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Case Report
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An Evolving and Rare Entity: SMARCB1(INI-1)-Deficient Sinonasal Carcinoma

SMARCB1 (INI-1) Eksikliği olan Sinonazal Karsinom

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Abstract: Malignant tumors of the paranasal sinuses and nasal cavity are rare. These tumors constitute very few of all head and neck tumors. With the developments in the field of molecular biotechnology, significant revisions have been made in The 2022 5th edition of the WHO Classification of the Head and Neck. Tumors with the definition of new entities. SWItch/Sucrose Non-Fermentable (SWI/SNF) complex-deficient carcinomas, which have been included as a separate entity under the general heading of sinonasal undifferentiated carcinomas, consist of two major subtypes caused by the loss of one of the SWI/SNF complex genes; SMARCB1-deficient sinonasal carcinoma and SMARCA4-deficient sinonasal carcinoma. The most common subtype is SMARCB1-deficient sinonasal carcinoma. These tumors have been misdiagnosed as neuroendocrine carcinoma, poorly differentiated carcinoma, sinonasal undifferentiated carcinoma and teratocarcinoma in previous years according to our current knowledge. Histopathologically, uniform cytologic features and appearance mimicking many tumors make it difficult to diagnose especially in small biopsies. Correctly naming this high-grade malignancy within the scope of molecular classification is important for treatment planning. Optimal treatment approaches are also limited. Although there is a consensus on radical resection/surgery followed by adjuvant treatment, the order of treatment may vary between institutions. Agents such as immune checkpoint inhibitors and EZH2 inhibitors are among the new treatment options. In this report, we present a case of SMARCB1-deficient sinonasal carcinoma according to the new molecular classification with recurrence at the age of 20. We aimed to emphasize the importance of histopathological and immunohistochemical findings and to raise awareness of the presence of this entity.
Keywords: Sinonasal carcinoma, SMARCB1, INI-1, SMARCB1 (INI-1) deficient, sinonasal undifferentiated carcinoma

Özet: Paranazal sinüsler ve nazal kavitenin malign tümörleri nadirdir. Tüm baş-boyun tümörlerinin çok azını oluşturur. Moleküler biyoteknoloji alanındaki gelişmeler ile Dünya Sağlık Örgütü Baş ve Boyun Tümörleri 2022 yılı 5.baskısında yeni antitelerin tanımlanması ile önemli değişiklikler yapılmıştır. Sinonazal indiferansiye karsinomlar genel başlığı altında ayrı bir antite olarak yerini alan SWItch/Sucrose Non-Fermentable (SWI/SNF) kompleksi eksikliği olan karsinomlar SWI/SNF kompleks genlerinin birinin kaybı ile oluşan iki majör subtipten oluşmaktadır; SMARCB1-eksikliği olan sinonazal karsinom ve SMARCA4-eksikliği olan sinonazal karsinom. En yaygın subtip SMARCB1- eksikliği olan sinonazal karsinomdur. Bu tümörler önceki yıllarda nöroendokrin karsinom, kötü diferansiye karsinom, sinonazal indiferansiye karsinom, teratokarsinom gibi bugünkü bilgilerimize göre yanlış tanılar almıştır. Histopatolojik olarak uniform sitolojik özellikler ve birçok tümörü taklit eden görünüm özellikle küçük biyopsilerde tanı koymayı güçleştirmektedir. Yüksek dereceli olan bu maligniteyi moleküler sınıflama kapsamında doğru olarak isimlendirmek tedavi planlaması açısından önemlidir. Optimal tedavi yaklaşımları da sınırlıdır. Radikal rezeksiyon/cerrahi ve sonrasında adjuvan tedavi konusunda fikirbirliği olsa da tedavinin sırası kurumlar arasında değişebilmektedir. İmmun kontrol noktası inhibitörleri ve EZH2 inhibitörü gibi ajanlar da yeni tedavi seçenekleri arasındadır. Bu sunumda 20 yaşında nüks ile seyreden yeni moleküler sınıflamaya göre "SMARCB1- eksikliği olan sinonazaal karsinom" olgusu ile histopatolojik ve immunohistokimyasal bulguların önemini vurgulamak ve bu antitenin varlığının farkındalığını artırmak istedik.
Anahtar Kelimeler: Sinonazal karsinom, SMARCB1, INI-1, SMARCB1 (INI-1) eksikliği, sinonazal indiferansiye karsinom

***Informed Consent:** Informed consent was obtained from the patient (with the consent form obtained preoperatively). In addition, the consent form prepared by the journal for case presentations was signed by the patient and this form was uploaded to the system.

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1. Introduction

Benign conditions (non-neoplastic and neoplastic) in the paranasal sinuses and nasal cavity are frequently encountered in our daily routine. Malignant tumors of this region are very rare, approximately 3-5% of all head and neck tumors (1). They usually have a poor prognosis. The most common types are squamous cell carcinoma and adenocarcinoma. Defined in the 2017 4th edition of the World Health Organization Classification of Head and Neck Tumors blue book, sinonasal undifferentiated carcinomas was a general title used for tumors without squamous and glandular features (2). In this classification, sinonasal malignancies included conventional squamous cell carcinoma, non-keratinized squamous cell carcinoma, adenocarcinoma (intestinal type and non-intestinal type), neuroendocrine carcinoma, poorly differentiated carcinoma, sinonasal undifferentiated carcinoma (as defined above) and other very rare subtypes (3). Today, with the developments in the field of molecular biotechnology, significant revisions have been made in the 2022 5th edition of the WHO Classification of the Head and Neck Tumors compared to the previous versions with newly added molecular groups. Under the general heading of sinonasal undifferentiated carcinomas, different malignancies were included as a separate entity; nuclear protein in testis (NUT) midline carcinoma (NMC), human papillomavirus (HPV)-related multiphenotypic carcinoma, SWItch/Sucrose Non-Fermentable (SWI/SNF) complex-deficient carcinomas (3). The latter entity consists of two major subtypes, SMARCB1-deficient sinonasal carcinoma and SMARCA4-deficient sinonasal carcinoma, which are caused by the loss of one of the SWI/SNF complex genes (4, 5). Most of such cases were misdiagnosed as neuroendocrine carcinoma, poorly differentiated carcinoma, sinonasal undifferentiated carcinoma and teratocarcinoma in previous years according to our current knowledge. SMARCB1-deficient sinonasal carcinoma has an aggressive course and since it is rare, few cases (approximately less than 200 cases) have been reported in the literature. In this article, we present a case of “SMARCB1 deficient sinonasal carcinoma” according to the new molecular classification in late adolescence and emphasize the importance of histopathological and immunohistochemical findings.

2.Case Presentation

2.1.Chief complaints: A 20-year-old female patient had intermittent epistaxis for the last 4 months and was admitted to the emergency department because of increased epistaxis for the last 3 days.

2.2.Personal history: She underwent ovarian cystectomy operation due to ovarian torsion 2 years ago. She has penicillin allergy.

2.3.History of illness and imaging examinations: Nasal examination in the emergency room revealed a necrotic mass in the right nasal passage originating from the middle meatus and leaning against the septum. Magnetic resonance imaging (MRI) showed a mass approximately 47x46x45mm in size extending into the nasopharynx infiltrating the turbinates in the nasal cavity, extending beyond the medial wall, extending into the maxillary sinus, ethmoid cells, terminating in the frontobasal, causing destruction of the olfactory fossa and foveo ethmoidalis, forming indentation into the intracranial space, and extending into the nasopharynx. PET-CT evaluation revealed a hypermetabolic expansile mass starting from the right half of the nasal cavity, infiltrating the septum in the midline, extending to the right lateral aspect of the nasopharynx and right maxillary sinus, showing indentation into the intracranial space, and reaching the sphenoid sinüs (Figure 1 and 2). Lymph nodes were seen in the cervical region without significant increase in metabolic activity. There were no findings in favor of metastatic lesions in all other body areas.

2.4.Histopathologic evaluation: The excision materials of the right nasal passage and the right middle mea mass were approximately 3 cm in the largest dimension and were grayish-brown, curated in places, with areas of bleeding. Histopathologic examination revealed fragments with indistinct sinonasal respiratory type mucosa. Immediately below the mucosa, an infiltrative tumor was (Figure 3) . The stroma was fibrotic with vascular structures in very narrow areas. Monomorphic appearance was dominant in most areas (Figurre 4). Nucleus size was significantly increased in the tumour cells and there was chromatin coarsening or prominent nucleolus (Figure 5). Diffuse mitotic figures were seen. Cells with eccentric nuclei and large acidophilic

cytoplasm were remarkable. Occasional bizarre pleormorphic cells were noticeable even at medium magnification. Foci of necrosis were seen intermingled with the tumor. The surface epithelium of all tissue samples was carefully evaluated for dysplasia. Immunohistochemically, CytoAE1/AE3, Cyto7, oscar keratin were positive (Figure 6). p63, p40, Cyto5/6, Cyto20, vimentin, desmin, S-100, Melan-A, SOX-10, LCA (CD45RO), synaptophysin, chromogranin, CD117, CD99 were negative. There was no staining with EBV and p16. Loss of SMARCB1 (INI-1) expression was demonstrated in tumor cells. Ki-67 proliferation index was found to be as high as 90%. With these findings, it was diagnosed as "poorly differentiated sinonasal carcinoma". Immunohistochemical INI-1 loss was interpreted in favor of SMARCB1-deficient sinonasal carcinoma. However, it was stated that NUT staining would be appropriate to clearly rule out the diagnosis of NUT carcinoma because he was a young patient (NUT antibody was not available in our laboratory during this period, so it could not be performed).

2.5.Treatment, Outcome and Follow-up: In the multidisciplinary head and neck study group, as a result of the evaluation of the patient with imaging studies, it was decided that the required chemoradiotherapy area was large and therefore only induction chemotherapy (3 cycles of DCF treatment -Dosetaxel+Cisplatin+5-Fluorouracil) should be administered to the patient. The patient was seen again by radiation oncology with response evaluation examinations and received concurrent chemoradiotherapy with cisplatin. Approximately seven months later, the cervical lymph node, which was suspicious on imaging studies, was evaluated by biopsy. Cisplatin + 5-Fluorouracil + Cetuximab treatment was started due to recurrence. The patient who received 4 cycles of chemotherapy with the last treatment was evaluated again in the head and neck study group with imaging examinations after treatment. Contrast-enhanced MRI revealed a space-occupying lesion with a transverse diameter of 18 mm at the level of the middle meatus on the right in the medial inferior orbital medial inferior to the maxillary osteum and ethmoidal infundulum. Right cervical lymph nodes were fusiform with a size of 18 mm. A mass lesion at the level of the right middle meatus was evaluated as recurrence. PET-CT suggested that the mass to the right of the nasal cavity might be compatible with recurrence. Lymph nodes were interpreted more in favor of reactive. However, due to the young age of the patient and the findings in favor of recurrence, a wide area operation including the mass material, right maxillary sinus, ethmoid, skull base, left submandibular gland and neck dissection was performed.

2.6.Histopathologic evaluation in recurrent tumor: The morphology of the mass material was similar to the tumor in the first operation material. In the young patient, auxiliary methods were again used for the differential diagnosis of sinonasal tumors and to clarify the molecular typing. The tumor was positive for cytoAE1/AE3, Cyto8/18, Cyto7. There was no staining with neuroendocrine markers except focal-faint CD56 positivity in one fragment. Lymphoid markers were also negative. CD99 was applied to exclude Ewing sarcoma/PNET group tumors and no staining was observed. Morphologically, squamous differentiation was not clearly seen, and squamous differentiation was excluded with p63, p40 and Cyto5/6 negativity. Although p63 and p40 negativity excluded NUT carcinoma, NUT-1 staining was performed for definitive diagnosis (it could not be performed at the time of the first biopsy). No staining with NUT-1. Loss of SMARCB1 (INI-1) expression in tumor cells was clearly demonstrated (Figure 7). There was no evidence of malignancy in other specimens. Lymph nodes were also reactive.

2.7.Molecular Final Diagnosis: SMARCB1-deficient sinonasal carcinoma was diagnosed with morphologic findings and immunohistochemical staining.

2.8.Outcome and Follow-up: The patient is still being followed up at 3-month intervals in the postoperative period.

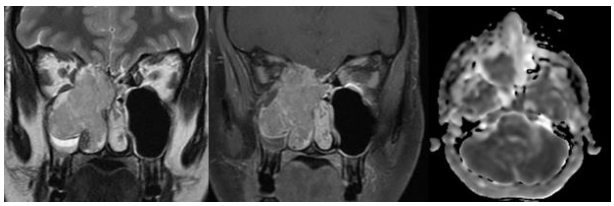

Figure 1. In coronal T2-weighted and post-contrast T1-weighted sections, invasion of the mass into the dura at the skull base and the extraconal space of the medial orbital wall is observed. In the axial ADC map, diffusion restriction (ADC value: $0.8x10^{-3}$), supporting a highly cellular and high-grade tumor, is noted.

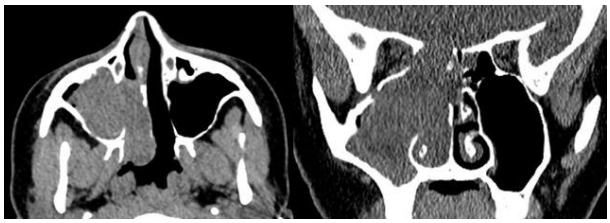

Figure 2. In axial and coronal non-contrast CT sections, a soft tissue mass is observed filling the right nasal cavity, extending into the conchae, ethmoid cells, and maxillary sinus, causing destruction in the medial wall of the maxillary sinus, medial orbital wall, nasal septum, and ethmoid roof.

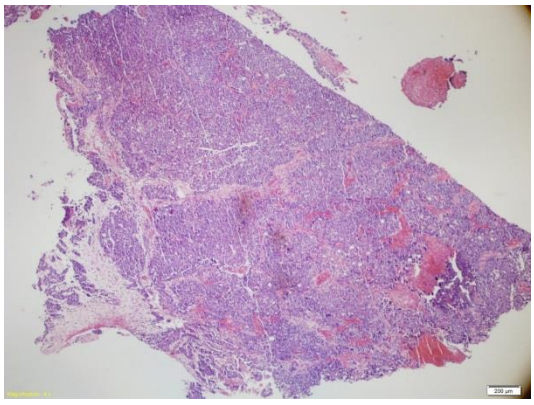

Figure 3. Tumor islands with a basaloid appearance in a diffuse pattern in the submucosal area.

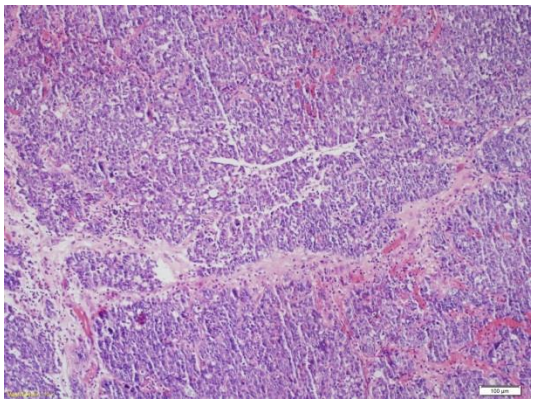

Figure 4. A diffuse, solid growth pattern with a monomorphic appearance was predominant in the majority of the tumor.

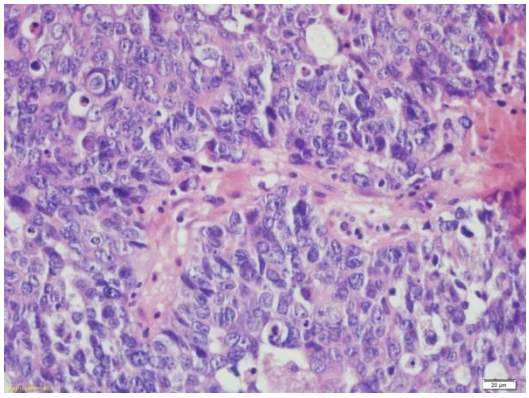

Figure 5. At higher magnification, tumor cells in certain areas exhibit chromatin clumping, prominent nucleoli, and marked pleomorphism.

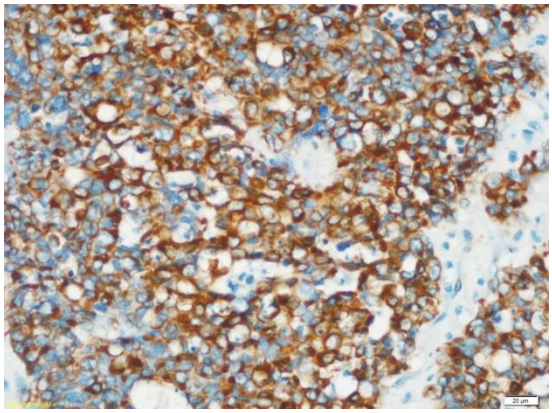

Figure 6: Immunohistochemically, tumour cells are positive for oscar keratin

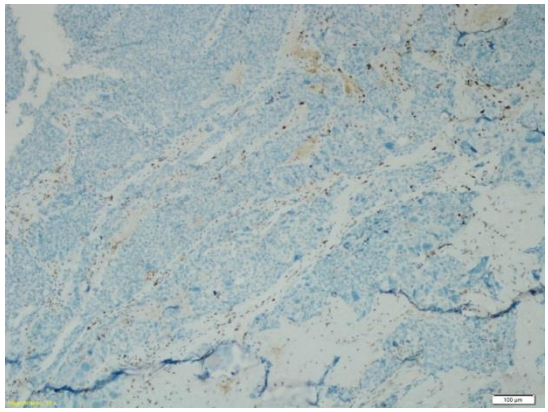

Figure 7: Sinonasal carcinoma with SMARCB1 deficiency is characterized by the loss of SMARCB1 (INI1) expression, while staining remains preserved in blood vessels and stroma.

3.Discussion

SMARCB1-deficient sinonasal carcinoma was first described in 2014 by two different authors in the same issue of the same journal in different papers (6, 7). Then, in 2017, "SMARCB1-deficient sinonasal carcinoma" was classified as a sinonasal undifferentiated carcinoma by WHO. With the developments in recent years, sinonasal carcinomas have been classified according to their new molecular profiles, as has been done in other malignancies. Thus, in the latest WHO edition of

head and neck tumors, separate entities were defined as NUT midline, SMARCA4-deficient, SMARCB1-deficient sinonasal carcinomas. The most common among these are sinonasal carcinomas that occur with loss of SMARCA4 and SMARCB1 by affecting SWI/SNF complex genes. SMARCB1 is a tumor suppressor gene located on chromosome 22q11. SMARCB1 loss is not only seen in SMARCB1-deficient sinonasal carcinomas. It can also be seen in various malignancies such as extraskeletal myxoid chondrosarcoma, myoepithelial carcinoma of soft tissues, malignant peripheral nerve sheath tumor (epithelioid type), epithelioid sarcoma. SMARCB1-deficient sinonasal carcinoma has two basic morphologic patterns including basaloid and plasmacytoid (8). The most common basaloid pattern is characterized by undifferentiated or "blue cell tumor" appearance in the form of solid layers, nests and trabeculae. The cytoplasm is usually very narrow. Prominent nucleolus is remarkable (9). The other pattern is the group of plasmacytoid/rhabdoid tumors. In contrast to the basaloid pattern, it has a "pink cell tumor" appearance. It is characterized by cells with eccentric nuclei and abundant abundant acidophilic cytoplasm. In general, rhabdoid cells are seen in almost all SMARCB1-deficient sinonasal carcinomas. Mitotic figures are very frequent. Necrosis is also a very common finding. With this appearance, it has highly aggressive histologic features. Prominent squamous and/or glandular differentiation is not an expected finding. However, squamous, squamous papillary, glandular (non-intestinal adenocarcinoma), clear-cell and yolk-salk pattern can be seen in decreasing rates. Carcinoma in situ or epithelial dysplasia is not seen in the surface epithelium.

Although SMARCB1-deficient sinonasal carcinoma is considered as a separate entity from undifferentiated sinonasal carcinoma with the new molecular classification, their morphologies overlap and they are defined as "small blue round cell tumor". Therefore, the morphological differential diagnosis spectrum includes many tumors including subtypes of undifferentiated sinonasal carcinoma, poorly differentiated sinonasal carcinoma, NUT carcinoma, lymphoma types, melanoma, rhabdomyosarcoma, olfactory neuroblastoma, Ewing sarcoma, and tumors with rhabdoid morphology. In our case, the tumors mentioned above were included in the differential diagnosis. The absence of surface epithelial dysplasia (including carcinoma in situ) in the microscopic evaluation morphologically excluded poorly differentiated squamous cell carcinoma and basaloid squamous cell carcinoma. Immunohistochemically, these tumors are reactive with p63, p40, Cyto5/6. These antibodies were negative in our case. Thus, we completely excluded the diagnosis of squamous cell carcinoma. Similarly, in melanoma, we usually expect to see the in situ component at least in one area. In addition, positivity with SOX-10, Melan-A and S-100 is compatible with melanoma. In our case, there was no in situ component and the negativity of the above-mentioned melanoma markers led us away from the diagnosis of melanoma. In terms of differential diagnosis, morphologically Ewing sarcoma was also an entity that should be excluded. Ewing sarcoma has diffuse membranous staining with CD99. CD99 was negative in our case. The neuroendocrine markers we applied to differentiate possible neuroendocrine carcinoma were negative. Loss of SMARCB1 (INI-1) expression in tumor cells was clearly seen. The staining of nonneoplastic lymphocytes and endothelium of vascular structures in the background was considered as internal control. Another tumor with loss of SMARCB1 (INI-1) in the head and neck region is malignant rhabdoid tumor. However, this tumor is almost always seen in children under 3 years of age. Differential diagnosis is difficult especially in small biopsies. The presence of a characteristic paranuclear dot-like pattern with vimentin supports malignant rhabdoid tumor. There was no staining with vimentin in our case. Negative lymphoid markers also excluded the possibility of lymphoma. In conclusion, we eliminated many tumors in the differential diagnosis with the loss of SMARCB1 (INI-1) expression.

A systematic review including 128 cases of SMARCB1-deficient sinonasal carcinoma was recently published by Lee et al. (10). Although nodal metastasis was approximately 6%, it was found to be at a regionally advanced stage at the time of diagnosis. Metastatic status was similar to lymph node metastasis. This study is the largest series to date. Since SMARCB1-deficient sinonasal carcinoma cases are rare, the optimal treatment approach is limited by institutional experience. Radical resection/surgery followed by adjuvant treatment has been recommended in large series (10, 11). Although there is a consensus on multimodal treatment, there is no complete agreement on the sequence of this treatment. With the definition of such new

entities, targeted treatment possibilities are also on the agenda. Although immune checkpoint inhibitors are increasingly being used in head and neck squamous cell carcinomas, data on their efficacy in SMARCB1-deficient sinonasal carcinomas are very limited. In a recent study, two cases of SMARCB1-deficient sinonasal carcinoma with and without immunotherapy were compared (12). The patient who received immunotherapy (anti-PD1-tislelizumab) had a longer disease-free survival. However, it needs to be supported by more clinical evidence. When SWI/SNF complex function is impaired in these tumors, EZH2 activity increases. EZH2 also promotes the oncogenic pathway. The EZH2 inhibitor Tazemetostat (EPZ-6438) may be an effective agent in SMARCB1-deficient sinonasal carcinomas (13). A study evaluating the antitumor effect of tazemetostat in the treatment of SMARCB1-deficient sinonasal carcinomas has begun (14).

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Conclusions

SMARCB1-deficient sinonasal carcinoma is a newly described rare tumor with a very aggressive clinical course. Histopathologically, the uniform cytological features and appearance mimicking many tumors make the diagnosis difficult, especially in small biopsies. It is very important to correctly name this high-grade malignancy within the scope of molecular classification. Accurate and early diagnosis, multimodality management, evaluation of new and targeted treatment options may improve the poor prognosis and survival.

REFERENCES

1. Contrera KJ, Woody NM, Rahman M, Sindwani R, Burkey BB. Clinical management of emerging sinonasal malignancies. Head Neck. 2020;42(8):2202-2212.
2. Thompson LDR, Franchi A. New tumor entities in the 4th edition of the World Health Organization classification of head and neck tumors: nasal cavity, paranasal sinuses and skull base. Virchows Arch. 2018;472(3):315-330.
3. Thompson LDR, Bishop JA. Update from the 5th edition of the World Health Organization classification of head and neck tumors: nasal cavity, paranasal sinuses and skull base. Head Neck Pathol. 2022;16(1):1-18.
4. Rooper LM, Uddin N, Gagan J, et al. Recurrent loss of SMARCA4 in sinonasal teratocarcinosarcoma. Am J Surg Pathol. 2020;44(10):1331-1339.
5. Agaimy A, Jain D, Uddin N, Rooper LM, Bishop JA. SMARCA4-deficient sinonasal carcinoma: a series of 10 cases expanding the genetic spectrum of SWI/SNF-driven sinonasal malignancies. Am J Surg Pathol. 2020;44(5):703-710.
6. Agaimy A, Koch M, Lell M, Semrau S, Dudek W, Wachter DL, et al. SMARCB1(INI1)-deficient sinonasal basaloid carcinoma: a novel member of the expanding family of SMARCB1-deficient neoplasms. Am J Surg Pathol. 2014;38(9):1274-1281.
7. Bishop JA, Antonescu CR, Westra WH. SMARCB1 (INI-1)-deficient carcinomas of the sinonasal tract. Am J Surg Pathol. 2014;38(9):1282-1289.
8. Parsel SM, Jawad BA, McCoul ED. SMARCB1-deficient sinonasal carcinoma: systematic review and case report. World Neurosurg. 2020;136:305-310.
9. Agaimy A, Hartmann A, Antonescu CR, et al. SMARCB1 (INI-1)-deficient sinonasal carcinoma: a series of 39 cases expanding the morphologic and clinicopathologic spectrum of a recently described entity. Am J Surg Pathol. 2017;41(4):458-471.
10. Lee VH, Tsang RK, Lo AWI, et al. SMARCB1 (INI-1)-deficient sinonasal carcinoma: a systematic review and pooled analysis of treatment outcomes. Cancers (Basel). 2022;14(13):3285.
11. Chung SY, Kenee P, Mitton T, Halderman A. SMARCB1(INI-1)-deficient sinonasal carcinoma: an evolving entity. J Neurol Surg Rep. 2023;84(1):e1-e5.
12. Zhang L, Gao AX, He YL, Xu MJ, Lu HJ. Immunotherapy in SMARCB1 (INI-1)-deficient sinonasal carcinoma: two case reports. World J Clin Cases. 2023;11(32):7911-7919.
13. Agaimy A. Proceedings of the North American Society of Head and Neck Pathology, Los Angeles, CA, March 20, 2022: SWI/SNF-deficient sinonasal neoplasms: an overview. Head Neck Pathol. 2022;16(1):168-178.
14. Lee VHF. Induction chemotherapy and tazemetostat for locally advanced SMARCB1-deficient sinonasal carcinoma. Available online: https://clinicaltrials.gov/ct2/show/NCT05151588. Accessed May 15, 2022.