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SMARCA4 loss irrelevant for *ARID1A* mutated ovarian clear cell carcinoma: A case report

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ABSTRACT

Clear cell carcinomas are rare and relatively chemo-insensitive ovarian cancers with a characteristic molecular pathogenesis. Alterations in *ARID1A*, a component of the multiprotein chromatin remodeling complex SWI/SNF, are likely early events in the development of ovarian clear cancers arising from atypical endometriosis. Insight into additional driver events and particularly mutations in the same chromatin remodeling complex is limited. Isolated loss of SMARCA4, encoding the ATPase of the SWI/SNF complex, characterizes other aggressive gynecologic cancers including small cell carcinomas of the ovary hypercalcemic type (SCCOHT), undifferentiated endometrial carcinomas (UDEC), and uterine sarcomas (SDUS). The ovarian clear cell carcinoma of a 48-year-old showed in the initial surgical specimen a subclonal loss of SMARCA4 in addition to an *ARID1A* mutation, i.e., two alterations in the SWI/SNF heterochromatin remodeling complex. We anticipated that the SMARCA4 loss would worsen the disease course in analogy to SCCOHT, UDEC, and SDUS. However, the disease did not accelerate. Instead, the recurrent disease showed restored SMARCA4 expression while retaining the *ARID1A* mutation. Combinatorial redundancy, diversity and sequence in the SWI/SNF complex assembly as well as DNA- and tissue-specificity may explain the observed irrelevance of SMARCA4 loss in the presented *ARID1A* mutated ovarian clear cell carcinoma.

1. Introduction

Ovarian clear cell carcinomas are a rare subtype of epithelial ovarian cancers representing 5%−10% of all ovarian cancers with a higher incidence in Asian populations. Clear cell carcinomas are considered high-grade ovarian cancers with a good prognosis in early stage and poor prognosis in advanced stage. Endometriosis is a risk factor and present in more than 50% of ovarian clear cell carcinomas (Park et al., 2018; Bai et al., 2016; Gadducci et al., 2021). Molecular pathogenesis with characteristic genetic mutations, paraneoplastic syndromes that include thromboembolic events and hypercalcemia, and intrinsic chemoresistance make ovarian clear cell carcinomas unique compared to other ovarian cancers. The most frequent somatic mutations are *ARID1A* (AT-rich interactive domain-containing protein 1A), and *PIK3CA* (phosphatidylinositol-4,5-bisphosphate 3-kinase, catalytic subunit alpha) which are present in about 50% of ovarian clear cell carcinomas (Bolton et al., 2022). These mutations in the SWI/SNF (Switch Sucrose Non-Fermentable) chromatin remodeling complex and the PI-3 kinase signaling cascade may coexist in ovarian clear cell carcinomas and likely cooperate in their carcinogenesis.

Here, we focus on the role of multiple mutations in the SWI/SNF chromatin remodeling complex. *ARID1A* and *SMARCA4* (SWI/SNF related matrix associated actin dependent regulator of chromatin, subfamily a, member 4) are prominent components of the SWI/SNF chromatin remodeling complex and frequently mutated in gynecologic malignancies. While *ARID1A* is often altered in ovarian clear cell carcinomas, *SMARCA4* alterations along with loss of SMARCA4 expression are found in 98% of small cell carcinomas of the ovary, hypercalcemic type (SCCOHT) (Ramos et al., 2014; Conlon et al., 2016) and similarly in over 90% of SMARCA4-deficient uterine sarcomas (SDUS) (Kolin et al., 2020; Howitt and Folpe, 2021). *SMARCA4* alterations have also been reported in undifferentiated and dedifferentiated endometrial carcinomas (UDEC) in 15%-37% (Karnezis et al., 2016; Kobel et al., 2018). Overall, 20% of all human cancers show mutations in subunits of the SWI/SNF complex (Krishnamurthy et al., 2022). The impact of different mutations in the same chromatin remodeling complex on cancer

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progression and prognosis poses an open question (Centore et al., 2020). The initial resection specimen of the ovarian clear cell carcinoma patient presented here showed an *ARID1A* mutation and a subclonal loss of SMARCA4 by both immunohistochemistry and next generation sequencing. At the time, we hypothesized that the subclonal loss of SMARCA4 would determine the disease course of this ovarian clear cell carcinoma and likely result in rapid disease progression analogous to SCCOHT, UDEC, and SDUS.

2. Case presentation

A 48-year-old presented initially to a local Gynecologist with two weeks of pelvic pain. Physical exam was notable for a 15 cm pelvic mass. Subsequent pelvic ultrasound and pelvic MRI demonstrated a complex mass with irregular enhancing components and moderate pelvic ascites. CA 125 at the time was 1,027 U/mL. CT imaging showed right-sided pulmonary emboli, and peripheral hypodensities in the spleen concerning for splenic infarcts. Left femoral and popliteal vein thromboses were diagnosed and therapeutic anticoagulation with rivaroxaban was started. Ten days later, the patient developed left facial droop, dysarthria, left face and arm paresthesia and was diagnosed with acute embolic ischemic infarcts. During the work-up, new renal infarcts were noted. Transthoracic echocardiography showed a negative bubble study. Heparin drip was begun and transitioned to enoxaparin. A CT-guided biopsy of the pelvic mass showed a high-grade carcinoma of gynecologic origin, consistent with a clear cell carcinoma. At this point, the patient transitioned care to Stanford Hospital. Given the multiple thromboembolic processes despite therapeutic anticoagulation likely due to malignancy-induced hypercoagulability and high tumor burden as well as the known relative chemo-insensitivity of OCCCs, upfront surgery with surgical cytoreduction was recommend after consultation with the Stanford Neurology and Hematology Services. Complete cytoreductive surgery was performed with no visible residual disease at the end of the case. Final pathology demonstrated a Stage IIIC ovarian clear cell carcinoma. On immunohistochemical studies, the neoplastic cells were positive for PAX8, Napsin A (patchy), and P504S (patchy). The cells were negative for WT1 and ER, with intact expression of PTEN and a wild type p53 pattern. Immunohistochemistry for BRG1, the gene product of *SMARCA4*, was performed with predominantly intact staining and focal tumor showing complete loss of expression, consistent with a subclonal loss pattern (Fig. 1**A and** 1B). Somatic mutation testing was performed using a commercially available next generation sequencing platform (Table 1) and 2 frameshift mutations were detected in *ARID1A*, a point mutation in *PIK3CA,* and a frameshift mutation in *MLL2* (histone-lysine N-methyltransferase 2). Also, a deletion of *SMARCA4* was noted based on copy number alterations that showed a homozygous (biallelic) loss of all 35 *SMARCA4* exons. The PD-L1 tumor proportion score was 1%, and the tumor was micro-satellite stable by mutational signature analysis. Germline testing was negative. After four cycles of adjuvant chemotherapy with paclitaxel and carboplatin, the patient noted a new 1–2 cm lump in her neck. Ultrasound showed a morphologically abnormal cervical lymph node concerning for disease recurrence. A PET/CT was notable for new lymph nodes above and below the

Table 1
Somatic mutations of the recurrent ovarian clear cell carcinoma identified by next generation sequencing in the initial surgical resection specimen (left side) and the supraclavicular lymph node biopsy of the recurrent disease (right side).

Surgical specimen (1/8/22)	Lymph node biopsy (5/16/22)
ARID1A A339fs*24	ARID1A A339fs*24
ARID1A Y2031fs*1	ARID1A Y2031fs*1
PIK3CA H1047R	PIK3CA H1047R
FGFR2 S252W	FGFR2 S252W
MLL2 F1115fs*4	MLL2 F1115fs*4
SMARCA4 loss	
ZNF217 amplification	
ARFRP1 amplification	

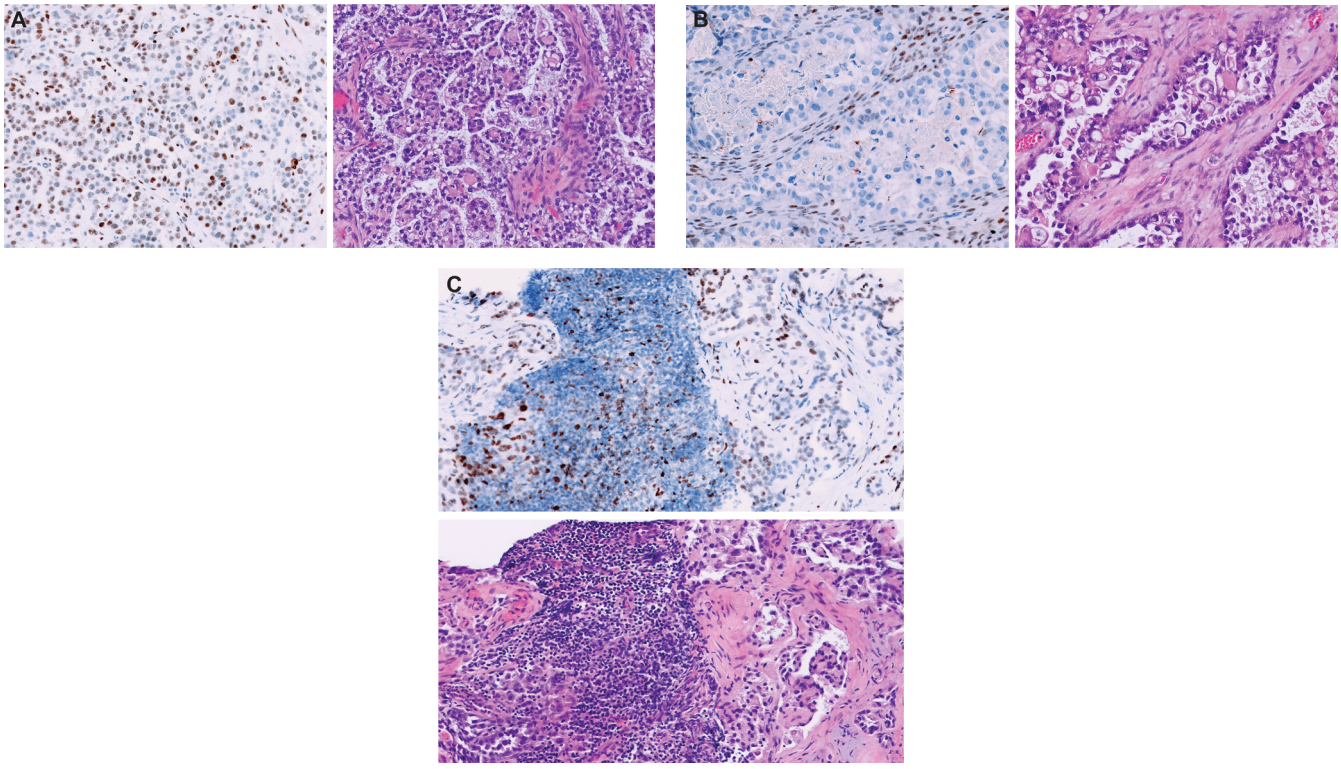

Fig. 1. Immunohistochemistry staining of BRG1, the gene product of *SMARCA4*, in representative sections of the cancer specimen. A. The initial surgical specimen showed intact expression of BRG1 in most of the areas. B. However, in smaller areas subclonal loss of BRG1 was detected.C. The fine needle aspiration of recurrent disease in a supraclavicular lymph node showed intact expression of BRG1 all throughout.

diaphragm and intraperitoneal nodules suspicious for platinum-refractory disease. A fine-needle aspiration of the neck lymph nodes was performed under ultrasound guidance with cytology notable for metastatic clear cell carcinoma. Immunohistochemistry for BRG1 (SMARCA4) was performed and revealed intact staining in the tumor cells consistent with restored BRG1 expression (Fig. 1C). This was corroborated by next generation sequencing (Table 1) which showed that *SMARCA4* was intact. Immunotherapy with Pembrolizumab and Lenvatinib was initiated. After more than a year, the disease process shows continued response to this immunotherapy regimen.

3. Discussion

The subclonal loss of SMARCA4 noted in the initial surgical specimen did not determine the course of the disease nor render it more aggressive beyond a *ARID1A* mutation which is commonly seen in OCCCs. In fact, the cancer cell clone harboring loss of SMARCA4 did not prevail, and the recurrent disease showed intact SMARCA4 expression as evidenced by next generation sequencing on the DNA level and immunohistochemistry on the protein level.

ARID1A mutations are considered early mutations in the pathogenesis of OCCCs. They are seen in atypical endometriosis cells which are considered precursor lesions (Ayhan et al., 2012; Maeda and Shih, 2013). Few is known about additional driver events in OCCCs. *TP53* mutation and deletion of *SMARCA4* could be such additional driver mutations. Although only present in 10%−20% of ovarian clear cell carcinomas, as opposed to 96%−100% of high grade serous tubo-ovarian cancers (Cole et al., 2016), a *TP53* mutation appears to be a negative prognosticator in ovarian clear cell carcinomas (Heckl et al., 2018). Gynecologic malignancies that show isolated loss of SMARCA4 are known to be aggressive and include small cell carcinoma of the ovary, hypercalcemic type (SCCOHT), SMARCA4-deficient uterine sarcomas (SDUS) and undifferentiated and dedifferentiated endometrial carcinomas (UDEC). In contrast to a *TP53* mutation which is present in greater than 50% of human cancers, *SMARCA4* alterations are less common and characteristic for the two rhabdoid gynecologic malignancies SCCOHT and SDUS. Loss of SMARCA4 has been detected in less than 10% in ovarian clear cell carcinomas (Takahashi et al., 2021) and rarely if ever in combination with an *ARID1A* mutation.

ARID1A and *SMARCA4* encode the gene products BAF250A and BRG1, respectively, both proteins in the SWI/SNF chromatin remodeling complex. The SWI/SNF complex was first described in yeast. It is highly conserved throughout species and one of the four chromatin remodeling complexes in humans (Clapier et al., 2017). Chromatin remodeling complexes are thought to unpack and loosen densely packed nucleosomes thereby making genes in compacted chromatin areas accessible to the DNA transcription machinery, recruit transcription factors, co-activators and histone modifiers. SWI/SNF is a multi-protein complex composed of at least 29 proteins (Kadoch et al., 2017). Differential assembly of the SWI/ SNF complex may account for different functions in different tissues and at different timepoints during development (Toto et al., 2016; Alver et al., 2017) and may help explain the clinical findings in this case report. Three subclasses of SWI/ SNF have been described: (i) cBAF, the canonical BAF (BRG1- or BRM1-associated factors), (ii) ncBAF, the non-canonical BAF, and (iii) PBAF (Polybromo-associated BAF); all 3 subclasses show distinct subunit compositions. BAF250A (*ARID1A*) and BRG1 (*SMARCA4*) are components of cBAF. BAF250A (*ARID1A*) or BAF205B (*ARID1B*) form the core of cBAF and are mutually exclusive. BRG1 (*SMARCA4*) and BRM1 (*SMARCA2*) are helicases and the ATPases of cBAF and mutually exclusive, too. Both *ARID1A* and *SMARCA4* alterations result in loss of function and are thus considered tumor suppressor genes (Wiegand et al., 2010). *ARID1A* (BAF250A) has at least two conserved domains: (i) a DNA binding domain which mediates binding to AT-rich DNA sequences and is thought to infer specificity to certain DNA regions, and (ii) a domain for protein–protein interactions that may facilitate interactions with nuclear hormone receptors including the glucocorticoid receptor (Heery et al., 1997; Guan et al., 2011). Preclinical data indicates that if the function of *ARID1A* (BAF250A) is lost, *ARID1B* (BAF250B) replaces it (Helming et al., 2014). Although BAF250A (*ARID1A*) and BAF250B (*ARID1B*) show high sequence homology, they display opposing roles: (i) BAF250A triggers cell cycle arrest in serum-deprived cells, while BAF250B promotes cell cycle re-entry and cell proliferation (Mathur, 2018; Nagle et al., 2011); (ii) BAF250B binds transcriptional enhancers that stop further differentiation and initiate cell proliferation instead (Alver et al., 2017; Kelso et al., 2017). If *SMARCA4* (BRG1) expression is lost, it is replaced by *SMARCA2* (BRM1). BAF250A and B have been shown to interact with both BRG1 (Wang et al., 1996; Zhao et al., 1998) and BRM1 (Oike et al., 2013). In summary, BAF250A and B proteins recognize specific DNA sites, determine the binding of the chromatin remodeling complex to specific DNA sites and display opposing roles. The BAF proteins assemble first and then recruit BRG1 or BRM1. Thus, the exchange of the upstream BAF250 protein from A to B could be the determining molecular event in ovarian clear cell carcinomas and additional downstream mutations such as the loss of SMARCA4 may not add any further effect. In a recent genomic profiling report of more than 113,000 solid cancers, homozygous *SMARCA4* mutations were found to be mutually exclusive with other alterations in the SWI/SNF complex (Fernando et al., 2020).

The here presented clinical course suggests that the loss of SMARCA4 in addition to an *ARID1A* mutation does not change the severity of the disease; in fact, the recurrent disease restored SMARCA4 expression and retained the same *ARID1A* mutations. The clinical findings may be related to the combinatorial redundancy, diversity, and sequence of the SWI/SNF subunit assembly, and their DNA- and tissue-specificity. Further research is needed to obtain a better understanding of underlying molecular alterations in the chromatin remodeling complex SWI/SNF that drive ovarian clear cell carcinomas and appear distinct from the isolated SMARCA4 loss in SCCOHT, UDEC, and SDUS. Understanding the molecular mechanisms will help improve and guide clinical decision making.

4. Conclusion

The initial subclonal loss of SMARCA4 did not determine the clinical course of the *ARID1A* mutated ovarian clear cell carcinoma presented here, and SMARCA4 expression was restored in the recurrent disease. Combinatorial redundancy, diversity, and sequence of SWI/SNF complex assembly, DNA- and tissue-specificity may be underlying mechanisms.

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Stanford Cancer Institute.

6. Prior presentation

None.

7. Patient consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

CRediT authorship contribution statement

Samantha Kay Wagner: Investigation, Data curation, Writing – original draft. **Ashley Moon:** Investigation, Writing – review & editing. **Brooke E. Howitt:** Data curation, Writing – review & editing. **Malte Renz:** Conceptualization, Investigation, Writing – review & editing.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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