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(CASE REPORT)

Goblet cell carcinoid, now goblet cell adenocarcinoma, masquerading as acute appendicitis: Case report of an uncommon clinical presentation and a brief review of the literature

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Abstract

A goblet cell carcinoid (GCC) is an appendiceal neoplasm with mixed neuroendocrine and glandular differentiation that is uncommon and most typically affects middle-aged adults. The terminology of this tumor has recently been changed to goblet cell adenocarcinoma (GCA) to indicate the malignant behavior of this tumor, which is more than that of a neuroendocrine tumor (NET). The WHO has removed the term “carcinoid” in naming tumors in its 2022 classification.

In this report, we present a 42-year-old man who was admitted to the hospital with complaints of severe pain in the right lower abdomen, fever, and elevated white blood cells, which were the common symptoms of acute appendicitis. He had a scheduled appendectomy and thought that he would recover like any other normal case. However, what was unexpected was the pathology report, which showed goblet cell adenocarcinoma (GCA), a rare and aggressive tumor that was previously known as GCC. In contrast to the general NETs, GCC acts rather like an adenocarcinoma, inclining to invade the adjacent tissues and metastasize. Here, the tumor has extended beyond the appendix and invaded the surrounding fat (T3N0M0, Stage IIA). The fat invasion also necessitated continued treatment, whereas the lymph nodes and distant organs were not affected. The carcinoid/adenocarcinoma terminology transformation signifies the actual nature of the tumor, which is more of a rapidly progressing cancer that needs vigorous treatment rather than a NET with a slow growth rate.

This case serves as a reminder to keep a wide differential diagnosis, even in classical presentations, such as appendicitis. Cross-disciplinary tumor board discussion was key in decision-making regarding the treatment plan. Four years after surgery, the patient is still disease-free. We provide a brief literature review on this uncommon tumor and clarify the recent change in classification terminology.

Keywords: Appendix; Goblet cell carcinoid; Goblet cell adenocarcinoma; Molecular; Neuroendocrine tumors

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1. Introduction

Appendiceal Goblet Cell Carcinoid (GCC), now goblet cell adenocarcinoma (GCA), is a complex disease and is more aggressive than the typical appendiceal well-differentiated neuroendocrine tumors (NETs) (carcinoids). [1] WHO has removed the term "carcinoid" in naming tumors in its 2022 classification. The current classification of the WHO no longer uses Goblet Cell Carcinoid (GCC). All so-called carcinoid tumors are currently grouped as well-differentiated neuroendocrine tumors (NETs), which are graded according to mitoses and Ki-67. GCC is now called GCA, and is no longer regarded as a NET. This transformation represents its actual malignant epithelial character, which puts it in the same category with other adenocarcinomas and not in the category of NETs, even though it is a mixed phenotype. GCC staging does not follow NET staging systems but adenocarcinoma protocols. [1] [2] [3] In this report, we are using the term "Goblet cell carcinoid" because this case presentation was before the new classification.

GCC oftentimes presents as acute appendicitis. Because GCCs are discovered incidentally during routine appendectomy, there is a gap in the identification and accuracy of markers used to diagnose this neoplasm. [4] There is a call to action in this misdiagnosis as the number of GCC diagnoses continues to rise. This rare tumor lodged in the appendix contains both epithelial and neuroendocrine features. The clinical presentation varies, but it most commonly presents with chronic abdominal pain and a palpable pelvic mass. [5] GCC is a rare finding, comprising 14% of malignant appendix neoplasms. [5] Although studies have indicated the expression patterns of CEA, CDX2, CK7, and CK20, which closely resemble colonic adenocarcinomas, GCC most frequently arises in the appendix. Histologically, this neoplasm involves the entire circumference of the appendiceal wall, characterized by small, rounded nests of signet ring-like cells resembling intestinal goblet cells. [9]

We present a case of a 42-year-old man who presented with appendicitis and was diagnosed with GCC after appendectomy. We briefly review the literature on GCC and clarify the new classification as GCA.

2. Case presentation

A healthy man, 42 years of age, with no previous medical problem, was admitted to the emergency department for two weeks of lower abdominal pain, progressively growing worse in the last 48 hours. Pain was first dull, but progressed into a localized irritation in the right lower quadrant. He had aching, steady discomfort, which would at times increase with movement. He began to feel nauseous and vomited multiple times over the past two days, accompanied by a feeling of general malaise. He had only mild fever, no altered bowel habits, no urinary symptoms, no recent travel, and no contact with ill persons. His past medical history was unremarkable, and his family history was also negative for gastrointestinal or oncologic disorders.

The patient was alert but slightly distressed at presentation on physical examination, secondary to pain. The patient's vital signs were within the normal range. On abdominal examination, there was localized tenderness in the right lower quadrant with minimal guarding. Acute appendicitis was suspected. However, with prolonged symptom duration, further work-up was advised. Laboratory tests showed a mildly increased white blood cell count. Tumor markers, including carcinoembryonic antigen (CEA) and CA 19-9, were evaluated for the suspicion of atypical or neoplastic process. These markers were mildly raised, leading to a carefully worded interpretation but sufficient anxiety to proceed with imaging.

Abdominal ultrasound demonstrated a thickened appendiceal wall at more than 6 mm with loss of its normal layered pattern. A small, hypoechoic nodule was seen near the appendix tip. No free fluid or peritoneal nodules were noted. CT abdomen and pelvis revealed a focus of soft tissue thickening in the distal appendix, which was approximately 1.5 cm in size. Mesenteric lymphadenopathy was absent, as well as ascites and peritoneal carcinomatosis. MRI further defined the lesion as a T2-hyperintense nodule in the distal appendix with restricted diffusion on DWI/ADC facilitated images, which was consistent with high cellularity and possible mucinous content. The patient was subjected to a laparoscopic appendectomy, which was uneventful based on both the clinical and imaging evidence. At operation, the appendix was noted to be slightly inflamed and fibrotic, with a firm nodule at the tip. There was no definite gross perforation and no peritoneal disease.

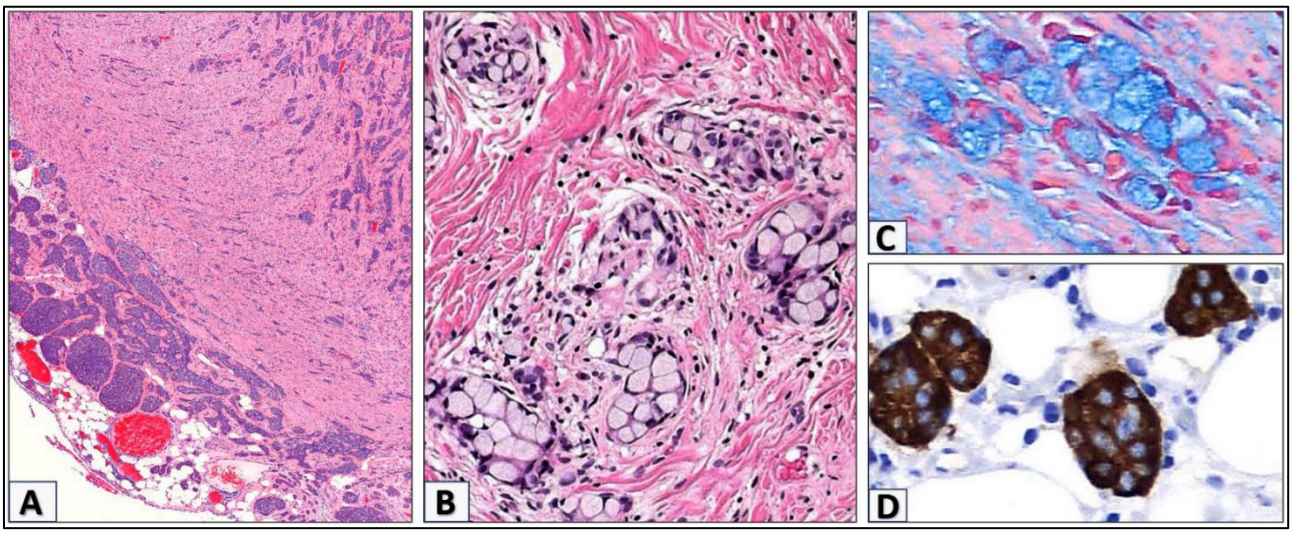
Grossly, the appendix contained a firm nodule measuring 1.4 cm in greatest dimension just below the mucosa, involving the entire circumference of the appendix with extension into the periappendiceal fat, but not beyond. Microscopic evaluation of the resected specimen revealed an invasive mass consisting of clusters and small nests of goblet-like cells, most of which had classic signet-ring morphology with abundant intracytoplasmic mucin displacing the nuclei to the periphery. The neoplastic cells invaded the muscularis propria, subserosa, and mesoappendix. A desmoplastic stromal reaction encircled the tumor nests. Nuclear pleomorphism was minimal with low mitotic count (<2 mitoses/10 high-

power fields). The population of the cells was a mixture of goblet cells, Paneth-like cells, and intermediate cells, which showed a biphasic pattern, as is characteristic of a goblet cell carcinoid tumor. (Figure 1 A, B, C, D) Pathology differential diagnosis included adenocarcinoma of the appendix (mucinous or signet-ring cell type), metastatic signet-ring cell cancer (especially gastric or pancreatic origin), classical appendiceal carcinoid tumor, and mucinous neoplasm (low-grade appendiceal mucinous neoplasm). Alcian blue histochemistry stain was positive, confirming the acidic mucin usually present in goblet cells. Histology was compatible with localized GCC of the appendix, but for definitive diagnosis, a large IHC panel was performed.

The tumor cells were positive for chromogranin A and synaptophysin, which indicates the existence of a neuroendocrine component. MUC2 was positive, indicating production of goblet cell mucin. CK20 was positive, and CDX2 was weakly positive, suggesting the presence of an intestinal epithelial component. The Ki-67 proliferation index was low (7%), compatible with slowly proliferating tumor cells. Of note, the tumor cells were CK7 negative, which is not in support of gastric or pancreatic origin, and TTF-1 and GATA3 negative, which ruled out pulmonary and urothelial primary. The biopsy results supported a diagnosis of primary GCC of the appendix with spread to the periappendiceal fat, consistent with T3 stage.

This was an unexpected diagnosis, and a multidisciplinary tumor board discussion took place. The patient's age, the tumor's biological behavior, and the well-known oncologic management rules for goblet cell carcinoid were considered and analyzed by the team. Although some suggested diagnostic laparoscopy to exclude microscopic peritoneal disease, most of the team opted for primary right hemicolectomy. Reasoning incorporated the tumor size (greater than 1 cm), the histology (GCC known to have a higher metastatic potential than the classic well-differentiated NETs before being classified now as GCA), evidence of periappendiceal invasion, and the fact that negative imaging could not be completely accurate to exclude microscopic metastasis. This case was diagnosed before the recent clarification by the WHO classification of appendiceal tumors, 5th edition, 2022, where the so-called carcinoid tumors are currently grouped as well-differentiated neuroendocrine tumors (NETs), which are graded according to mitoses and Ki-67.

The patient had an uneventful right hemicolectomy. Histological examination of the resected colon specimen revealed no residual tumor, a sufficient free surgical margin, and metastasis was not detected in all sixteen dissected regional lymph nodes. In the absence of peritoneal, regional, or distant dissemination, the final stage was recorded as Stage IIA (T3N0M0). No additional treatment was considered necessary in the postoperative setting because of the completeness of resection, negative nodal status, and low proliferative index. The patient was on a carefully monitored protocol of clinical examinations and thoracoabdominal and pelvic CT scans every 3–6 months for the first year, and less frequent intervals thereafter. At the last follow-up, 4 years after the initial presentation, the patient continued to be well, with no evidence of disease recurrence or progression on clinical, radiological, or biochemical evaluation. The patient has returned to a normal life.



1A Low-power view showing an invasive mass consisting of clusters and small nests of tumor cells. The neoplastic cells invade the muscularis propria, subserosa, and mesoappendix (H&E stain X20); **1B:** High power view showing clusters of neoplastic goblet cells with signet ring features (H&E stain X60); **1C:** Tumor cells positive for alcian blue;**1D**: Tumor cells positive for cytokeratin and CD20

Figure 1 Histomorphology of goblet cell carcinoid (GCC), now (GCA) of the appendix

3. Discussion

3.1. History, epidemiology, and risk factors

GCC was first described in 1969 by Gagné et al, when they described three cases of unusual appendiceal tumors characterized by the presence of both neuroendocrine cells and mucus-secreting glandular structures. [6] Several reports indicate that GCC affects males and females at roughly equal rates and is more frequently noted in individuals of Caucasian ethnicity. The average age for diagnosis is 58 years. At the same time, patients with this carcinoma confined to the appendix tend to be diagnosed at a younger age compared to those with tumors that have spread beyond the appendix. In comparison to the traditional clinical presentation of appendiceal carcinoids, GCCs are more aggressive with a higher rate of metastasis. [7] [8] GCC tumor, unlike other cancers, does not appear to have any predisposing conditions, such as family history or environmental factors. The lack of identifiable risk factors and the rarity of documented cases make it difficult to diagnose at an early stage. Additionally, the lack of specificity of the clinical manifestation of nonspecific abdominal pain in GCC explains the misdiagnosis as acute appendicitis in many instances. [9]

3.2. Clinical presentation

The common clinical manifestation of appendiceal GCC can hardly be differentiated from acute appendicitis, and it is very challenging to diagnose it before surgery. The typical patient presents with right lower quadrant abdominal pain, nausea, vomiting, and fever, and a presumptive diagnosis of appendicitis is made. This general disguise implies that GCC is often an accidental discovery in the course or postoperative period of an appendectomy done on suspicion of acute appendicitis. [10]

Imaging-wise, GCC of the appendix is not usually characterized by any distinct features that could make a definite preoperative diagnosis. The ultrasound, CT, and MRI scans can have findings of appendicitis, which include a thickened appendiceal wall, peri-appendiceal inflammation, or a dilated appendix. These are, however, nonspecific findings which could be observed in other inflammatory and neoplastic diseases of the appendix. Some studies indicate that the finding of elevated mucinous content or a large mass of the appendix on imaging may increase suspicion of a neoplastic process; however, it is not pathognomonic of GCC. [10] [11]

The case of our patient is an ideal example of such diagnostic difficulties. He had classical symptoms of acute appendicitis: progressive aggravation of lower abdominal pain, nausea, and vomiting. On initial abdominal ultrasound, appendicitis was very likely as the appendix wall was thickened, and there was a small hypoechoic nodule at the tip. CT abdomen and pelvis also showed thickening of the soft tissue of the distal appendix. Although these results were in line with appendicitis, the delayed onset of this symptom, along with the moderately elevated tumor markers (CEA and CA 19-9), added an important aspect of suspicion of an unusual or malignant process, which led to further examinations with MRI. The MRI revealed a T2-hyperintense nodule with limited diffusion, which was suggestive of high cellularity and mucinous content, which, in hindsight, were some minor hints to the neoplastic nature of the etiology as opposed to mere inflammation.

Such close attention to slight deviations of the normal course of inflammation and the greater diversity of the imaging manifestations, including increased mucinous content, played a key role in our work. However, we could not make a final diagnosis of GCC before surgery. The surprise eventual pathology, which exposed a goblet cell carcinoid tumor, demonstrated the difficulty in differentiating GCC and acute appendicitis using clinical and imaging presentation alone.

3.3. Differential Diagnosis

Diagnosis of GCC is very important because of its virulent nature and management plan that is different from appendicitis. Nevertheless, the diverse histological appearances and clinical manifestations tend to give it a wide differential diagnosis. The main items to include in the differentiation of GCC are conventional adenocarcinoma of the appendix, classical appendiceal carcinoid tumors, as well as metastatic signet-ring cell carcinoma of other primary sites. [10]

Conventional Adenocarcinoma of the Appendix (Mucinous or Signet-Ring cell): Appendiceal adenocarcinomas and those with mucinous or signet-ring cell histology may closely resemble GCC. They both may show mucin production and infiltrative growth patterns. The most distinctive characteristic is the dual neuroendocrine and glandular differentiation of GCC. Although adenocarcinomas are strictly epithelial tumors, GCC has both epithelial and neuroendocrine differentiation. Histologically, the existence of well-developed glandular formation and lack of neuroendocrine differentiation would support the diagnosis of adenocarcinoma. The presence of both goblet-like cells and Paneth-like

cells with positive neuroendocrine markers (chromogranin A and synaptophysin) in our patient's case helped distinguish it from a pure adenocarcinoma. [10] [12]

Classical Appendiceal Carcinoid Tumor (Well-Differentiated Neuroendocrine Tumor): The classical carcinoid tumors of the appendix are normally well-differentiated neuroendocrine neoplasms with a less aggressive nature as compared to GCC. They have homogenous cells having round nuclei and granular cytoplasm which form nests, cords, or trabeculae. They are positive with neuroendocrine markers, but lack the large mucin-producing goblet cells and the glandular differentiation of GCC. The lack of signet-ring cell, as well as the absence of significant mucin production, are the most important characteristics that differentiate classical carcinoids from GCC. The tumor of our patient had obvious glandular differentiation and mucin production, which is not characteristic of classical carcinoids. [10] [13]

Metastatic Signet-Ring Cell Carcinoma (Particularly Gastric or Pancreatic): Metastatic signet-ring cell carcinoma, especially those of gastric or pancreatic origin, may metastasize to the appendix and simulate primary appendiceal neoplasms, such as GCC. Such metastatic tumors are frequently rich in signet-ring cells and may be difficult to distinguish only on morphological grounds. In this case, immunohistochemistry is essential. The primary site of origin is determined using markers like CK7, CK20, CDX2, TTF-1, and GATA3. As an example, gastric adenocarcinomas are frequently CK7 and CDX2 negative, and pancreatic adenocarcinoma may be CK7 positive. The tumor cells in our case were CK7 negative, TTF-1 negative, GATA3 negative, and CK20 positive, effectively eliminating the gastric, pancreatic, pulmonary, and urothelial primaries, which is a strong indication towards the primary appendiceal origin of the GCC. [10] [14]

Mucinous Neoplasm (Low-Grade Appendiceal Mucinous Neoplasm - LAMN): LAMNs are defined by the presence of excessive extracellular mucin and may appear as a mucinous cyst of the appendix. Although they are mucin-producing lesions, they do not usually have the invasive pattern of growth and neuroendocrine differentiation as GCC. The epithelial cells of LAMN are low-grade dysplastic with no true invasion. The fact that the case of our patient had invasive clusters of goblet-like cells and neuroendocrine differentiation allowed it to be differentiated from a LAMN. [10] [15]

3.4. Management and outcomes

Appendiceal GCC, now GCA, is a complex disease and is considered be more aggressive than the typical appendiceal well-differentiated neuroendocrine tumors (NETs) (carcinoids) (1) Guidelines from the ASCRS, NANETS, and ENTS recommend hemicolectomy with regional lymph node dissection for all patients with GCCs since there is a high incidence of lymph node metastasis. [16] Surgery is vital, and a right hemicolectomy is the recommended procedure, even when the disease appears to be local. [9] These tumors are difficult to identify preoperatively, even on colonoscopy, due to neoplastic growth into the submucosa with invasion into the appendiceal wall. [16]

In localized GCC, simple appendectomy itself is usually not thought to be adequate, given the tendency of local invasion, regional lymph node metastasis, and peritoneal spread of the tumor. [3] The indication of right hemicolectomy is backed by the fact that there is a high possibility of the presence of residual tumor or lymph node metastases in a patient despite having what seems to be a complete appendectomy. [17] [18] Tumor size, grade, and extension play an important role in favoring hemicolectomy. These include size > 1-2 cm, high-grade components, perineural or vascular invasion, and extension of the tumor beyond the wall of the appendix. [8] [17] In our case, even though the initial presentation was that of acute appendicitis and laparoscopic appendectomy had been uneventful, the final pathology showed that it was a T3N0M0 (Stage IIA) GCC with extension to periappendiceal fat. It was this proactive and aggressive surgical intervention based on cross-disciplinary expertise that helped our patient have a successful outcome.

Adjuvant chemotherapy is less well established in GCC and is subject to controversy, both because the tumor is rare and because large-scale randomized controlled studies have not been performed. However, if the tumor is at an advanced stage or displays unfavorable factors such as positive lymph nodes, high-grade histology, or peritoneal spread, adjuvant chemotherapy regimens for colorectal adenocarcinoma can be considered. [9] [18] It is an extremely individual decision regarding adjuvant therapy that is made in the framework of a multidisciplinary tumor board, considering the possible utility of the treatment against the risk and side effects. In the case of our patient, no further therapy was deemed necessary in the postoperative environment.

The prognosis for goblet cell carcinoid is variable based on the stage at diagnosis. However, there is a favorable prognosis with early detection by multidisciplinary teams and successful surgical removal. On the other hand, GCC has a worse prognosis than neuroendocrine tumors due to GCC possessing both neuroendocrine and adenocarcinoma components. [16] Overall, there is a better prognosis in patients diagnosed with stage I or II, compared to stages III or IV, where there is a poorer prognosis for which chemotherapy is usually recommended [9] Even four years after surgery,

this patient was able to resume his previous lifestyle with no signs or symptoms of disease recurrence, supporting the benefits of timely diagnosis and surgical intervention.

3.5. What does the future hold for the diagnosis and treatment of GCC (GCA)?

Changing the name of GCC to GCA highlights its aggressive biological behavior, and new technologies could transform the diagnosis, molecular subtype, and treatment of GCA. GCA is molecularly differentiated into both the NETs and typical adenocarcinomas, and they are characterized by mutations in the KRAS/CDX2/TP53 genes. [19] Information technology (IT), artificial intelligence (AI), and liquid biopsies, as well as multi-omics (genome, epigenome, transcriptome, proteome, metabolome, exposome, and microbiome, are some fast-emerging technologies that can greatly improve GCA subtype. In terms of therapy, KRAS inhibitors, immunotherapy, and optimization of Hyperthermic Intraperitoneal Chemotherapy (HIPEC) are on the way. Also, the enrollment in trials of enrolling RAS-mutant or microsatellite instability-high (MSI-H) appendiceal cancers is prioritized. It is estimated that 10% of prenatal deaths are caused by a particular ministry [20] The future of gastrointestinal cancers is precision medicine, and soon GCA will be treated similarly to other molecularly defined forms of gastrointestinal cancers. Several clinical trials of GCA are currently ongoing and planned, focusing on precision oncology and innovative treatments. [2] [6] [16]

Most of the clinical trials on GCA can be incorporated into the wider study of the gastrointestinal cancers because of the logistics involved in recruiting a rare disease. These trials are likely to change the way GCA is managed, as it switches to biomarker-based therapy over empiric chemo. The molecular characteristics and classification schemes are transitional as well, especially when genomic profiling data are included, as with The Cancer Genome Atlas. (21)

3.6. What did we learn from this case?

This case is an alert to the fact that early and aggressive surgical intervention, informed by an evidence-based decision-making approach, may result in positive long-term outcomes in the setting of rare and possibly malignant tumors. The four-year survival of the patient without the disease is a demonstration of the success of early and broad intervention. It supports the notion that in tumors that tend to recur or metastasize, an aggressive and comprehensive approach to initial treatment and a vigilant follow-up is the only way to deliver favorable oncological results. This case, thus, offers significant information on the diagnostic dilemmas and effective management approaches to appendiceal GCC, which is adding to the expanding knowledge base on this rare and significant entity.

We know why we want to abandon the name of “carcinoid,” which implies a NET, and does not characterize the aggressive nature of GCC. The clinical implication is that GCC, now GCA, requires a more aggressive treatment and follow-up than NETs. Our tumor is regarded as a low-grade GCA.

Abbreviations

- Goblet cell carcinoid (GCC);
- Goblet cell adenocarcinoma (GCA);
- Neuroendocrine tumor (NET);
- Immunohistochemistry (IHC)

4. Conclusion

This case demonstrates that the GCC, now GCA, of the appendix may be diagnostically mistaken as acute appendicitis. That is why a wide range of differential diagnoses is imperative even in the context of seemingly simple clinical situations. The histopathological and immunohistochemical assessment is critical to the definitive diagnosis, and is essential to differentiate GCC and its mimics and direct proper management. The good long-term result in this patient demonstrates the immense contribution of a multidisciplinary approach and early aggressive surgery to attain good results in this rare and difficult malignancy.

Compliance with ethical standards

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Disclosure of conflict of interest

The following declarations are made by all authors:

- Payment/services information: All authors have declared that they received no financial support from any organization for the submitted work.
- 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 was lost to follow-up, and all attempts to reach the family members were unsuccessful. Therefore, the paper has been sufficiently anonymized to maintain patient confidentiality.

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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