Case Report

Alveolar adenoma and coexisting atypical adenomatous hyperplasia: a case report and literature review

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Summary

Alveolar adenoma is a rare tumour of the lung. It is typically found in asymptomatic adults as a peripheral or subpleural nodule on imaging examination. Microscopically, the tumour is composed of admixture of epithelial and mesenchymal component in variable sized cystic or alveolar structures. The tumour shows a benign nature. There have been no reported recurrences or metastases. Malignant transformation of alveolar adenoma and coexisting with lung carcinoma have been rarely described. In this article, we report a case of an alveolar adenoma and coexisting atypical adenomatous hyperplasia. This case, contributing to the limited numbers of cases described to date, illustrates the importance of awareness on the possibility of alveolar adenoma being associated with lung carcinoma and its precursor lesions especially when diagnosed by small biopsy specimens.

Key words: alveolar adenoma, atypical adenomatous hyperplasia, lung carcinoma

Introduction

Alveolar adenoma (AA) is a rare pulmonary tumour with proliferation of alveolar epithelium and septal mesenchyme. Yousem and Hochholzer described the first six cases of AA in 19861. To date, approximately 57 cases have been reported in the literature. AA represents one type of adenoma of lung in the 2021 World Health Organization classification2. There are only few articles about coexistence of AA with other neoplasms3-6. Here, we report a rare case of an AA coexisting with atypical adenomatous hyperplasia (AAH). We also review the clinical, radiologic, pathologic and molecular features of AAs.

Case report

A 52-year-old woman, a non-smoker, was incidentally found to have a solitary pulmonary nodule during a regular medical examination. The computed tomography (CT) of the chest revealed a 1.2 cm subpleural nodule in the right upper lobe. The positron emission tomography (PET) scan showed no definite uptake in the nodule. She underwent a wedge resection of the right upper lobe lesion. Frozen section analysis indicated a benign lesion.

The pathological examination of the wedged lung tissue showed a well-demarcated grey white nodule grossly. Microscopic examination
disclosed a well-fined tumour containing multiple cystic spaces filled with eosinophilic granular material and lined by flattened to cuboid epithelial cells without significant atypia (Fig. 1A). The associated stroma between the cystic spaces consisted of bland spindle cells, inflammatory cells and myxoid matrix (Fig. 1B). Immunohistochemically, the lining epithelial cells were positive for pan-cytokeratin (CK), CK7 and thyroid transcription factor 1 (TTF-1) (Fig. 1C). The interstitial cells were focally positive for cluster of differentiation 34 (CD34) (Fig. 1D), while negative for TTF-1 and CK. Both the histopathological and immunohistochemical studies confirmed the lesion to be an AA. Incidentally, an AAH, 1 mm in size, was identified in

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**Figure 1.** (A) In low-power view, the tumour was well defined, containing multiple variable-sized cystic spaces filled with eosinophilic granular material. (haematoxylin-eosin, original magnifications 40X) (B) The epithelial cells lining the cystic spaces were bland, flat to cuboidal. The stroma between the cysts consisted of inflammatory cells, bland spindle shaped cells and loose, myxoid matrix. (haematoxylin-eosin, original magnifications 200X) (C) TTF-1 immunoreactivity was observed in the epithelial cells and negative in the interstitial cells. (original magnifications 200X) (D) The interstitial cells were partially immunoreactive for CD34. (original magnifications 200X) (E) An atypical adenomatous hyperplasia was incidentally identified in the surrounding lung parenchyma. (haematoxylin-eosin, original magnifications 100X) (F) In high-power view, the atypical adenomatous hyperplasia revealed cytological atypia of the proliferative pneumocytes. (haematoxylin-eosin, original magnifications 400X)
the surrounding lung parenchyma. Cytological atypia was found (Figs. 1E, F). There was no parenchymal inflammation or fibrosis in the background. No recurrence or metastasis was observed after 3-year follow up.

**Discussion**

AAs occur more commonly in women than in men (M:F ratio about 1:2) (Tab. I). The tumour usually presents in the five to sixth decade of life. Most often, patients are asymptomatic. Other symptoms, such as cough, chest pain, dyspnoea and haemoptysis, have been reported. Chest plain film and computed tomography typically show a peripherally or subpleurally located, well circumscribed nodular lesion with occasional central cavitation. There is usually no or minimal, thin-rim contrast enhancement. More central location of the lesions has been described. The most common location was the left lower lung field. The reported size ranges from 2 mm to 98 mm (average 24 mm). Most of the cases are solitary; however, two cases of multiple occurrences have been reported. Few reported cases with follow-up imaging studies have shown slight size enlargement. Positron emission tomography (PET) scan often shows no or faint uptake. AAs are grossly well-circumscribed, grey-white or yellow-brownish nodules with a soft, spongy or glistening cut surface. Occasionally they may be cystic or haemorrhagic. Prominent cystic change had been reported. Microscopic examination typically shows a well-defined lesion that contains admixture of epithelial and mesenchymal component in variable sized cystic or alveolar structure. Proteinaceous, eosinophilic granular material can be observed in the cysts,

**Table I. Clinical features of reported cases of AA. (Continues)**

| Author         | Cases | Age (y) /Sex | Imaging | Location | Size (cm) | Treatment       | F/U (mo.) |
|----------------|-------|--------------|---------|----------|-----------|-----------------|-----------|
| Yousem1        | 6     | 45/F         | Solitary nodule | LLL      | 2         | Wedge           | 13        |
| 54/F           | Solitary nodule | RUL     | 2.5     | Lobectomy | 12        |
| 59/F           | Solitary nodule | RUL     | 1.3     | Lobectomy | 13        |
| 74/F           | Solitary nodule | RML     | 2.5     | Lobectomy | 120       |
| 58/M           | Solitary nodule | LLL     | 1.5     | Wedge     | 56        |
| 64/M           | Solitary nodule | RUL     | 1.2     | Lobectomy | N/A       |
| Al-Hilli31     | 1     | 60/F         | Solitary nodule | LUL     | 1         | Wedge           | N/A       |
| Semeraro1      | 1     | 67/F         | Solitary nodule | RML     | 2.8       | Enucleation     | 3         |
| Oliveira29     | 1     | 55/F         | Solitary nodule | RLL     | 6         | Segmentectomy   | 32        |
| Böhm32         | 1     | 52/F         | Solitary nodule | LLL     | 2         | Wedge           | 12        |
| Burke28        | 10    | 41/F         | Solitary nodule | LLL     | 1.1       | N/A             | N/A       |
| 41/F           | Solitary lesion | LLL     | 2.5     | N/A       | N/A       |
| 46/F           | Round shadow | N/A     | N/A     | N/A       | N/A       |
| 52/F           | Solitary lesion | LLL     | 3       | N/A       | N/A       |
| 39/M           | Solitary nodule | RLL     | 2       | N/A       | N/A       |
| 45/M           | N/A   | LUL           | 1.5     | N/A       | N/A       |
| 50/M           | Solitary lesion | LLL     | N/A     | N/A       | N/A       |
| 58/M           | Solitary nodule | LLL     | 1.9     | N/A       | N/A       |
| 68/M           | Coin lesion | LLL     | 1.8     | N/A       | N/A       |
| N/A            | Shadow | Right         | 3       | N/A       | N/A       |
| Fujimoto7      | 1     | 47/F         | Three nodules | LLL*2, RUL | 2,1,1     | Wedge (largest nodule) | 15        |
| Yilmaz33       | 1     | 51/F         | Solitary nodule | RUL     | 1.8       | Wedge           | 24        |
| Cakan12        | 1     | 34/F         | Solitary nodule | LUL     | 1.6       | Wedge           | 12        |
| Palpa34        | 2     | 54/M         | Solitary nodule | Right    | 2.5       | Wedge           | 144       |
| 66/F           | Solitary nodule | RML     | 1.4     | Resection | N/A       |
| Hartman9       | 1     | 51/F         | Solitary nodule | RUL     | 3.4       | Wedge           | 18        |
| Golubovic13    | 1     | 64/F         | Solitary nodule | LUL     | 4         | Resection       | N/A       |
| Cavazzz14      | 1     | 69/M         | Solitary, cystic nodule | RUL     | 3.5       | Wedge           | 13        |
| Halldorsson25  | 1     | 43/M         | Solitary nodule | LLL     | 1.1       | Wedge           | 18        |
| Saito35        | 1     | 35/F         | Solitary nodule | RUL     | 2         | Wedge           | N/A       |
| Sak27          | 2     | 62/M         | Solitary nodule | LLL     | 1.5       | Wedge           | 22        |
| 54/M           | Solitary nodule | LLL     | 4       | Wedge     | 32        |
some of which may also contain macrophages, fresh blood with cholesterol clefts or hemosiderin-laden macrophages. The larger cysts tend to be concentrated towards the centre of the lesion. Small lymphoid aggregates could be seen at the periphery of the lesion. Microcystic formation and follicular growth pattern, morphologically mimicking thyroid tissue, has been described.

The cystic spaces are separated by varying thickness of stroma containing mostly bland spindle cells, loose or myxoid matrix, capillaries and scattered inflammatory cells including lymphocytes, plasma cells, and eosinophils. Round-shaped cells as well as spindle-shaped interstitial cells over the septal mesenchyme has been mentioned in one report. Foci of interstitial haemorrhage and hemosiderin deposition can be observed. One reported case showed presence of mature adipocytes within the tumour. High power examination of the cystic lining shows single layer of flat, cuboidal or hobnail epithelial cells. Nuclear atypia, mitotic activity, and necrosis are absent. Immunohistochemical analysis of AAs typically shows positive immunoreactivity for CK, CK7, carcinoembryonic antigen (CEA), epithelial membrane antigen (EMA), TTF-1 and surfactant apoprotein in the epithelial component with expression of CK20 in one reported case. There is variable immunoreactivity for CD34, smooth muscle actin and S100 in the mesenchymal component. The eosinophilic granular material within the cysts is PAS-positive. The proliferation

### Table I. Clinical features of reported cases of AA. (Follows)

| Reference | Age | Gender | Tumor Type | Size | Procedure | Follow-up |
|-----------|-----|--------|------------|------|-----------|-----------|
| Nakamura | 58/F | Solitary nodule | LUL | 0.8 | Wedge | 3 |
| González | 71/M | Solitary nodule | RLL | 1.7 | Segmentectomy | N/A |
| Petrella | 38/F | Giant cystic mass | LUL | 9.8 | Resection | N/A |
| Kondo | 61/F | Solitary nodule | LUL | 2.4 | Segmentectomy | 12 |
| Bhavsar | 59/M | Undetected | RUL | 0.2 | Lobectomy | N/A |
| Panagiotou | 42/F | Solitary nodule | RLL | 1.5 | Wedge | 12 |
| Nosti | 54/F | Solitary nodule | LLL | 2 | Lobectomy | 12 |
| De Rosa | 24/M | Solitary nodule | LLL | 1.8 | Wedge | 7 |
| Wang X | 60/F | Solitary nodule | RLL | 7.3 | Wedge | 6 |
| Wang L | 48/F | Solitary nodule | RLL | 4 | Lobectomy | 48 |
| Kazerouni | 41/F | Solitary nodule | LLL | 1.2 | Lobectomy | N/A |
| Lee | 57/F | Two nodules | LLL | 1.6 | Wedge | N/A |
| Yamamoto | 65/F | Double barrel-shaped nodule | LLL | 1.3 | Wedge | N/A |
| Tang | 47/F | Solitary nodule | RLL | 4 | Segmentectomy | 52 |
| Hsiai | 67/M | Solitary, cystic | RLL | 4 | Wedge | N/A |
| Okada | 83/M | Solitary nodule | LUL | 1.8 | Segmentectomy | 48 |
| Gan | 48/F | Solitary nodule | LLL | 3.5 | Lobectomy | 60 |
| Zhang | 40/M | Solitary nodule | LLL | 5.2 | Lobectomy | 26 |
| Kavas | 36/M | Solitary nodule | LLL | 2.6 | Resection | 34 |
| Volk | 26/F | Multicystic | LUL | N/A | Lobectomy | N/A |
| Roshkovan | 48/F | Solitary nodule | LUL | 1.2 | Wedge | N/A |
| Present case | 52/F | Solitary nodule | RUL | 1.2 | Wedge | 36 |

**Abbreviations:** y, years; mo., month; F/U, follow up; F, female; M, male; RUL, right upper lobe; RML, right middle lobe; RLL, right lower lobe; LUL, left upper lobe; LLL, left lower lobe; N/A, not available

* The size of 18mm includes both components of alveolar adenoma and adenocarcinoma.
index (Ki-67) is less than 1%. Absence of p53 immunohistochemical expression is found. Based on the immunohistochemical profiles and some ultrastructural studies, the epithelial component of AA is considered to be type II pneumocytes. Additionally, the interstitial cellular component is thought to be made up of fibroblasts or fibroblast-like cells. The exact histogenesis of AA is uncertain. It is unclear if both the epithelial and mesenchymal components are neoplastic. It has been postulated that the cell origin of AA is probably primitive mesenchymal cells with the capacity to differentiate towards type II pneumocytes. Some authors thought of the CD34 immunoreactivity in the interstitial cells as a manifestation of the primitive mesenchymal nature being able to differentiate into specific lineages, such as adipocytes. By contrast, the hypothesis that the mesenchymal proliferation is secondary to the epithelial proliferation and stimulated by the epithelial growth has also been proposed. Limited studies have described the molecular alterations of AA. Cavazza et al. used microsatellite instability analysis to show that the stromal and epithelial components are genetically unrelated, supporting the dual nature of the lesions. Flow cytometric studies of the AAs in some reports showed a diploid DNA pattern. Roque et al. reported a non-balanced translocation demonstrated by fluorescence in situ hybridisation analysis. The importance of the chromosomal abnormality on the pathogenesis of AA is still unknown. Lack of EGFR mutation and anaplastic lymphoma kinase (ALK) protein expression was shown in one report.

The differential diagnosis of AA comprises both benign and malignant lesions, including sclerosing pneumocytoma (SP), lymphangioma, AAH, adenocarcinoma in situ (AIS) and lepidic predominant adenocarcinoma. Proliferation of both epithelial and stromal component of SP can resemble AA. The absence of diverse growth patterns as well as negative TTF-1 and EMA staining in the mesenchymal component in AA can help one distinguish between these two considerations. Lymphangioma might be confused with AA because both tumours contain cystic structure and proteinaceous material. The flat lining cells of AA may resemble endothelial cells. However, unlike AA, lymphangioma does not contain the mesenchymal component. The absence of CK immunoreactivity in the lining cells of lymphangioma also helps to differentiate. Other diagnostic considerations include AAH, AIS and lepidic predominant adenocarcinoma. Areas of small cystic or glandular spaces with regular lining cells in AA might simulate these three entities. Lack of cytologic atypia and infiltrative growth in AA can be helpful features in the differential diagnosis.

The reported cases of AA have had an indolent clinical course. Regardless of the type of surgical intervention, no recurrences or metastases have been reported with up to 15 years of follow-up. However, one patient presented with malignant transformation of an AA to an adenocarcinoma in one report. Borderline and transitional areas between the AA and adenocarcinoma has been shown in the case. There are two reported cases of AA with a concurrent lung carcinoma or AIS. To the best of our knowledge, our case is the first description of coexisting AA and AAH. AAH is a putative precursor of AIS or lung adenocarcinoma. The above-mentioned cases including ours, albeit in limited numbers, remind us of the possibility of AA with the potential for malignant transformation or being associated with lung carcinoma and its precursor lesions especially when diagnosed by small biopsy specimens.

**Conclusion**

In summary, AA is a rare pulmonary tumour often found in asymptomatic adults in their five to sixth decade of life. No recurrences or metastases have been reported to date. It should be considered in the differential diagnosis of a solitary pulmonary nodule. Our case demonstrates the rare occurrence of coexisting AA and AAH. Further molecular analyses might assist in clarifying its pathogenesis and nature.

**Conflict of interest**

The authors declare no conflicts of interest.

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There is no relevant financial interest in the products or companies described in this article.

**Ethical consideration**

The report complied with ethical standards. The report was approved by the Institutional Review Board/Ethics in the Taoyuan General Hospital, Ministry of Health and Welfare. (IRB NO: TYGH110-05).

**Author contributions**

All listed authors contributed to the production of this manuscript and are listed in the appropriate order.

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