Clinical characteristics and management of primary granulocytic sarcoma of the oral cavity
A case report and literature review

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Abstract

Introduction: Granulocytic sarcoma (GS) is a commonly occurring tumor comprising immature myeloid cells, which are usually related to acute or chronic myelocytic leukemia. The tumor rarely precedes leukemia without bone marrow involvement and is called primary GS. Although primary GS can occur in any body part, the involvement of the oral cavity is uncommon.

Patient concerns: A 49-year-old woman hospitalized at the Department of Plastic and Maxillofacial Surgery presented with a growing mass in her left maxillary hard palate dating two months back. No obvious physical findings were noted during general examination. She was diagnosed with an oral ulcer at a local clinic, and received antibiotics. However, the symptoms did not improve; the mass became bigger and painful.

Diagnosis: An incisional biopsy of the oral mass was performed, the immunohistochemistry showed that the tumor cells tested positive for myeloperoxidase, CD4, BCL-2, Ki-67. Bone marrow aspiration was negative for malignant cells, and the laboratory test results revealed only monocytosis. Standard bone marrow cytogenetic analysis showed a normal karyotype and leukemia-related fusion gene detection was normal. Therefore, the final diagnosis was intraoral primary GS.

Interventions: The patient was treated with a chemotherapy regimen based on idarubicin and cytarabine arabinoside.

Outcomes: After 2 cycles of idarubicin and cytarabine arabinoside regimen chemotherapy, the patient achieved complete remission. The tumor was barely visible in the left maxillary hard palate. There has been no evidence of disease spread and progression after 1 year of follow-up.

Conclusions: Careful morphological and immunohistochemical analyses, correlating with clinical data are necessary to establish the diagnosis of oral primary GS. Early aggressive systemic chemotherapy can effectively relieve symptoms, significantly reducing primary GS conversion into acute myelocytic leukemia and prolonging overall survival.

Abbreviations: AML = acute myelocytic leukemia, BMT = bone marrow transplantation, GS = granulocytic sarcoma, HSCT = hematopoietic stem cell transplantation, IHC = immunohistochemistry, MPO = myeloperoxidase, MS = myeloid sarcoma.

Keywords: primary granulocytic sarcoma, oral tumor, clinical characteristics

1. Introduction

Granulocytic sarcoma (GS) is a malignant tumor derived from the bone marrow and located outside the bone marrow. The tumor is also called extramedullary myeloid tumor, or myeloid sarcoma (MS), which generally presents with a green color due to the presence of myeloperoxidase (MPO).[1,2] GS usually occurs with acute myelocytic leukemia (AML), myelodysplastic syndrome, myeloproliferative neoplasm or as a recurrence of AML. In majority of conditions, it occurs after the onset of leukemia. When it precedes leukemia without any apparent symptoms, it is termed as the primary or isolated type. It is reported that the ratio of primary GS reaches about 0.85% to 2.2% in patients with AML. It has a high risk of progression to AML within months or years.[3–5] GS can be found in any part of the body but more predominantly in the skin, soft tissues, and lymph nodes. Oral cavity GS rarely occurs, and the clinical characteristics are varied and usually atypical.[6–7] Therefore, it is crucial for oral clinicians to be able to accurately diagnose GS, which ensures prompt treatment to control the disease progression and reach a stable remission period.

Here, a rare case of primary GS is reported, presenting with intraoral involvement, including the mucosa of the left maxillary hard palate, and the adjoining edentulous region. Relevant literature was also reviewed to provide clinicians with additional clarifications on the clinicopathologic manifestation, differential diagnosis, treatment regimens, and prognosis of primary GS of the oral cavity.
2. Case report

A 49-year-old woman hospitalized at the Department of Plastic and Maxillofacial Surgery, presented with a growing mass in her left maxillary hard palate dating 2 months back. She was diagnosed with an oral ulcer at a local clinic, and received antibiotics. However, the symptoms did not improve; the mass became bigger and painful. Her past medical history was unremarkable without any systemic disease. No obvious physical findings were noted during general examination. She had no bouts of fever, chills, night sweats, vomiting, or weight loss. The intraoral examination revealed that there was a mass measuring 3.0cm \( \times \) 2.0cm in size, with ulcerated surface mucosa in the left maxillary hard palate (Fig. 1).

To obtain a definitive diagnosis, an incisional biopsy of the oral mass was performed under local anesthesia. Histologic examination using hematoxylin and eosin staining revealed that there was diffuse cell infiltration growth. Most cells were large, with vacuolated nuclei, obvious nucleoli and a basophilic cytoplasm containing granules (Fig. 2). For the final diagnosis, immunohistochemistry (IHC) was performed, and the tumor cells tested positive for MPO, CD4, BCL-2, KI-67 (Fig. 3), and CD117 and negative for CD3, CD5, CD20, CD56, bcl-6, Mum-1, CD123, MUM-1, TdT, Syn, SOX11, and C-myc. These results supported the diagnosis of GS.

The patient was then referred to the Hematology Department. Bone marrow aspiration was negative for malignant cells, and the laboratory test results revealed only monocytosis. Standard bone marrow cytogenetic analysis showed a normal karyotype and leukemia-related fusion gene detection was normal. Therefore, according to IHC and bone marrow aspiration results in combination with morphological features, the final diagnosis was intraoral primary GS.

A chemotherapy regimen comprising idarubicin 8mg VD d1–3 and cytarabine arabinoside 150mg VD d1–7 idarubicin and cytarabine arabinoside was initiated. During chemotherapy, the patient only appeared mild nausea, vomiting and canker sores (grade II). These symptoms gradually eased without special therapy. After 2 cycles of idarubicin and cytarabine arabinoside regimen chemotherapy, the patient achieved complete remission. The tumor was barely visible in the left maxillary hard palate (Fig. 4). The patient denied consolidation therapy or bone marrow transplant. Nevertheless, there has been no evidence of disease spread and progression after 1 year of follow-up.

3. Discussion

GS is an extramedullary solid tumor composed of immature myeloid cells. First described by Burns in 1811, then in 1853, King named GS as “chloroma,” due to its green colored appearance when exposed to air, resulting from the presence of myeloperoxidase in the tumor cells. In 1966, Rappaport formally proposed the concept of GS. The classification includes non-leukemic GS (isolated or primary GS) and leukemic GS (extramedullary infiltration of leukemia).[8,9]

Primary GS occurs in approximately 2 per million persons. The oral cavity occurrence of primary GS is extremely rare. The clinical characteristics of oral primary GS are variable and usually nonspecific.[10] It is a challenge to diagnose oral primary GS based on symptoms and through routine examination. To improve the awareness of the disease, we retrospectively analyzed all cases of oral primary GS, which the tumor precedes leukemia without bone marrow involvement. There are only 31 cases published in PubMed literature together with our case (Table 1).[11–41] The average age of the patients was 44 years (ranging from 2 to 89 years), with a predilection for females (2:1). The tumor was mostly isolated. Commonly involved sites were the gingiva (40.6%), mandible (21.9%), and hard palate (15.6%). Only a few involved the lip, buccal mucosa, and multiple sites. The most common clinical feature of oral primary GS is a painful swelling or a nodule with a reddish to brownish ulcerated surface. In addition, imaging changes that present as soft tissue-occupying lesions, with or without bone erosions, are atypical.[42–43] Therefore, it is usually misdiagnosed as a dental ulcer, epulis and gingival hyperplasia, as well as malignant neoplasms. The challenge to dental practitioners...
is in differentiating malignant, infectious, and inflammatory lesions, which can often have overlapping clinical features.[44] To accurately diagnose oral primary GS, it is usually based on histopathological and immunohistochemical analysis, and a history of symptoms associated with hematological diseases that might be absent. Morphologically, oral primary GS exhibits variable numbers of primitive, poorly differentiated cells with granular cytoplasm, round to oval nuclei with well-defined membrane and prominent nucleoli, intermingled with reactive inflammatory infiltrate.[45] Different phases of myeloid differentiation are shown in tumor cells containing the eosinophilic myelocytes and blastic cells with minimal granulocytic differentiation. It seems difficult to differentiate the histopathological diagnosis of GS from that of large B-cell lymphoma, Burkitt lymphoma, lymphoblastic lymphoma, and poorly differentiated squamous cell carcinoma. IHC can improve diagnostic accuracy.[16,25] IHC proves the granulocytic origin of tumor cells, which are usually positive for MPO, lysozyme, CD34, CD45, CD68, CD117, and some important markers for the diagnosis of oral primary GS (Table 1). Thereinto, MPO and lysozyme are specific markers of oral primary GS and are associated with the process of tumor cell differentiation.[3,6,45,46]

For nonleukemic GS, it is critical to comprehensively analyze both morphological and immunophenotypic results. In the present
case, the pathomorphological examination was nonspecific, but IHC showed positive for EBER, MPO, CD4, BCL-2, CD117, which hinted that tumor cells were from the myeloid cells. IHC was negative for CD3 and CD20. This did not support the source of T cells. Therefore, the definitive diagnosis was GS. Moreover, before the diagnosis of primary GS, we viewed the bone marrow image, chromosome karyotype analysis, and fusion gene detection. Bone marrow changes occurred before peripheral blood changes, and chromosome karyotype analysis and fusion gene detection could prompt diagnosis when there was no obvious abnormality in bone marrow cell morphology.49

With regards to available therapeutic options, there is an absence of a treatment guideline for primary GS with the recommended treatment regimen being conventional AML type chemotherapeutic protocols. As Table 2 shows, majority of patients have shown complete remission of the disease after the advent of Ara-c and anthracycline-based therapeutic regimens. It is recommended that early aggressive systemic chemotherapy contributes in the control of the progression of the disease and lengthens survival.50,51

| Cases (first author, year) | Age/yr/ Sex | Location | Size(cm) | Symptoms | Diagnosis basis |
|---------------------------|-------------|----------|----------|----------|----------------|
| Brooks, 197419 | 8/M | Maxilla R | — | Nodular right upper maxilla tumor | HE |
| Conran, 198222 | 2/F | Mandible R | 2 x 3cm | Swelling of the lower right mandible | HE |
| Takagi, 198320 | 25/F | Gingiva L | 4 x 1cm | Swelling of the gingiva with pain | IHC - MPO |
| Reichart, 198414 | 35/F | Mandible R | 1.5 x 1.5cm | Brownish color tumor | CS - chloracetate esterase |
| Castella, 198415 | 89/F | Hard palate | 2 x 1cm | Exophytic, ulcerated gray-white lesion | CS - chloracetate esterase |
| Rodriguez, 199016 | 56/M | Gingiva L | 5 x 3cm | Exophytic, reddish lesion with pain | IHC - lysozyme |
| Eisenberg, 199113 | 33/M | Multiple gingiva | — | Multiple, raised, granular-appearing, red nodules | CS - Sudan black, MPO |
| Menasse, 199918 | 63/F | Gingiva | — | NR | IHC - MPO |
| Tong, 200221 | 76/F | Gingiva R | 4cm in diameter | Diffuse, ulcerative, granular-appearing lesion | IHC - MPO, CD45 |
| Koudstaal, 200626 | 36/M | Gingiva | 4cm in diameter | Periappendicular granuloma and chronic abscesses | IHC - MPO, CD43, CD15 |
| Amten, 200322 | 12/G | Gingiva | 4 x 3cm | Bright red, soft, friable, edematous mass | IHC - MPO, lysozyme |
| Colella, 200527 | 62/F | Gingiva | — | Large swelling of the upper vestibular region | IHC - MPO, lysozyme, CD45 |
| Koudstaal, 200626 | 36/M | Hard palate | 1 x 3cm | Blue-gray, mucosa infiltrat, normal texture | IHC - CD45, CD43, HLA-DR |
| Gateri, 200626 | 84/F | Hard palate | — | Ulcerated, nodular, infiltrative mass | IHC - MPO, CD45, CD43, CD34 |
| Yenin, 200028 | 44/F | Gingiva R | — | Progressive enlargement mass with pain, ulcer | IHC - MPO, CD68 |
| Lu, 200029 | 63/F | Gingiva R | — | Pus mass, surface graininess, easily bleeding | IHC - MPO, CD34, Bcl-2 |
| Qi, 201030 | 16/F | Condyle L | — | Preauricular swelling, restriction mouth open | IHC - MPO |
| Cokovi, 201131 | 55/M | Mandible L | — | Large mucosal tissue swelling | IHC - CD117, CD45, CD68, lysozyme |
| Guastafierro, 201330 | 56/F | Gingiva | — | Large swelling in the upper vestibular region | IHC - MPO, CD45, CD68, lysozyme |
| Mei, 201331 | 56/M | Maxilla L | 4cm in diameter | Soft and solid mass | IHC - CD34, CD45, CD65, CD117, MPO |
| Chaudhuri, 201332 | 60/M | Lip | — | Non tender, firm, lumpy swelling | CS - Immature myeloid cells |
| Sharma, 201733 | 9/M | Maxilla L | 3 x 3cm | Single ill-defined, diffuse swelling | IHC - CD31, MPO, vimentin, CD99 |
| Ponnam, 201735 | 45/F | Gingiva L | 5 x 5cm | Lobulated, firm, non tender and erythematous growth | IHC - CD45, CD68, CD117, MPO |
| Mostref, 201436 | 45/M | Gingiva, Palate | — | Red and soft with irregular surfaces | IHC - CD45, CD117 |
| Wang, 201437 | 27/M | Buccal mucosa L | 3cm in diameter | Firm mass without tenderness | IHC - MPO, CD34, CD68, lysozyme |
| Dinesh Kumar, 201538 | 62/F | Gingiva | — | Gingival enlargement without purulent discharge | IHC - MPO, CD43 |
| Sengupta, 201639 | 2/M | Mandible L | 4 x 2.5cm | Firm to hard, circumscribed, mildly tender swelling | IHC - CD45, CD68, lysozyme |
| Abeelbassam, 201740 | 67/F | Hard palate | 4 x 5cm | Red painless swelling | IHC - MPO, CD43, CD117 |
| Kumar, 201741 | 28/M | Mandible L | — | Ill defined bony hard swelling | IHC - CD45, MPO |
| Shen, 201842 | 41/F | Gingiva | — | Blue-gray discoloration gradually developed | IHC - MPO, CD68, CD117, Ki-67 |
| Present case | 49/M | Hard palate | 3 x 2cm | Ulcerated surface mucosa | IHC - MPO, CD4, Bcl-2, CD117 |

CS = cytochemical staining, F = female, HE = histologic examination, IHC = immunohistochemistry, L = left, M = male, NR = not record, R = right.

Table 1: Clinical characteristics and diagnosis of reported cases with primary oral cavity GS.

Whether surgery depends on the size change of the tumor after radiotherapy and chemotherapy is unclear.

Although systemic chemotherapy can reduce the risk of GS conversion to AML, it cannot completely stop the progression of AML (averagely 10.5 months).12 At the same time, chemotherapy has side effects such as cardiotoxicity and myelosuppression. Some patients die of sepsis before the end of chemotherapy.11,22,29 Therefore, choosing a safe and effective treatment method that will prolong long-term survival is difficult. In recent years, with the development of bone marrow transplantation (BMT) and hematopoietic stem cell transplantation (HSCT) technology, the treatment of primary GS has entered the era of comprehensive treatments such as combined radiotherapy and chemotherapy with BMT/HSCT. While there are no prospective trials evaluating the role of BMT/HSCT in primary GS, some retrospective studies have shown good results with a 5-year survival rate of 48% and even encourage considering allogenic BMT/HSCT after the patients' first induction of remission.53-55

Targeted therapy is a new orientation of primary GS treatment. These agents include histone deacetylase inhibitors, DNA methyltransferase inhibitors, FLT3 inhibitors, and farnesyl-transferase inhibitors. However, it was most reported by cases lacking multicenter controlled trials.50,56 With the deepening of the study of primary GS molecular mechanisms, targeted therapy may be a new effective therapy.
Table 2

| Cases (first author, years) | Age(yr)/Sex | Treatment | Remission/mo | Progression/mo | Retreatment | Outcome/mo |
|----------------------------|-------------|-----------|--------------|---------------|-------------|------------|
| Brooks, 1974[14]           | 8/M         | RT        | —            | —             | —           | A&W/48     |
| Conran, 1982[12]           | 2/F         | RT + CT   | CR/2         | —             | —           | A&W/16     |
| Takagi, 1983[13]           | 35/F        | Surgery + CT | CR/4       | AML/18        | CT + RT     | DOD/24     |
| Castella, 1984[14]         | 25/F        | CT        | —            | AML/18        | CT          | DOD/13     |
| Rodríguez, 1990[16]        | 56/M        | CT        | —            | AML/8         | CT          | DOD/2      |
| Jordon, 1991[17]           | 36/M        | CT        | —            | AML/24        | CT          | DOD/10     |
| Tong, 2000[18]             | 76/F        | CT + RT   | R2           | AML/7         | NR          | DOD/17     |
| Lee, 2001[19]              | 43/F        | CT + RT   | CR/6         | —             | —           | A&W/6      |
| Moshref, 2014[20]          | 62/F        | CT        | —            | MML/2         | CT          | DOD/7      |
| Koudstaal, 2006[21]        | 36/M        | CT + RT   | CR/24        | Bone marrow relapse | CT       | NR         |
| Goteri, 2007[22]           | 84/F        | Surgery + RT | CR/-        | —             | —           | A&W/7      |
| Lu, 2009[23]               | 69/F        | CT        | CR/-         | —             | —           | A&W/6      |
| Qin, 2010[24]              | 16/F        | Surgery + CT | CR/2        | —             | —           | NR         |
| Colovic, 2011[25]          | 55/F        | CT        | —            | —             | —           | DOD/17     |
| Guastafiore, 2013[26]      | 56/F        | CT        | —            | —             | —           | DOD/7      |
| Mel, 2013[27]              | 56/M        | Surgery + CT | CR/4        | —             | —           | A&W/5      |
| Chaudhuri, 2013[28]        | 60/M        | RT        | CR/8         | —             | —           | A&W/24     |
| Sharma, 2014[29]           | 9/M         | CT + RT   | CR/5         | —             | —           | DOD/2      |
| Ponnam, 2014[30]           | 45/F        | CT        | —            | —             | —           | A&W/6      |
| Mohtel, 2014[31]           | 45/M        | CT        | CR/2         | —             | —           | A&W/15     |
| Wang, 2015[32]             | 27/M        | CT + RT   | CR/2         | —             | —           | A&W/15     |
| Dinesh Kumar, 2016[33]     | 62/F        | NR        | —            | —             | —           | NR         |
| Sengupta, 2016[34]         | 2/M         | CT        | CR/2         | —             | —           | A&W/12     |
| Aboelhassan, 2017[35]      | 67/F        | Surgery + CT | —           | Multiple occurrence | CT       | NR         |
| Shen, 2018[36]             | 41/F        | CT        | R4           | —             | —           | A&W/12     |
| Present case               | 49/F        | CT        | CR/2         | —             | —           | A&W/12     |

A&W = alive and well, AML = acute myeloid leukemia, CR = complete remission, CT = chemotherapy, DOD = die of disease, F = female, M = male, MML = myelomonocytic leukemia, NR = not record, R = remission, RT = radiotherapy.

4. Conclusion

Oral primary GS is a rare tumor with poor clinical outcome. It has protean clinical manifestations and histological overlap with numerous tumors making it a diagnostic challenge for clinicians and pathologists alike. Careful morphological and immunohistochemical analyses, correlating with clinical data are necessary to establish the diagnosis of oral primary GS. Early aggressive systemic chemotherapy can effectively relieve symptoms, significantly reducing primary GS conversion into AML and prolonging overall survival. However, there is no uniform standard on radiotherapy whether combined with systemic chemotherapy synchronously or after the therapy. There is no verdict that BMT/HSCT should be used as preferred alternative for the treatment of disease progression after chemoradiotherapy. Moreover, there is a lack of effective management over long-term treatment. Considering the abovementioned findings, there is a need for further studies to be done.

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References

[1] Stoopher Eric T, Pinto Andres, Alawi Faizan, et al. Granulocytic sarcoma: an atypical presentation in the oral cavity. Spec Care Dentist 2004;24:65–9.
[2] Campidelli Cristina, Agostinelli Claudio, Stitson Richard, et al. Myeloid sarcoma: extramedullary manifestation of myeloid disorders. Am J Clin Pathol 2009;132:426–37.
[3] He Jingsong, Zhu Lixia, Ye Xiujin, et al. Clinical characteristics and prognosis of nonleukemic myeloid sarcoma. Am J Med Sci 2014;347:434–8.
[4] Lou Yinjun, Qian Wenbin, Meng Haitao, et al. Frequent extramedullary recurrence of isolated myeloid sarcoma in the long-term follow-up. Ann Hematol 2012;91:1317–9.
[5] Vardiman James W. The World Health Organization (WHO) classification of tumors of the hematopoietic and lymphoid tissues: an overview with emphasis on the myeloid neoplasms. Chem Biol Interact 2010;184:16–20.
[6] Yilmaz Asu F, Saydam G, Sahin F, et al. Granulocytic sarcoma: a systematic review. Am J Blood Res 2013;3:263–70.
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[30] King A. A case of chloroma. Monthly J Med 1853;17:97.

[32] Chaudhuri T, Paul S, Srivastava K. Primary granulocytic sarcoma of lip – a rare extramedullary presentation of myeloid leukemia. Indian J Med Paediatr Oncol 2013;34:126–7.

[35] Ponnamp SR, Srivastava G, Jampani N, et al. A fatal case of rapid gingival enlargement: case report with brief review. J Oral Maxillofac Pathol 2014;18:121–6.

[36] Mohsref M, Lotfi A, Mashhadi-Abbas F, et al. Granulocytic sarcoma (chloroma) presenting as multiple sites in oral cavity: report of a case. Iran J Cancer Prev 2014;7:53–7.

[37] Wang CC, Chang KP, Chang MS, et al. Isolated myeloid sarcoma of the buccal region. Br J Hosp Med (Lond) 2014;75:468–9.

[38] Dineshkumar T, Suresh V, Ramya R, et al. Primary intraoral granulocytic sarcoma: a rare case presenting as generalized gingival enlargement. J Oral Maxillofac Pathol 2016;20:523–6.

[39] Sengupta M, Das I, Chatterjee U, et al. De novo myeloid sarcoma involving mandible in a child: report of a rare occurrence. J Oral Maxillofac Pathol 2015;19:304–7.

[40] Abolhasan M, Ali HA, Mohammed A, et al. Management of hard palate fistula caused by granulocytic sarcoma: case report. Gulf J Oncol 2017;1:72–6.

[41] Menasce LP, Banerjee S, Melis C, et al. Clinicopathological characteristics of de novo and secondary myeloid sarcoma: a monocentric retrospective study. Eur J Haematol 2018;100:603–12.

[42] Meyer HJ, Pönisch W, Schmidt SA, et al. Clinic and imaging features of myeloid sarcoma: a German multicenter study. BMC Cancer 2019;19:1150.

[43] A practical approach to diagnose soft tissue myeloid sarcoma preceding or coinciding with acute myeloid leukemia.

[44] Markow Fatma, Rozdogan Nazan, Yuksuk Fuun Ardic, et al. Granulocytic sarcoma: difficulties in diagnosis. Tumori 2010;96:149–53.

[45] Mourad W, Kfouri H, Al Hussein H. The value of CD34, myeloperoxidase and chloroacetate esterase (Leder) stain in the diagnosis of granulocytic sarcoma. Ann Saudi Med 2001;21:287–91.

[46] Amador-Oritz C, Hurley MY, Ghahramani GK, et al. Use of classic and novel immunohistochemical markers in the diagnosis of cutaneous myeloid sarcoma. J Cutan Pathol 2011;38:945–53.

[47] Chang CC, Eschoa C, Kampalath B, et al. Immunophenotypic profile of myeloid cells in granulocytic sarcoma by immunohistochemistry. Correlation with blast differentiation in bone marrow. Am J Clin Pathol 2000;114:807–11.

[48] Alexiev BA, Wang W, Ning Y, et al. Myeloid sarcomas: a histologic, immunohistochemical, and cytogenetic study. Diagn Pathol 2007;2:42.

[49] Avni R, Koren-Mitchowitz M. Myeloid sarcoma: current approach and therapeutic options. Ther Adv Hematol 2012;3:309–16.

[50] Lan TY, Lin DT, Tien HF, et al. Prognostic factors of treatment outcomes in patients with granulocytic sarcoma. Acta Haematol 2009;122:234–46.

[51] Churgari C, Jacob J, Bauduceau O, et al. Granulocytic sarcoma in a nonleukemic patient: place of radiotherapy and systemic therapies. Case Rep Med 2011;2011:92961.

[52] Tan D, Wong GC, Koh LP, et al. Successful treatment of primary granulocytic sarcoma by non-myeloablative stem cell transplant. Leuk Lymphoma 2006;47:159–62.

[53] Chevallier P, Labopin M, Cornelissen J, et al. ALWP of EBMT: Allogeneic hemato-poietic stem cell transplantation for adult AML patients with granulocytic sarcoma. Cell Transplant 2011;20:929161.

[54] Connolly A, Xue J, Eshoa C, et al. A case of granulocytic sarcoma occurring in the oral cavity: a report of a rare occurrence. J Oral Maxillofac Pathol 2016;20:304–7.

[55] Shimizu H, Saitoh T, Tanaka M, et al. Allogeneic hemato-poietic stem cell transplantation for adult AML patients with granulocytic sarcoma. Cell Transplant 2011;20:929161.