTs. [6] [11] [12] The two most popular endoscopic techniques include the endoscopic mucosal resection (EMR) and endoscopic submucosal dissection (ESD). EMR can, in many instances, be used in the smaller, more superficial lesions, especially those of submucosal origin. EMR has been successfully performed using various techniques such as ligation-assisted, cap-assisted, and conventional EMR. [6] [23] In contrast, ESD enables en bloc resection of bigger lesions and those arising deeper in the muscularis propria, with a more accurate and complete resection that is essential to accurate histopathological evaluation and to reduce recurrence. [12] [24] The use of ESD is becoming more popular because it enables a complete resection even of submucosal malignancies [12] [17].

Surgical resection (e.g., esophagectomy) is usually reserved for situations where endoscopic removal is not possible, when there is a very large tumor, suspected malignancy, or when there is evidence of deep invasion into the surrounding organs or structures. [11] Nevertheless, due to the development of endoscopic treatment methods, the indications for surgical treatment have been limited to a considerable degree. After resection, routine endoscopic follow-up should be considered to look out for recurrence, but the chance of recurrence of fully removed benign GCTs is quite low. [6] [11]

The prognosis of benign esophageal GCTs is good when the mass is completely removed through appropriate management and procedures. [23] Close endoscopic follow-up is advised, especially in tumors less than 2 cm or multifocal tumors. The follow-up periods are usually 6-12 months in the first year, followed by annual follow-up, primarily to identify recurrence or new lesions. [7] Larger tumor size >2-4 cm, rapid growth, necrosis, high mitotic rate, and cellular atypia were other features raising concern about malignancy. [7] [23] These features were absent in our case.

3.6. What did we learn from this case?

This case highlighted the significance of the differential diagnosis of esophageal submucosal lesions that occur in young patients with progressive nonspecific symptoms. When diagnosed early and treated minimally invasively, full resolution of the symptoms and a good long-term outcome can be achieved. The initial barium swallow imaging in our case showed a nonspecific filling defect, and further investigation was done. Endoscopic ultrasound (EUS) was very helpful as it accurately described the 1.3 cm homogeneous, hypoechoic lesion in the submucosa with maintained layer structure and clear borders that were highly suggestive of a benign submucosal tumor. This agrees with the common opinion that EUS is the best modality to evaluate GCTs and to determine further management. These findings were further supported by contrast-enhanced CT, which revealed a well-defined, moderately enhancing mass without evidence of invasion or lymphadenopathy, and in keeping with a benign nature as depicted in imaging literature.

The histopathologic analysis, in combination with IHC, was essential to the definitive diagnosis, as it was emphasized in the literature. Pseudoepitheliomatous hyperplasia of overlying mucosa, as well as proliferation of polygonal cells with abundant eosinophilic granular cytoplasm, small nuclei, and low mitotic activity, are typical microscopic features of GCTs. The diffuse positivity of S-100 protein, CD68, inhibin, and neuron-specific enolase (NSE) with negativity of epithelial, melanocytic, and myogenic markers was indicative of a Schwann cell origin. It ruled out other differential diagnoses as well, in line with the known IHC profiles of GCTs. Our patient also fits the literature in normal laboratory values since GCTs do not generally cause systemic abnormalities in laboratory values.

Regarding treatment, the endoscopic resection that was employed signifies the state of the art in the treatment of benign esophageal GCTs. EMR or ESD is a less morbid, more effective, minimally invasive procedure. This endoscopic intervention has been successful in curative resection of lesions of this size, as evidenced by the total resection of the 1.3 cm mass with negative margins. The excellent prognosis and absence of recurrence at the 3-year follow-up of our patient are consistent with the very favorable prognosis of completely resected benign GCTs of the esophagus described in the literature. The importance of timely diagnosis and management in this case also supports the need to offer an excellent prognosis to patients of this rare entity in the long run.

4. Conclusion

This case report of esophageal granular cell tumor highlights the necessity of a wide differential diagnosis of submucosal lesions of the esophagus, especially in younger patients with atypical and nonspecific symptoms such as dysphagia and weight loss. Although they are not common, early diagnosis of GCTs may be achieved by prioritizing them in the early

stages through proper imaging and histopathology tests, including essential immunohistochemical analysis. Our case shows that complete resection with good long-term outcomes is possible using minimally invasive endoscopic management. Further documentation of these cases is essential to increase awareness among clinicians and pathologists, which will eventually lead to better diagnoses and patient care of this rare tumor.

Compliance with ethical standards

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Disclosure of conflict of interest

All authors make the following declarations:

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- **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might be interested in the submitted work.

Statement of ethical approval

Ethical review and approval were not required for this study involving human participants. The paper has been sufficiently anonymized to maintain the patient's confidentiality.

Statement of informed consent

The patient's consent was obtained for the publication of this case report.

Data access statement

All relevant data are included in the paper.

Author contributions

All authors contributed equally to producing this manuscript.

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