Collision tumor of invasive squamous cell carcinoma and adenoid cystic carcinoma in the oropharynx: a case report

Senmi Qian
First Affiliated Hospital Zhejiang University
Meibao Feng (11318069@zju.edu.cn)
First Affiliated Hospital Zhejiang University

Case Report

Keywords: Collision tumor, Adenoid cystic carcinoma, Squamous cell carcinoma, Diagnosis, Case report

Posted Date: March 30th, 2022

DOI: https://doi.org/10.21203/rs.3.rs-1491593/v1

Abstract

Introduction: Primary collision tumors composed of two histologically distinct components are rare in the head and neck, its pathobiological features remain unclear.

Case presentation: A man in his late 50s presented with paranesthesia pharynges and underwent upper endoscopy, followed by histopathological examination of biopsy specimens. He seems to have two tumors in the oropharynx with a size of 1.5*1.3cm, and an ulcer focus in the esophagus measuring about 1*0.8cm. Histopathological examination revealed a coexistence tumor of two components in the oropharynx, one was an adenocarcinoma with cribriform and solid growth pattern, with interstitial vitreous degeneration and central necrosis, the other was with a solid growth pattern, which was observed in both oropharynx and esophagus. The patient had a favorable outcome.

Conclusion: Correct diagnosis can be challenging and is essential and beneficial for the prognosis of the patient.

Introduction

Squamous cell carcinoma is the most common epithelial malignancy of the head and neck. Approximately 1% of invasive carcinomas are adenoid cystic carcinoma. McGee et al introduced the currently accepted designation of ACC in 1965[1]. ACC has a worse prognosis than the conventional SCC. However, because of the rarity of the coexistent tumor, there is no universal consent in the standard treatment of this devastating malignancy. To our knowledge, there have been few reports of the coexistence of the ACC with invasive SCC. In this case, there are collision tumor of SCC and ACC of the head and neck, with another invasive SCC in esophagus, which invaded to submucosa.

Case Presentation

A man in his late 50s presented with paranesthesia pharynges for 25 days, no obvious dysphagia and any significant health problems. Magnetic resonance imaging (MRI) showed thickening sinus mucosa in the right side of piriform recess, iso-intensity on T1-weighted image, and mild hyper-intensity on T2-weighted image and DW image, with inhomogeneous enhancement. Standard upper endoscopy showed a protruded lesion at 35 cm from the Incisor. Hypopharyngectomy and partial esophagectomy were subsequently performed. The surgical specimen showed a solid tumor in the right dipper, which was involved in the piriform recess with a size of 1.5*1.3cm, and another ulcer focus in the esophagus measuring about 1*0.8cm.

The hematoxylin and eosin (H&E)-stained section showed the tumor was composed of two distinct morphological types. One was a SCC component which had a solid pattern with a small area of squamous cell carcinoma invasion (Fig. 1A□B) and vascular tumor thrombus; the other was an adenocarcinoma with a cribriform and solid growth pattern, interstitial vitreous degeneration and central necrosis were also observed (Fig. 1C), tumor cells showed moderate-to-severe atypia and no clear nerve

invasion, mitotic activity were easily seen; There was an abrupt transition between these two components (Fig. 1D).

To further classify the tumor, a panel of immunohistochemical stains was performed. Immunohistochemical analysis revealed that CK7 was positive for the ACC but negative for the SCC component (Fig. 2A), CK5/6 and p63 were diffusely and strongly positive in SCC but patchily positive in ACC (Fig. 2B), p63 was only expressed in the outer luminal surface of the pseudocysts of the ACC component (Fig. 2C), furthermore, CD117 positively showed in the inner luminal surface of the pseudocysts of the ACC component but negative in the SCC component (Fig. 2D), p53 were negative in both of the two components (Fig. 2E); MYB, MYBL1 and NFIB genes detected by in situ hybridization method harbored no gene fusion in ACC components (Fig. 2F).

About 92% of ACC showed positive expression of CD117, which is the valuable factor in diagnosing ACC. CD117 was mainly expressed in the inner luminal surface of the pseudocysts of ACC, however, full-thickness expression could be observed in solid nest of cancer[1]. In this case, CD117 mainly expressed in the cytoplasm and cell membrane of cancer cell, which is consistent with earlier reports.

Fusion gene of MYB-NF1B leaded by t(6;9) (q22-23;p23-24) translocation is significant discovery for the diagnosis of ACC[2–4], but only happened in about 50% of ACC[5]. According to recent reports, other molecular events such as NOTCH, PI3K signaling pathways, and epigenetic modification and DNA damage repair were also found to play an important role in the tumorigenesis of ACC[6, 7]. There is no gene fusion observed in this case, implied other mechanisms in the progression of ACC, which conforms to the reports.

Discussion And Conclusions

ACC has tubular, cribriform, solid growth pattern. Any solid growth pattern is the unfavorable factor of DSS and RFS. AJCC also suggested that clinic stage and more mitosis were also independent prognostic factors of ACC[8]. The solid growth pattern and mitosis were easily observed in this case, suggested the poor prognosis of the patient.

The differential diagnosis included basaloid squamous cell carcinoma, basaloid squamous cell carcinoma with adenoid cystic-like features, basal cell carcinoma. Basaloid squamous cell carcinoma with adenoid cystic-like features could be excluded with an abrupt transition between these two components. Basal cell carcinoma could be ruled out by interstitial vitreous degeneration and cribriform growth pattern, CK5/6 negative expression, p63 was only positive in the outer luminal surface of the pseudocysts and CD117 expression pattern could further excluded the basal cell carcinoma and basaloid squamous cell carcinoma.

ACC is much more aggressive than SCC. However, because of the rarity of the coexistent tumor, there is no universal consent in the standard treatment of this devastating malignancy. Generally, treatment for ACC is the same as that for SCC of the head and neck; a combination of surgery and radiation therapy

has been the primary treatment. Therefore, early recognition of the coexistence of ACC and SCC is significant to guide clinical management and is extremely beneficial for patients, a close follow up is highly recommended.

Declarations

Acknowledgements

The authors would like to thank Ms Mei Kong and Zhihui Wang for their technical assistance.

Funding

This project was supported by the fund of National Natural Science Foundation of China (No. 82002488) and Natural Science Foundation of Zhejiang Province (No. LQ21H160020).

Author's contributions

Each author made substantial contributions to the conception and design of this paper. The author(s) read and approved the final manuscript.

Availability of data and materials

All the data regarding the findings are available within the manuscript.

Ethics approval and consent to participate

This case report was approved by the clinical research ethics committee of the First Affiliated Hospital of Zhejiang University, College of medicine, reference number [illegible]2022[illegible]154[illegible]-[illegible].

Competing interests

The authors state that there are no conflicts of interest to disclose.

References

1. Shi X, Chang X, Wu H, et al: Co-existing Adenoid Cystic Carcinoma and Invasive Squamous Cell Carcinoma of the Uterine Cervix: a Rare Case Report and Literature Review. 2014, 44:502-7.
2. Stenman G, Sandros J, Dahlenfors R, et al: 6q- and loss of the Y chromosome—Two common deviations in malignant human salivary gland tumors. Cancer Genetics and Cytogenetics 1986, 22:283-93.
3. Nordkvist A, Mark J, Gustafsson H, et al: Non-random chromosome rearrangements in adenoid cystic carcinoma of the salivary glands. 1994, 10:115-21.
4. Persson M, Andrén Y, Mark J, et al: Recurrent fusion of MYB and NFIB transcription factor genes in carcinomas of the breast and head and neck. Proceedings of the National Academy of Sciences of

the United States of America 2009, 106:18740-4.
5. Xu B, Drill E, Ho A, et al: A Clinicopathologic Study With Correlation Between MYB Fusion and Protein Expression. Am J Surg Pathol 2017, 41:1422-32.
6. Ho AS, Kannan K, Roy DM, et al: The mutational landscape of adenoid cystic carcinoma. Nat Genet 2013, 45:791-8.
7. Ho AS, Ochoa A, Jayakumaran G, et al: Genetic hallmarks of recurrent/metastatic adenoid cystic carcinoma. J Clin Invest 2019, 129:4276-89.
8. van Weert S, van der Waal I, Witte BI, et al: Histopathological grading of adenoid cystic carcinoma of the head and neck: Analysis of currently used grading systems and proposal for a simplified grading scheme. Oral Oncology 2015, 51:71-6.

Figures

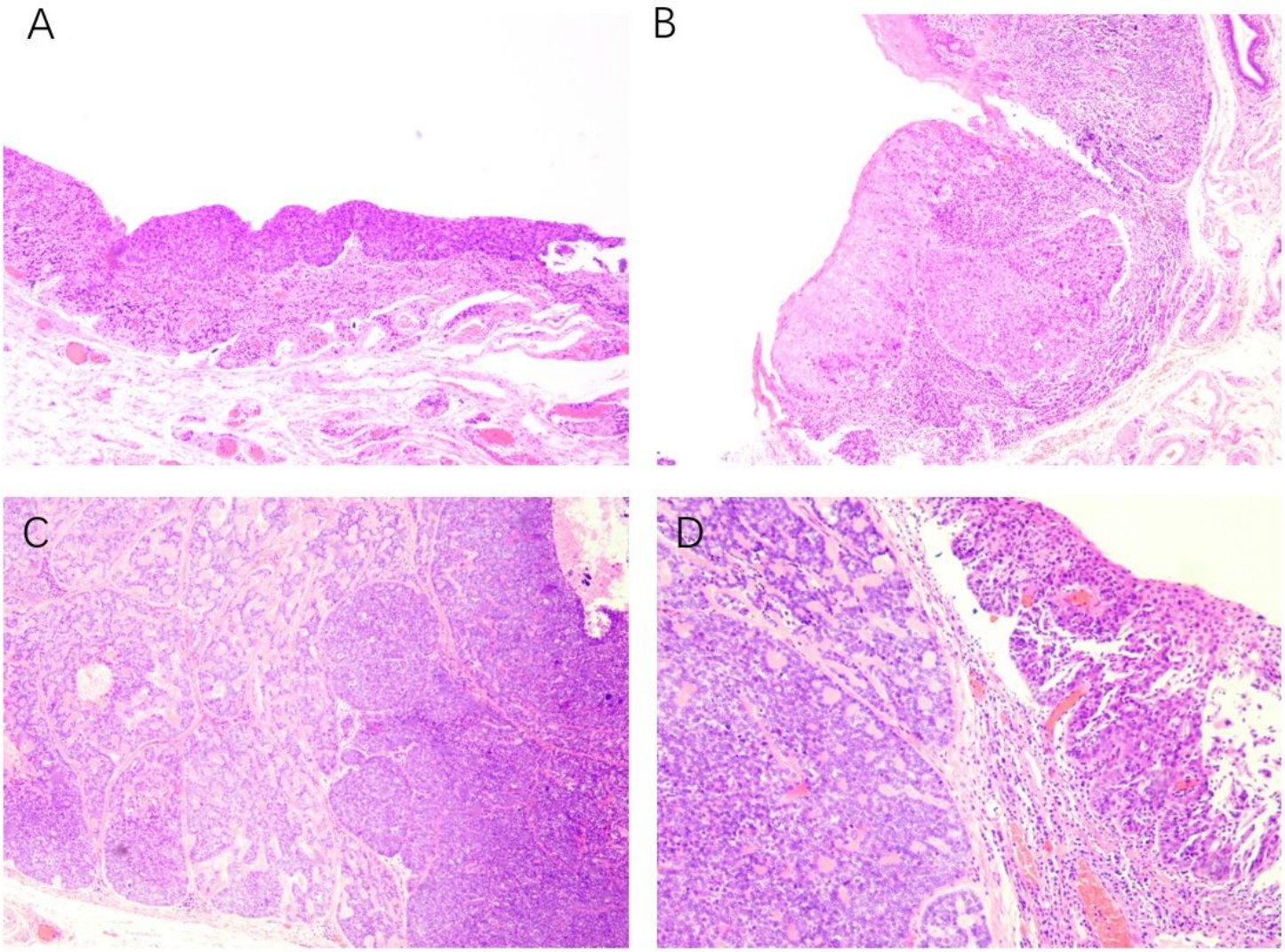

Figure 1

(A) Carcinoma in situ of squamous epithelium, and focal squamous cell carcinoma invaded to the submucosa (B). (C) H&E showed cribriform and solid growth pattern of adenoid cystic carcinoma, the stroma showed interstitial vitreous degeneration and central necrosis; (D) there is clear boundary between squamous cell carcinoma and adenoid cystic carcinoma.

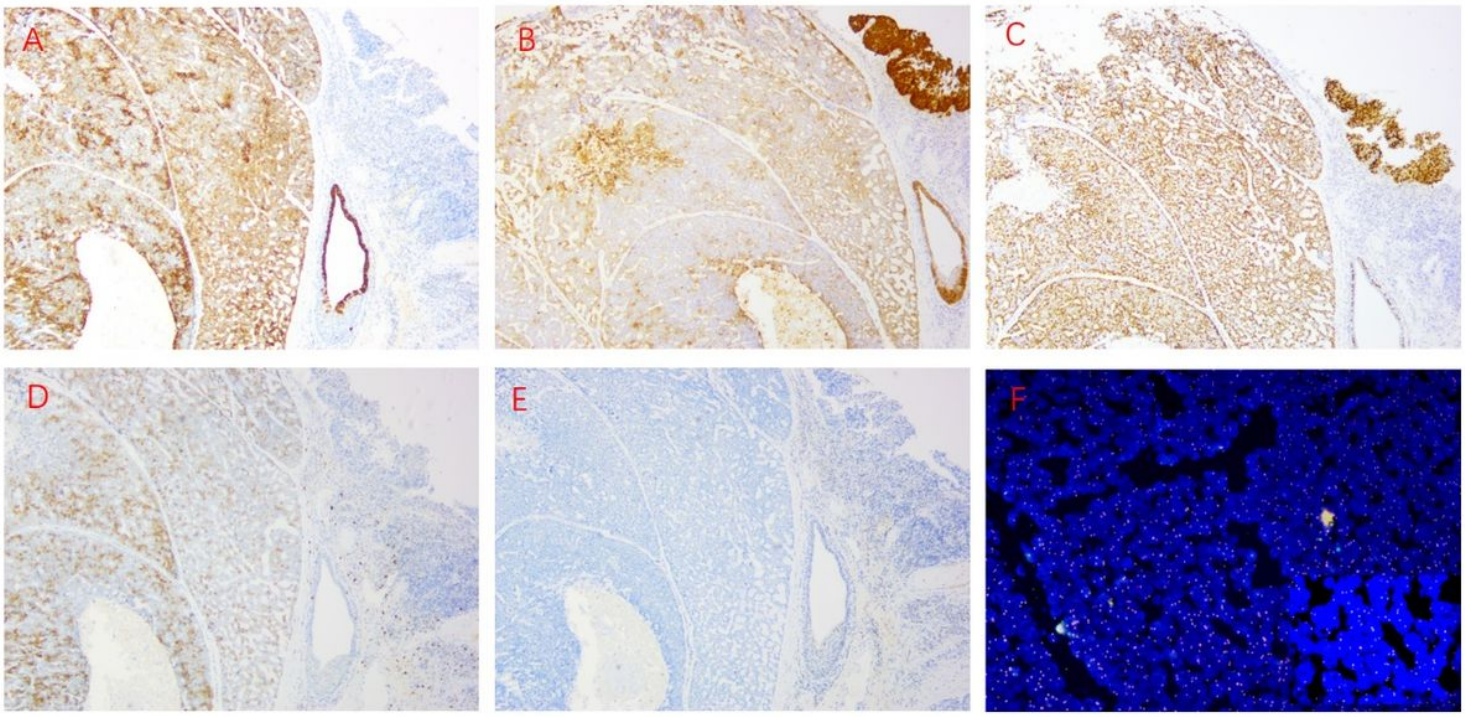

Figure 2

(A) Tumor cells showed intense expression of CK7 and CD117 of adenoid cystic carcinoma component (D). (B□C) CK5/6 and P63 expression pattern in squamous cell carcinoma and adenoid cystic carcinoma. (E) Negative expression of P53 in both carcinomas. (F) FISH showed no gene fusion in adenoid cystic carcinoma.