# **SMARCB1 (INI1) - deficient Sinonasal Undifferentiated Carcinoma the First Reported Case in Lebanon: A Case Report and Literature Review.**

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# **Abstract**

# Primary carcinomas of the sinonasal tract are uncommon malignancies in the head and neck. First reported in the in literature in 2014, SMARC-B1 deficient sinonasal carcinomas is an aggressive variant of sinonasal malignancies characterized by loss of expression of SMARCB1 (INI), which is a vital component of the SW1/SNF complex. Due to its peculiarity and the histopathologic properties shared with other sinonasal malignancies, along with limited treatment guidelines, SMARCB1 deficient tumors own significant therapeutic and diagnostic challenges.

# In this report we describe the first case of SMARCB1 deficient sinonasal carcinoma in Lebanon, it is a case of a 51-year-old male patient, presented with sensation of fullness in his nostrils and night snoring. Investigations done showed a large a mass which was excised and surgical pathology showed SMARCB1 deficient sinonasal carcinoma confirmed by immunohistochemistry.

# This report sheds the light on the diagnostic challenges and aggressive nature of SMARCB1 deficient sinonasal tumors. Given the lack of appropriate number of cases to establish guidelines for management on this topic we aim to provide a comprehensive review of the available literature in order to enhance understanding of this complex malignancy and provide insights for future directions.

# **Key Words:** Sinonasal undifferentiated carcinoma; SMARCB1; Head and neck carcinoma; Chemotherapy; Radiation Therapy; CNS involvement.

# **Introduction:**

# Primary malignancies of the sinonasal tract are fairly rare tumors with an incidence of 5% or less of all head and neck tumors [1]. Sinonasal carcinomas constitute a diverse spectrum of histological subtypes, each with its unique molecular characteristics and clinical behavior, nevertheless, have the propensity to occur as a poorly differentiated neoplasm with histopathologic deviation from the frequently encountered head and neck tumors. The latter seem to be a very distinct feature for sinonasal malignancies. In 1986, sinonasal undifferentiated carcinoma (SNUC) was first recognized as a unique and tremendously aggressive subtype of sinonasal carcinomas [2]. Due to its uncommonness and poor prognosis, SNUC should only be considered as an exclusion diagnosis, histologically appearing as monomorphic or basaloid, poorly differentiated cells with epithelial origin and no other features suggesting a different neoplasm.

# SMARCB1-defficienent SNUC stands out as a compelling entity, characterized by its distinctive histopathological features and complex interplay of genetic alterations [2]. First described in 2014 by Agaimy et al, SMARCB1(INI1)-deficient Sinonasal Carcinoma has since garnered attention owing to its challenging diagnosis, aggressive clinical course, and limited treatment options [3][4]. SMARCB1 alternatively known as INI1, constitutes a core element of the SWI/SWF chromatin remodeling complex. SWI/SWF complex regulates gene expression through the modulation of chromatin structure, therefore governing basic cellular functions such as proliferation, differentiation, and DNA repair [5]. Since 2014, four major disease entities have been described on different deficiencies in the SW1/SNF complex SMARCB1 (INI1)-deficient among them: SMARCB1 deficient sinonasal adenocarcinoma, SMARCA4-deficient carcinoma [5]. Loss of SMARCB1 function, regardless of the mechanism can lead to tumorigenesis. A major hurdle in addressing SMARCB1 deficient sinonasal carcinoma remains the lack of diverse treatment modalities with significant impact on long term survival, prognosis and disease recurrence. Surgical resection with adjuvant concurrent radiotherapy and chemotherapy remains the preferred treatment modality in many cases, with the occasional use of immunotherapy. Another factor contributing to higher mortality and disease burden is the difficult detection of SMARCB1 deficient sinonasal carcinoma and differentiating it from other sinonasal neoplasms [5]. In this case report we will provide an analysis of a case of SMARCB1 deficient sinonasal carcinoma with a comprehensive literature review.

# **Case presentation:**

# Our patient, a 51-year-old gentleman, previously healthy, presented on September 6, 2021 referred by the radiation oncology team for a multidisciplinary treatment of his care.

# He had sought medical advice in early June 2021 because of sensation of fullness of his right nostril and snoring at night. He had undergone a first surgery soon after for a supposed nasal septal deviation which worsened his breathing and snoring. Imaging studies were then ordered. A non-enhanced CT scan of the head & Neck showed a large non-calcified heterogenous mass with cystic degeneration in the right nasal cavity replacing the nasal septum on the left side and extending to the right maxillary sinus with destruction of its medial wall. It measured about 43 x 31 mm causing complete obstruction on the right side associated with complete obliteration of the right maxillary sinus and both frontal sinuses. He also had an enhanced MRI Scan which revealed an intermediate and heterogeneously enhancing mass lesion on the gadolinium enhanced T1 sequences, invading the medial wall of the orbit and the upper right maxillary sinus, the anteromedial parasagittal right frontal lobe of the brain with the left nasal septum deviation suggestive of a malignant nature. It had intermediate mildly hyper T2 signal. He underwent a supposedly debulking surgery which was merely a generous biopsy.

# He was then referred to us. After analyzing his images, we referred him for a more complete surgery which he underwent without major complications. This was a major cytoreductive surgery as mentioned in the post operative report with nonetheless residual disease. We elected with the radiation oncology team to start with a concurrent approach the earliest possible.

# Such cases are scarce in the literature, only a few are published as case reports only, without series allowing recommendations or guidelines. Our choice of therapy (Cisplatin infusional and VP16 (etoposide) infusional as well x 3 days Q 21 days, was based on the best reported responses mentioned in all the publications we gathered.

# The treatment plan was discussed with the patient and his family prior to initiation, and he was warned about the potential severe toxicities. He was in Lebanon visiting from Paraguay. He was also informed about the necessity of staying in Beirut for the entire duration of his treatment.

# He received 2 cycles of neoadjuvant Cisplatin and etoposide with an excellent response by repeat imaging criteria, allowing smaller fields of radiotherapy.

# He was planned to receive 70 Gy in 35 fractions (3 dose levels 50-60-70 Gy), VMAT, 6MV-FFF, multiple arcs on a Halcyon machine from VARIAN. Volumes were delineated based on MRI and PET-CT scan images. (below)

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# Figure 1:

# a)*coronal view:* 50Gy covering bilateral neck nodes (Ib🡪V); b)*transverse view:* RT to the intracranial involvement; c)*sagittal view:* high dose to SN/BOS/intracranial part of the tumor; d)*transverse view:* High dose to the SN tumor bed.

# The patient was re-planned at mid treatment to adjust for weight change and tumor shrinkage especially for the intracranial portion of the tumor that had not been removed surgically.

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# Figure 2:

# A/pre-Radiation MRI + Gadolinium showing brain invasion, B/Mid-Radiation therapy MRI + Gadolinium showing tumor regression inside the Brain.

# The following 2 cycles were concurrent but complicated by severe loco-regional grade 4 toxicities: grade 4 pancytopenia, ensuing severe grade 3 to 4 radiation or combination induced mucositis, florid candidiasis, grade 2-3 dermatitis, grade 2 esophagitis, grade 2 xerostomia, grade 3 anorexia. He experienced severe weight loss requiring prolonged hospital admissions, feeding tube insertion (PEG Tube) and radiation sessions breaks because of several overall deterioration episodes, and KPS decrease to 50—60% coming from a baseline of 100%. Pancytopenia episodes required repeated transfusions of PRBCs & platelets, with G-CSF injections, and continued triple prophylaxis with Bactrim Forte, Valtrex and high dose fluconazole. He also required

# Additional broad-spectrum antibiotics, and the change of his antifungal therapy to Cancidas for suspicious resistance to fluconazole, and fungal on top of aspiration pneumonia. He luckily did not require respiratory assistance, his KPS improved to 70% after couple of admissions.

# He remained neurologically & cognitively intact. By the end of a 6 week break he had somehow recovered but weighed 40 kgs coming down from a baseline of 72 Kgs. The decision was to pursue therapy with single modality radiation therapy. A radiological re-evaluation prior to resumption, showed near complete remission.

# Once radiotherapy was terminated, he was allowed a second holiday of almost 3 months to recover further and become autonomous and was sent for rehabilitation. When he was seen again in the clinic, he was almost back to his baseline status and regained close to 14 kgs.

# Since his treatment was interrupted due to major toxicities, and he was radiologically in complete remission, he was allowed an additional month off, to receive reconditioning and aggressive physical and occupational therapy.

# During the following multidisciplinary meeting he had finished his month off, it was decided that since he received only 4 cycles of cisplatin and etoposide 2 as concurrent, and he was not going to receive after such an interruption the remaining 2 cycles, fearing the same toxicities, it would probably be beneficial to offer him adjuvant chemotherapy alone with carboplatin and taxotere. He completed 4 cycles of this consolidative therapy without any toxicity whatsoever, allowing him to drive back and forth on treatment days alone 60 miles each way, of course with triple prophylaxis and G-CSF. This was applied as days 1 & 8 Q 21 days basis. We elected this regimen once more because the literature was not informative as for the choice or offered no recommendations. He completed all therapies by late May 2022 (9 months from presentation). Repeat imaging studies comprised an FDG PET CT Scan and a HR 3 T-gadolinium enhanced brain and neck MRI Scan, both revealing no abnormalities and continuous complete remission, except for the flair signal abnormalities of the frontal lobes (L>R) & his olfactory nerves, owing to the radiation scars. He had not yet recovered his smell sensation and most of his taste. He tolerated dysgeusia and was able to et and drink almost normally without any food aversion.

# He was followed closely initially with repeat MRI scans and FDG PET CT Scans every 4 months, during the first year, and every 6 months the second year. At this point he was allowed to travel back to Paraguay for his work, with yearly follow up visit comprising a gadolinium enhanced brain and head & neck MRI scan and an FDG PET CT Scan with a full laboratory work up. He has remained in complete remission until this date early January 2025. He was last seen in June 2024 and mailed us his imaging studies and laboratories results in January with an email stating how good he was feeling. There was no noticeable residual damage to his face

# **Literature review and discussion:**

# Carcinomas of the sinonasal tract consist of malignancies of the nasal and paranasal sinuses, as well as malignancies of the frontal, ethmoid, sphenoid maxillary and middle ear. Up to 75% of all sinonasal tract cancers have an epithelial histopathological origin with squamous cell carcinoma while adenocarcinoma being the most prevalent [4]. Sinonasal carcinomas account for 3-5% of head and neck cancers, affecting approximately 0.5% per 100,000 individuals. These cancers exhibit a male to female ratio of 1.8:1. [5] [6].

# What makes sinonasal tract a unique site for carcinomas is the ability of those tumors to deviate from the frequently encountered histopathological presentations, their aggressive nature at the time of presentation, and their usually poor differentiation with an underlying pathophysiology that is still vaguely understood [5]. The risk factors for sinonasal malignancies are also not very well understood.

# Many studies report that occupational exposure and wood dust can be considered as well-established risk factors, but only for the infrequent intestinal-type adenocarcinoma [7]. On the other hand, tobacco being a major risk factor for most head and neck carcinomas [7]. Another risk factor was previously considered in multiple studies is the human papilloma virus (HPV) due to its well identified role in causing head and neck squamous cell carcinomas arising predominantly (up to 80%) in the oropharynx. However, those studies failed to show the association between sinonasal carcinoma and HPV due to large differences in the detection rates which were between 0-100%, therefore ruling out HPV as a risk factor. [7]. Following the first discovery of SNUC in 1986 as distinguished and aggressive from of sinonasal tumors, subtyping of poorly or undifferentiated sinonasal carcinomas has went through continuous refining to further classification of these rare tumors. Commonly accepted subtypes of poorly differentiated sinonasal carcinomas include SNUC, basaloid squamous cell carcinoma (SCC) (most common 60%), small cell carcinoma of neuroendocrine type, and lymphoepithelial carcinoma as well as many others [2]. SNUC is a malignancy that is distinct for the sinonasal tract and one of the most fatal sinonasal carcinomas. This could be attributed to difficulty in diagnosis, tumor location in the paranasal sinuses or nasal cavity, SNUC's invasive nature, in addition to the scarcity of reported cases in the literature. SNUC usually manifests as an obstructive mass in the nasal canal, accompanied by facial tenderness and headaches [8]. Other signs may arise as a result of the fast-growing nature of the tumor that might cause compressive symptoms, such as visual impairments due to the optic nerve compression or the rare extension of the neoplasm to the anterior cranial fossa compressing other neurologic tracts. It is important to note that the initial manifestation of the disease are not acute or specific in the matter and often mimic different benign conditions hence, up to 85% of patients are diagnosed at later stages of their (stage IV) [8][9]. Diagnosing SNUC can be extremely challenging since only a biopsy histopathological and immunohistochemical techniques confirms the diagnosis. Imaging studies whether contrast enhanced computed tomography (CT) scan or gadolinium enhanced Magnetic Resonance Imaging (MRI) are not very helpful for the determination of the nature of the disease but for staging. Indeed, SNUC appears as a heterogenous tumor with notable bony invasion and destruction similar to various other pathologies of this anatomic location. The most frequent being SCC, olfactory neuroblastoma, lymphoma, neuroendocrine cell tumors since only a biopsy with histopathological can confirm the diagnosis. [9][10].

# SNUC histologically originates from the epithelial cells that line the sinonasal tract called " Schneiderian epithelium" [10]. As mentioned previously, the diagnosis of SNUC can only be made through histological studies and so, under simple light microscopy SNUC cells appear as nests of intimidate-sized cells, with high mitotic rates, necrosis and vascular involvement. Immunohistochemical (IHC) analysis is the best way to confirm the diagnosis of SNUC and distinguish it from other mimics such as olfactory neuroblastoma or SSC. SNUC IHC typically shows high expression for both epithelial membrane antigen (EMA) and simple epithelial type cytokeratin (CK7, CK8, CK17) with inconsistent reactivity for tumor marker P53, synaptophysin, chromogranin and neurospecificity enolase (NSE). In addition, the majority of SNUC show no activity as regards to S-100 protein and vimentin [10]. The first discovery of SWI/SNF "switching/sucrose non-fermentable" gene was in 1984 in yeast, followed by the evolution of SWI/SNF complex (also known as BAF complex) encoded by multiple genes with a heterogenous structure. This complex is a large ATP-dependent chromatin remodeling complex [3]. The SWI/SNF complex has a major tumor suppressor role and hence, any somatic mutation ~~s~~ in this complex could potentially lead to the development of a neoplasm. Tumor genes like MYC, TP 53, PRCA 1, RB1 also somehow interact with the SWI/SNF complex any mutation of those genes can increase the risk of uncontrolled cell-proliferation. As high as 25% of all human cancers have some deficiency in the any of the 9 subunits SWI/SNF complex genes including SMARCA1, SMARCA2, SMARCB1(INI1), ARID1A, ARID1B, PBRM1, and ARID2 [3]. Located on chromosome 22q11.2, SMARCB1 is a vital tumor suppressor gene [3]. SMARCB1 gene mutations were first described in the literature in 1998 in rhabdoid tumors. According to a large meta-analysis of 10,849 patients only 5% of all human cancers had some form of SMARCB1 mutation. SMARCB1 mutations have been documented in many types of human malignancies some of which are malignant rhabdoid tumors, atypical teratoid rhabdoid tumor, renal medullary carcinoma, epithelioid carcinoma and many more [3]. It was found that the loss of SMARCB1 staining in IHC would give SMARCB1 deficient tumors unique features on pathology. SMARCB1 deficient SNUC have glandular features like (micro glandular, tubular, and signet-ring patterns) but more imperfectly they exhibit an obvious eosinophilic figure with a rhabdoid appearance followed by a blue basophilic figure [3].

# The significance of SMARCB1 deficiency is that it carries the worse prognosis for patients. It was found that SMARCB1 deficient tumors have the lower overall survival (OS), and highest rates of reoccurrence approximately 75% morbidity and mortality upfront and the remainder 14% after a short course [11]. SAMRCB1 SNUC treatment still has no guidelines due to its rarity, its treatment modalities are inspired from case reports and rare publications, and all agree on the importance of the patient’s management by a multidisciplinary team . Surgery followed by adjuvant concurrent chemo radiotherapy seems the most common treatment modality however the lack of sufficient studies to allow outcomes comparison makes it difficult to establish guidelines [12]. The regimen consisting of combining a platinum-based regimen with often a Taxane seems the most agreed upon amongst various case reports [13]. The role of immuncheckpoint inhibitor (ICI) in SMARCB1 deficient SNUC is yet to be studied but it was found that many patients have had PD-L1 expressed and showed promising response to ICI treatment.

# In our case we extrapolated from various reports. The very high ki 67 at presentation warranted the use of a topoisomerase inhibitor such as VP 16, we did not find data form Campotecan, although it might have been as efficient like in most neuroendocrine small or large cell tumors. The Taxane was nonetheless used down the road in consolidation.

# About the role of RT:

# Radiotherapy, particularly when following surgery, alone or combined with chemotherapy, has shown promise in the treatment of SMARCB1 (INI1)-deficient Sinonasal Undifferentiated Carcinomas. (SNUC). Studies have reported improved outcomes, including high rates of local control and overall survival – with multimodality approaches (14,15,16,17). The use on intensity modulated radiotherapy (IMRT) has been associated with decrease late toxicity and improve organ preservation (15). However, the prognosis for patients treated with definitive radiotherapy alone is less promising than for those who received surgery and postoperative radiotherapy (14). Therefore, a multimodality approach, with a focus on high-precision high dose radiotherapy, is recommended for the management of SNUC (16).

# Resteghini (2023) et al. discuss the integration of various radiotherapy modalities, including photon, proton, and carbon-ion-based radiotherapy, as part of a multimodal treatment approach for Sinonasal tumors, and carbon-ion-based radiotherapy, as part of a multimodal treatment approach for Sinonasal tumors (18). However, they do not provide a direct comparison of the efficacy or outcomes between proton and photon therapy alone. Therefore, it is not possible to determine if proton therapy has improved radiation therapy outcomes compared to photon therapy (17).

# **Conclusion:**

# In this report, we presented a very rare entity of sinonasal carcinoma with SMARC B1 deficiency, as a case report with a brief review of the available scarce literature. This entity having a molecular feature that has been linked to an aggressive clinical course, our patient was exceptionally lucky for attaining a durable complete remission until today. This was probably the result of multidisciplinary approach, such as the second generous resection and debulking during second surgery, and the concurrent approach of chemotherapy with radiotherapy, followed by consolidative chemotherapy to compensate the lost time owing to severe complication mid-way during therapy. This line of therapy used later seems the most common treatment modality reported in the literature, however the lack of sufficient studies to allow outcomes comparison makes it difficult to establish guidelines. This case highlights the need for multi-disciplinary approach for diagnosis and treatment. Further research and retrospective case review study is essential. Exploring targeted therapies and Immunotherapy for future cases by running NGS molecular profiling, could also be another way to improve outcomes for patients with this extremely rare genetic alternation.

# **Ethical statement:**

# **Data availability statement:**

# The underlying data supporting the results of our study are available upon request. Please contact the corresponding author Francois Kamar at [francois@kamarclinic.com](mailto:francois@kamarclinic.com) for access. We aim to ensure transparency and reproducibility of our findings, and we are committed to providing access to the data underlying our findings.

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# **Author contributions:**

# Hasan Numan\* and Jawad Zrein\*: Writing- original draft.

# \*Both authors contributed equally to this manuscript.

# Youssef Sultan: Writing – original draft.

Francois G. Kamar: Writing – review and editing, Visualization, Supervision and Project administration.

# Caroline Jabbour: Supervision.

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