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A exceptional collision tumor of primary adrenal angiosarcoma and non-functioning adrenocortical adenoma

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ABSTRACT

Primary adrenal angiosarcoma is an extremely rare vascular tumor. We report a case of a 63-year-old man with a collision tumor of epithelioid angiosarcoma and adrenocortical adenoma of the right adrenal gland. The adrenal tumor was incidentally observed by a preoperative computed tomography (CT) scan of penis squamous cell carcinoma. The patient underwent a right laparoscopic adrenalectomy, and the tumor size measured 34 × 34 × 15 mm. Histological examination revealed two different tumor cell proliferations, namely epithelioid angiosarcoma and adrenocortical adenoma. He had no symptoms or abnormality in his endocrine studies, so the adrenocortical adenoma was considered non-functioning. Three months after the adrenalectomy, bilateral pleural metastasis was observed by CT scan and pleural biopsy. Paclitaxel monotherapy was performed, and the tumor retreated. The patient died one and a half years after the adrenalectomy, but the cause of death was believed to be another disease (metastatic penis squamous cell carcinoma). To the best of our knowledge, this is the fourth report of primary adrenal angiosarcoma combined with adrenocortical adenoma.

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1. Introduction

Angiosarcoma is a high-grade malignant neoplasm composed of cells that demonstrate endothelial differentiation [1]. Angiosarcomas commonly occur in the skin, soft tissue, breast, bone, liver and spleen. Primary angiosarcoma of the adrenal gland (PAA) is an extremely rare neoplasm with only 31 cases reported so far in the literature [2–7]. We report herein a case of a collision tumor in which primary adrenal angiosarcoma was combined with adrenocortical adenoma. To the best of our knowledge, this is the fourth report of primary adrenal angiosarcoma combined with adrenocortical adenoma [5–7].

2. Case report

2.1. Clinical presentation

A 66-year-old Japanese man presented with an enlarged tumor of the glans penis. The tumor was diagnosed as squamous cell carcinoma, so a preoperative examination was done. A computed tomography (CT) scan of the chest and abdomen to search for metastases revealed a 34 mm diameter heterogeneous mass of the right adrenal gland (Fig. 1). The lesion was characterized by low density with focal vascularization after intravenous injection of contrast material without evidence of local tissue invasion or metastatic spreading. The lesion was suggestive of cortical adenoma, but the possibility of malignancy could not be excluded because of the size (diameter >3 cm). He had been diagnosed with high blood pressure two years prior, and had achieved good control (105-125/65-75 mmHg) by 5 mg amlodipine treatment. On physical examination, no other specific findings including obesity were identified. No abnormality was shown in complete blood count or electrolytes. Endocrine studies showed in normal levels of adrenocorticotropic hormone, cortisol, renin, aldosterone and urinary cortisol. First, partial penectomy was performed and the lesion was diagnosed as penis carcinoma, pT3. Pathologically and immunohistochemically, the penis tumor shows well to moderately differentiated squamous cell carcinoma, and the carcinoma cells were negative for CD31. Then, two months after the first operation, a laparoscopic right adrenalectomy was performed.

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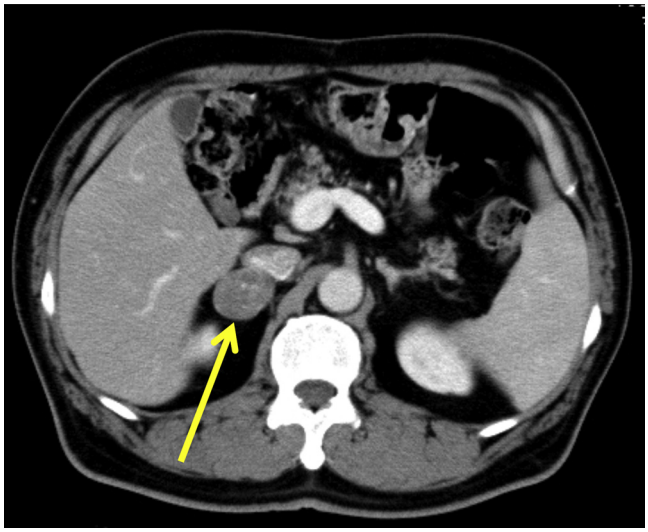

Fig. 1. CT scan after intravenous injection showed a 34 mm-diameter, low-density mass with focal vascularization of the right adrenal gland.

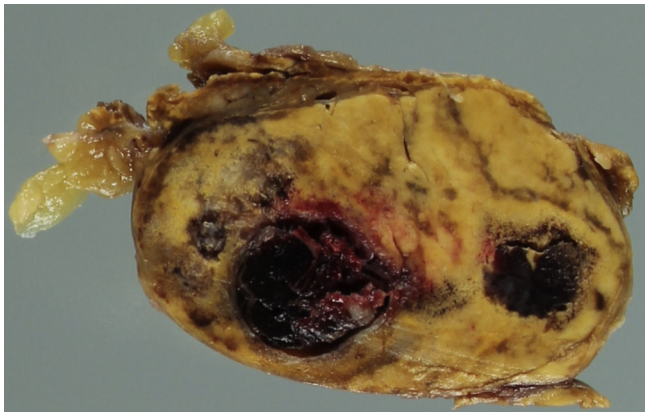

Fig. 2. The cut surface was yellowish in color with focal hemorrhaging.

3. *Pathological findings*

Grossly, the adrenocortical tumor appeared well-demarcated, and measured 34 × 34 × 15 mm. The cut surface was yellowish in color and showed focal hemorrhaging (Fig. 2). The residual adrenal gland measured 15 × 17 × 3 mm and was displaced laterally. Histopathology revealed two different tumor cell components (Fig. 3A). Most of the lesion showed a proliferation of adrenocortical cells without atypia and mitoses, indicating adrenocortical adenoma (Fig. 3B). The Weiss score was 0 (clear cytoplasmic tumor cells, proliferating in an alveolar pattern, no necrosis, no venous invasion, no sinusoidal invasion, no capsular invasion). Another component within the adrenocortical adenoma showed a proliferation of atypical epithelioid cells having oval to polygonal nuclei, prominent nucleoli and eosinophilic cytoplasm, arranged in sheet-like, irregular cord or papillary structures with red blood cells, invading the surrounding cortical adenoma (Fig. 3C, D). Intracytoplasmic vacuoles containing red blood cells were observed (Fig. 3E). Mitotic figures were occasionally seen (3/20 HPF).

Immunohistochemically, the atypical epithelioid cells were positive for CD34, CD31, factor VIIIRA, AE1/AE3, ERG, FLI-1 and focally positive for CAM5.2 and vimentin, but negative for D2-40, EMA, calretinin and WT-1 (Fig. 4A,B). The MIB-1 labeling index was about 30% in atypical cell components. Thus, we identified the adrenal lesion as collision tumor, combining epithelioid angiosarcoma within adrenocortical adenoma.

4. *Post-operative course*

Three months after the adrenalectomy, the patient developed difficulty in breathing. Right pleural effusion and bilateral pleural masses were detected by chest X-ray and CT. These features were considered bilateral pleural metastases originating from the adrenal angiosarcoma by CT-guided percutaneous biopsy. Paclitaxel monotherapy was used in the four months after the adrenalectomy, and the tumor retreated. One year after the penectomy and eleven months after the adrenalectomy, CT revealed

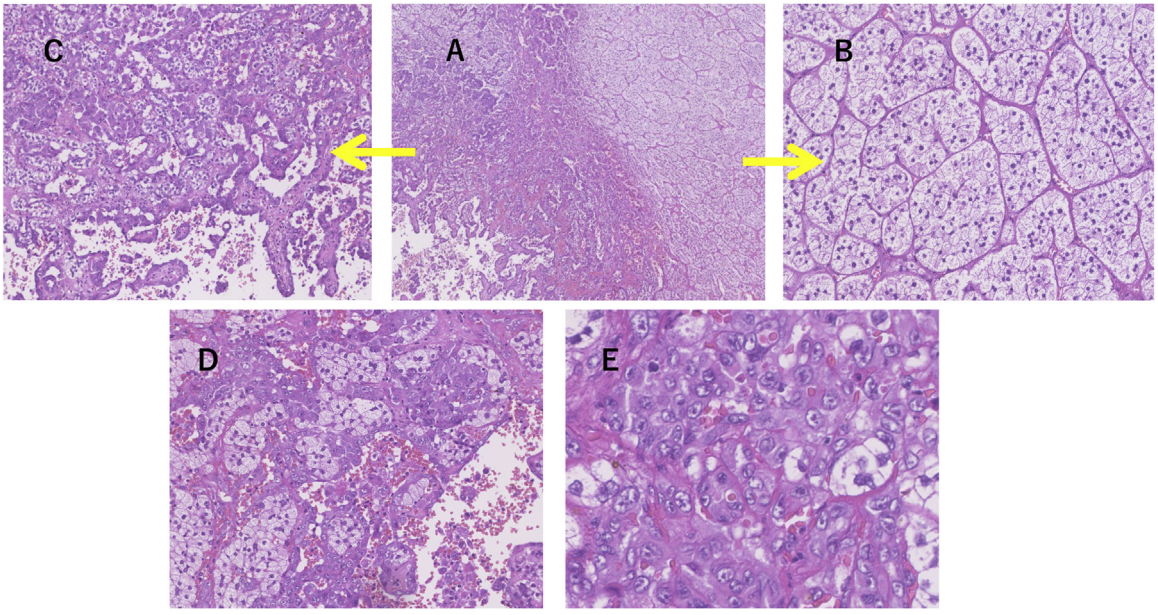

Fig. 3. Histopathological HE (A) Two different tumor cell components (X40).
(B) Adrenocortical adenoma component (X100) (C) Epithelioid angiosarcoma component (X100) (D) Atypical epithelioid cells invade the cortical adenoma (X200) (E) Atypical epithelioid cells proliferated in a sheet-like pattern and had intracytoplasmic vacuoles containing red blood cells (X400)

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