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Primary Cutaneous Apocrine Adenocarcinoma of the Axilla in Klinefelter's Syndrome: A Case Report

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ABSTRACT

Background: Primary Cutaneous Apocrine Adenocarcinoma (PCAA) is a rare cutaneous malignancy that arises from areas rich in apocrine glands, particularly the axilla. There are less than 200 cases described in the literature, but none has been reported in patients with Klinefelter syndrome, who are known to have an increased risk of breast cancer.

Case Presentation: We present the case of a 66-year-old man with a significant family history of breast cancer who developed a right axillary mass initially thought to be hidradenitis suppurativa. On physical examination, he had bilateral gynecomastia. Laboratory workup was significant for hyper-gonadotrophic hypogonadism. Mammography showed bilateral gynecomastia with no radiologic evidence of malignancy in the breast, while magnetic resonance imaging of the breast revealed two suspicious masses seen in the right breast. Excision biopsy of the right axillary mass revealed a high-grade invasive adenocarcinoma involving the dermis and subcutis; the cells had prominent nucleoli vesicular chromatin pattern and voluminous eosinophilic cytoplasm consistent with primary cutaneous apocrine adenocarcinoma. Immunohistochemistry was positive for GATA-3, GCDFP-15, E-cadherin, ER positive, PR negative, HER 2 by IHC 3 +, and androgen receptor-positive 100 %. Positron emission tomography showed mildly hypermetabolic asymmetric gynecomastia, right greater than left, but no abnormal hypermetabolic activity to suggest malignancy. Karyotype confirmed 47 XXY chromosomes. The patient underwent bilateral mastectomy due to his preference; excised breast tissue was negative for malignancy.

Conclusion: We presented the first case report of PCAA of axillary in Klinefelter syndrome. There is a need for awareness of this association and differenting it from breast carcinoma.

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INTRODUCTION

The apocrine gland is a sweat gland that connects directly to hair follicles, produces viscid secretions, and empties indirectly to the skin surface; it is primarily confined to the axilla, nipples, and perineal regions[1-3], occuring in areas rich in apocrine glands.[4]

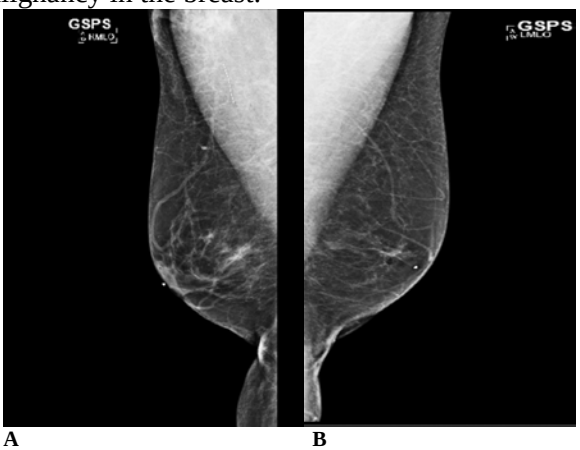
Primary Cutaneous Apocrine Adenocarcinoma (PCAA) is an extremely rare cancer first described by Horn in 1944, and since then, less than 200 cases have been reported in the literature.[5-7] The incidence ranges from 0.0049 to 0.0173 per 100,000 patients annually.[6]

The risk factors for developing PCAA are not well-established in the literature. Patients with Klinefelter syndrome are known to be at increased risk of breast cancer and gonadal and extra-gonadal tumors, but PCAA has never been reported in these patients.[8,9] We report a case of PCAA in a patient with Klinefelter syndrome.

CASE PRESENTATION

A 66-year-old male was referred to the Oncology clinic following a 2-year right axillary. The patient had a history of hypertension, human immunodeficiency virus (HIV), and a family history of breast cancer in the mother and two sisters. He smoked marijuana and was a former tobacco and alcohol user. There was no history of weight loss, fever, or easy fatiguability. Significant findings on examination revealed bilateral gynecomastia and healed scar in the right axilla—no palpable lymphadenopathy. The initial differential was cutaneous metastatic breast cancer.

The baseline laboratory workup (complete blood count, serum urea, creatinine, and electrolytes) was unremarkable. Luteinizing hormone and follicle-stimulating hormone were both elevated (15.8mIU, reference, 1.7-8.6mIU/mL, and 14.9mIU, reference, 1.5-12.4mIU/mL, respectively) while free testosterone was low (2.8pg/mL, reference, 6.6-18.1pg/mL). The plasma estradiol level was normal (30.3pg/mL, reference, 7.6-42.6pg/mL). Mammography (Figure 1) showed bilateral gynecomastia with no radiologic evidence of malignancy in the breast.



A B

Figure 1. Mammogram showing the right (**A**) and left (**B**) breasts. No suspicious mass or tumor calcifications are present in either breast. Breast tissue is seen within both breasts, the right greater than the left. A scar marker is seen within the right breast.

Magnetic resonance imaging of the breast revealed a suspicious mass in the right breast (Figure 2).

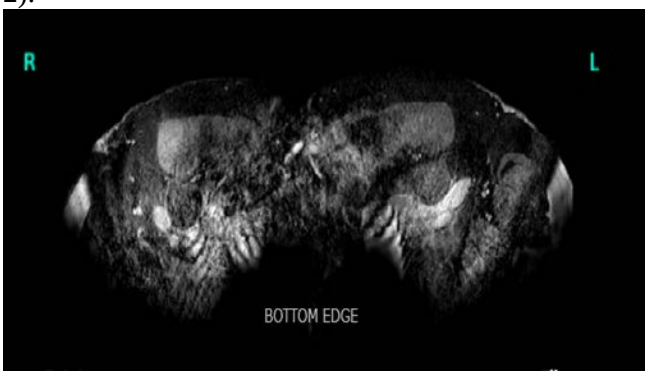


Figure 2: Suspicious mass seen in the right breast (arrow). The left breast is unremarkable.

The left breast was unremarkable. Positron emission tomography showed mildly hypermetabolic asymmetric gynecomastia, right greater than left. Otherwise, no abnormal hypermetabolic activity was observed to suggest malignancy. The patient had an MRI-guided breast biopsy of the mass which was negative for malignancy. Genetic studies, including *BRCA 1 & 2, CHECK 2, ATM, TP 53,* and *STK-11*, were all negative. However, analysis of quality control markers across the X chromosome suggested a possible sex chromosome complement of 47, XXY. Based on this, we decided to request karyotyping, which came back as 47XXY.

The right axillary mass was excised, and the tumor measured about 4cm by 5cm. The margin of resection of the tumor was about 6mm, while the intraepidermal component of the carcinoma reached as close as 2mm from the inked epidermal edge, suggestive of poorly differentiated adenocarcinoma consistent with cutaneous invasive ductal carcinoma of the breast.

On immunohistochemistry (Figure 3), the pathologic specimen from the excised right axillary tumor was positive for GATA-3, Gross cystic disease fluid protein- 15 (GCDFP-15), and Estrogen receptor (ER). It was also positive for the androgen receptor (100% 3+) and HER-2 receptors but negative for the progesterone receptor. Additional positive staining markers included E-cadherin, *CAM 5.2, EMA*, and *CK7*. Monoclonal HMB-45, *CEA, P40, P63, PAX 5. TTF-1, SMMHC, CK20*, and *CK5/6* were all negative. The Ki67 proliferation marker was about 50% (Figure 4).

Due to the negative PET scan and MRI-guided biopsy of the breast for malignancy, a second pathologist's opinion of the histology of the slide of the excised right axillary mass was sought, and this revealed a high-grade invasive adenocarcinoma involving the dermis and subcutis; the cells had prominent nucleoli vesicular chromatin pattern and voluminous eosinophilic cytoplasm consistent with

apocrine adenocarcinoma (Figure 5). Bone and computerized tomography (CT) scans of the chest, abdomen, and pelvis were negative for metastases.

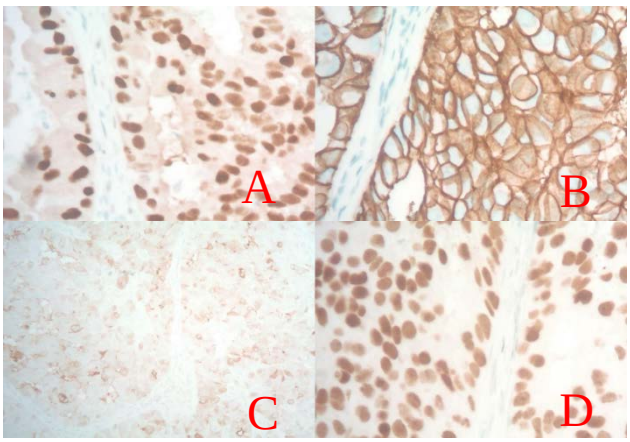


Figure 3. The neoplastic cells are diffusely immunoreactive for Androgen (A),Her2/neu (B), GCDFP-15 (C), GATA3 (D), E-cadherin, CK7, Cam 5.2, EMA; weakly and partially reactive to the ER; no reactive to PR, P63, P40, CK5/6, CK20, Pax5, TTF-1, monoclonal CEA, HMB-45 and SMMHC

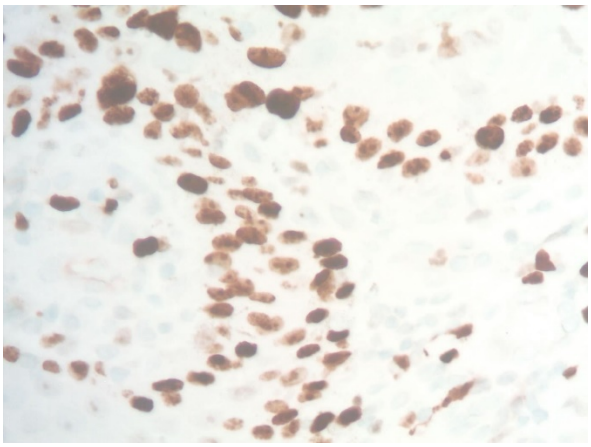


Figure 4. The neoplastic cells demonstrate moderately proliferativerate (~50%) by Ki67 stain

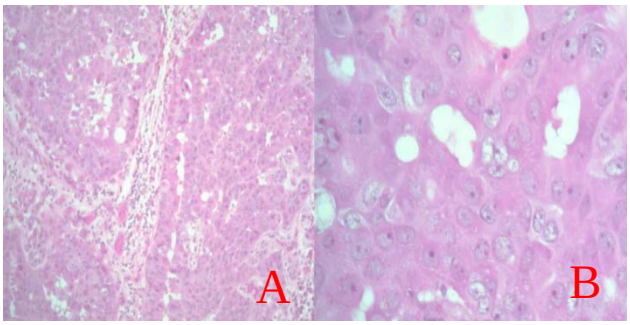


Figure 5. Primary cutaneous apocrine carcinoma: A. At intermedium magnification (100 x), the moderately differentiated adenocarcinoma with glandular and ductular structures is located in the dermis. B. At high magnification (400 x), the neoplastic cells demonstrate apocrine differentiation with abundant eosinophilic cytoplasm and oval-to-round nuclear with aprominent nucleolus.

The nature of the tumor and available treatment options were discussed with the patient. He was started on an androgen receptor blocker Enzalutamide 160 mg daily, due to the overexpression of the receptors by the tumor cells, after explaining the risks and benefits to him. He was also given adjuvant chemotherapy with paclitaxel and trastuzumab. Considering the high-grade nature of the disease and the high likelihood of local recurrence, the patient was treated with external beam radiation therapy to the right axilla and regional lymph nodes. The patient personally decided to undergo a bilateral mastectomy for cosmetic reasons; the excised breast tissue was negative for malignancy. He continues to be followed up in the Oncology clinic until now.

DISCUSSION

We could make a definitive diagnosis of PCAA of the axilla in our patient because of the histological features, immunochemical characteristics, and radiologic and pathologic findings of the breast that were negative for mammary cancer.

Our case represents the first report of PCAA in Klinefelter syndrome. PCAA is extremely rare; less than 200 cases of PCAA have been reported in the literature, with an annual incidence of 0.0049 to 0.0173 per 100,000 patients.[6] Typically, it presents as a nodule or papule, which may be erythematous or violaceous, and occurs predominantly in the axillary and perineal areas; however, it can also be found in other locations such as the eyelid, scalp, nipple, chest, ear, and fingers.[10-13] PCAA affects males and females equally, with an over-representation in Caucasians and a peak age between the 6th and 7th decades of life.[6,14] At the time of diagnosis, most cases of PCAA are localized in more than half of the cases, while regional or distant metastases occur in about 35% of the cases.[6]

Patients with Klinefelter syndrome (KS) are known to be at increased risk of breast cancer and gonadal and extra-gonadal tumors.[8,9] The increased ratio of estrogen to testosterone has been postulated to increase the risk of breast cancer in affected individuals.[9] We propose that this hormonal balance may also play a role in the development of PCAA in patients with KS.

PCAA can easily be confused for cutaneous metastases of metastatic breast cancer; both cancers share similar embryological and histological immune-histochemical characteristics, making the diagnosis of PCAA even more challenging.[15] The apocrine axillary glands have a predominance of androgen and estrogen receptors in their secretory epithelium.[16,17]

Key histologic features that suggest PCAA include decapitation secretion and PAS-positive and diastase-resistant granular material in the cytoplasm of tumor

cells.[7,18] Fernandez-Flores group[19] proposed an eight-panel immunohistochemical staining pattern, as shown in Table 1, that may help differentiate between PCAA and breast cancer. Furthermore, PCAA has been reported in some cases to be adipophilin−and stains strongly for cytokeratin 5/6, while breast cancer tends to be adipophilin+.[20] Wick *et al.*[21] reported that about 30% of cutaneous ductal sweat gland carcinomas strongly express HER-2 receptors. In addition, there are documented cases in the literature of ER positivity in PCAA.[6,7] Our patient was HER-2 positive, progesterone-negative, and estrogen-positive.

Table 1. Immunohistochemical Staining for PCAA versus Breast Cancer

Immunohistochemical staining	PCAA	Breast Cancer
Estrogen receptors	-	+
Progesterone receptors	-	+
Androgen receptors	+	-
Mammaglobin	+	+
Calretinin	+/-	-
Gross cystic disease fluid protein 15 (GCDFP-15)	+	- (50%)
p63	+	-
D2-40	+	-

Due to the rarity of PCAA, there are only expert opinions regarding its treatment, with no consensus on the management guidelines. The common practice reported in the literature for treating PCAA is wide local excision with clear margins; adjuvant options may include chemotherapy and/or radiotherapy.[22-27] A literature review shows that targeted chemotherapy based on receptors overexpressed by the tumors is commonly used in the management of PCAA. Chemotherapeutic agents used include anthracyclines, taxanes, and platinum-based agents.[22-24] Targeted chemotherapy has also been used, especially when cancer cells over-express a specific receptor. For example, anti-androgen therapy has been used for AR+, HER-2 blocking agents such as trastuzumab, pertuzumab, or lapatinib for HER-2 + cancer, pembrolizumab for PD-L1 + cancers, and tamoxifen or letrozole for ER+ tumors.[23,25-27] As documented in the literature, other receptors may be over-expressed by the cancer cells, including RANK-L, which can be treated with denosumab, an anti-RANK-L drug.[24] The National Comprehensive Cancer Network recommends a combination of an HER-2 blocking agent and taxane as first-line treatment for metastatic breast cancer.[28] Furthermore, in metastatic breast cancer, which over-expresses AR receptors, studies have demonstrated the effectiveness of anti-androgen drugs, with a better efficacy associated with a higher level of AR expression.[29-31] Enzalutamide had the best overall response and clinical benefit rates among the three anti-androgen agents used in these studies. In addition, Robson *et al.*[32] examined 24 cases of PCAC and suggested that tumors which express steroid receptors may be susceptible to chemotherapy.

Collette *et al.*[33] reported the first case of PCAA treated with anti-androgen with a good response; at 19 months of treatment with 160mg of enzalutamide per day, the patient achieved a complete response confirmed by 18fluoro-2-deoxy-d-glucose-positron emission tomography–computed tomography (18FDG-PET-CT). Our patient over-expressed AR and HER-2 receptors; consequently, he was started on an androgen receptor inhibitor and HER-2 receptor blocker in addition to paclitaxel, a taxane. Our report represents the second case of PCAA managed with an androgen receptor blocker.

The rarity of PCAA and the lack of observational studies on this tumor make it challenging to establish the prognosis of this cancer. The median overall survival from the most extensive retrospective study of PCAA cases has been 51.5 months, with the presence of lymph node metastases conferring a worse prognosis.[6,34,35] Our patient is currently being followed up in the Oncology clinic.

CONCLUSION

We have described the first case of primary cutaneous apocrine adenocarcinoma – a rare tumor – in a patient with KS. Our case represents the second report of PCAA treated with an anti-androgen drug to the best of our knowledge. Diagnosis may be difficult, but a careful history and some distinct histologic and immunohistochemical features may help differentiate between PCAA and cutaneous breast cancer metastases. The treatment modality is wide surgical excision of the tumor. Depending on the receptors over-expressed by the cancer cells, targeted chemotherapy may be used as an adjunct treatment.

ETHICAL CONSIDERATIONS

The patient informedly consented to present her medical history and data in this medical journal by signing an informed consent form.

FUNDING

This study has not used any fund from public or private grant providers.

CONFLICT OF INTEREST

There is no conflict of interest in presenting this report.

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