

Maxillary Metastasis from Rectosigmoid Adenocarcinoma: A Case Report and Review of the Literature

Snikah Jihane^{1*}, Choumi Faiçal², Alilou Mohammed¹, Moumine Mohammed²

¹Resident, Department of Reconstructive and Maxillofacial Surgery, Sidi Mohamed Ben Abdellah University, Fez, Morocco

²Professor, Department of Reconstructive and Maxillofacial Surgery, Sidi Mohamed Ben Abdellah University, Fez, Morocco

DOI: <https://doi.org/10.36347/sjmcr.2026.v14i07.005>

| Received: 08.04.2026 | Accepted: 26.05.2026 | Published: 06.07.2026

*Corresponding author: Snikah Jihane

Resident, Department of Reconstructive and Maxillofacial Surgery, Sidi Mohamed Ben Abdellah University, Fez, Morocco

Abstract

Case Report

Background: Bone metastases are most commonly derived from lung, prostate, kidney, thyroid, or breast cancers. Colorectal origin is rare. Few studies have reported such metastases, with the axial skeleton being the most frequent site. Craniofacial involvement is exceptional. **Case presentation:** We report the case of a 73-year-old man in whom a right maxillary swelling and digestive symptoms were simultaneously discovered, revealing a metastatic rectosigmoid adenocarcinoma. Imaging, biopsy, and molecular analysis confirmed the diagnosis. The patient died three months after initiation of treatment, highlighting the poor prognosis associated with this rare presentation.

Keywords: Maxillary metastasis; colorectal adenocarcinoma; colorectal cancer; bone metastases; FOLFOX; KRAS.

Copyright © 2026 The Author(s): This is an open-access article distributed under the terms of the Creative Commons Attribution 4.0 International License (CC BY-NC 4.0) which permits unrestricted use, distribution, and reproduction in any medium for non-commercial use provided the original author and source are credited.

INTRODUCTION

Bone metastases represent a common site of spread in the natural history of many solid tumors, particularly those of the prostate, breast, lung, and kidney [1]. In contrast, their occurrence in colorectal cancers remains relatively uncommon, with an estimated incidence of 3 to 10% depending on the series [2,3].

These metastases most often arise at an advanced stage of disease and typically occur in the context of widespread multivisceral dissemination [1,4]. The topographic distribution shows a clear predominance of axial skeletal involvement, particularly the spine and pelvis, consistent with hematogenous spread via venous drainage pathways [5,6].

Craniofacial localizations are exceptional. Among these, maxillary involvement is an even rarer entity, most often described in the form of isolated case reports [7].

We report a case of synchronous maxillary metastasis from a rectosigmoid adenocarcinoma and discuss the epidemiological, clinical, and prognostic aspects of this unusual localization in light of the existing literature.

CASE REPORT

A 73-year-old man with a known history of type 2 diabetes managed with insulin therapy presented with a progressive right maxillary swelling associated with masticatory discomfort and adjacent tooth mobility. Symptoms had been evolving for approximately three months prior to admission.

Concomitantly, the patient reported digestive symptoms including constipation, melena, fatigue, and a weight loss of approximately 2 kg over the same period. Both the orofacial and digestive symptomatology evolved simultaneously over three months, with the maxillary swelling being the initial reason for consultation. The patient's general condition was preserved, with an ECOG performance status of 0.

Maxillofacial examination revealed a paramedian mass of the right upper maxilla, firm and tender on palpation, causing displacement and mobility of adjacent teeth. The overlying mucosa appeared inflamed without ulceration. No cervical lymphadenopathy was detected.

Abdominal examination revealed moderate tenderness in the left iliac fossa without a palpable mass

or hepatomegaly. Digital rectal examination identified a tumoral process located approximately 8 cm from the anal margin, without obstructive features.

Colonoscopy demonstrated a circumferential ulcero-vegetating tumoral process of the rectosigmoid junction causing luminal narrowing without complete obstruction. Biopsies confirmed a well-differentiated invasive adenocarcinoma of NOS type, arising on a transformed tubulo-villous dysplastic adenoma.

Thoraco-abdomino-pelvic CT scan demonstrated tumoral wall thickening of the rectosigmoid, along with four non-specific pulmonary micronodules, with no hepatic metastases identified. Craniofacial CT showed an osteolytic mass centered on the right upper maxilla, measuring approximately 30 × 52 mm, causing focal destruction of the dental arch and extending posteriorly toward the hard palate and anteriorly into the soft tissues of the right hemiface (Figure 2).

Pelvic MRI revealed circumferential tumoral thickening of the rectosigmoid extending over approximately 50 mm, with infiltration of the mesorectal fascia and no evidence of adjacent organ invasion or suspicious lymphadenopathy, corresponding to a cT3N0 staging.

Whole-body ¹⁸F-FDG PET-CT demonstrated a hypermetabolic right maxillary mass alongside pathological hypermetabolic rectosigmoid wall thickening, with no other foci of abnormal uptake elsewhere in the body (Figure 3).

Biopsy of the maxillary mass revealed a well-differentiated infiltrating glandular proliferation morphologically identical to the simultaneously diagnosed rectosigmoid adenocarcinoma. Immunohistochemical analysis was not performed. The diagnosis of maxillary metastasis was established on the basis of histomorphological concordance between the two lesions, supported by imaging findings and the absence of any other primary tumor on the staging workup.

Molecular analysis by next-generation sequencing (NGS) identified a KRAS gene mutation.

The case was discussed at a multidisciplinary tumor board. Based on the totality of clinical, radiological, histological, and molecular data, the diagnosis of metastatic rectosigmoid adenocarcinoma staged cT3N0M1 (stage IV), presenting with a synchronous maxillary metastasis, was established. First-line systemic chemotherapy with FOLFOX (5-fluorouracil, folinic acid, and oxaliplatin) was initiated in accordance with international guidelines. Palliative radiotherapy targeting the maxillary lesion was also recommended to address local symptoms.

The clinical course was unfavorable. The patient died three months after initiation of treatment in the context of rapid general deterioration, confirming the poor prognosis associated with osseous metastases from advanced colorectal cancer.

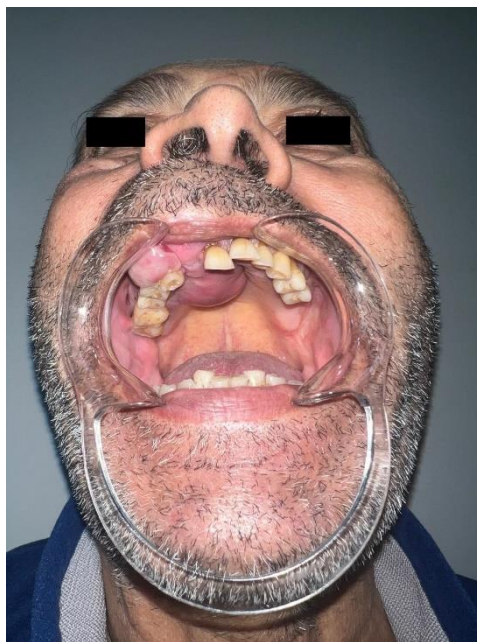


Figure 1 :paramedian maxillary tumor

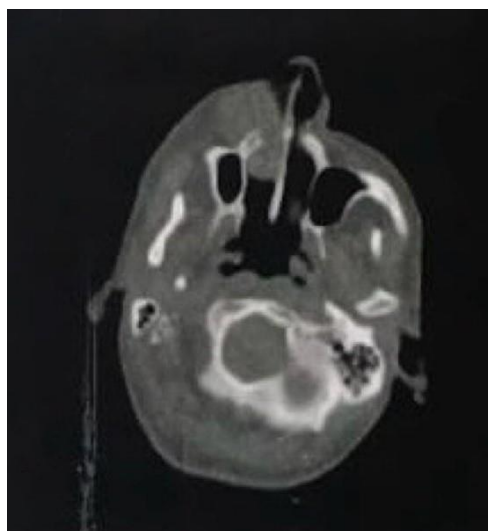


Figure 2: Axial CT scan showing an osteolytic mass of the right maxilla

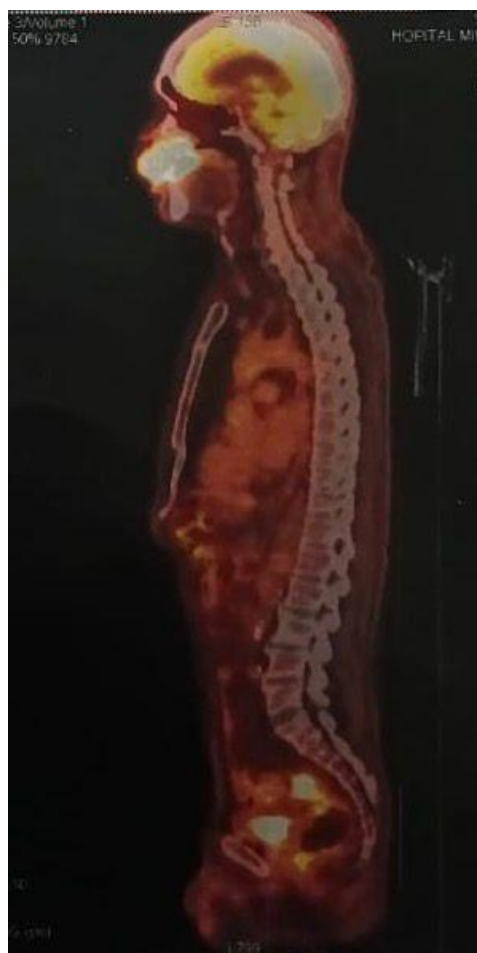


Figure 3 : PET-CT scan showing hypermetabolic right maxillary mass and rectosigmoid thickening

DISCUSSION

Bone metastases from colorectal cancer are rare compared to those arising from breast, prostate, or lung primaries [1]. Their incidence ranges from 3 to 10% across published series [2]. They typically occur at an advanced stage of disease and are frequently associated with visceral metastases, particularly hepatic and pulmonary [3].

Skeletal involvement in colorectal cancer predominantly affects the axial skeleton, including the spine, pelvis, and ribs [8]. Craniofacial localizations are exceptional, and maxillary metastases are rarely reported in the literature [9]. This rarity may be explained by the low proportion of hematopoietic marrow in the adult maxilla [10].

Tumor dissemination to osseous structures is thought to occur primarily via the hematogenous route through Batson's vertebral venous plexus, a valveless venous system connecting the pelvic veins to the vertebral and cranial venous networks, enabling tumor spread without obligatory pulmonary passage [5]. This mechanism may account for the absence of hepatic or pulmonary metastases observed in our patient.

In our case, the maxillary metastasis and the primary tumor were identified synchronously, with the orofacial symptomatology prompting the initial presentation. Although bone metastases from colorectal cancer are most commonly metachronous and arise in the setting of advanced known disease, synchronous forms have been described, indicating early dissemination at the time of diagnosis [19]. Clinically, these lesions most often manifest as painful swelling, tooth mobility, facial pain, or masticatory dysfunction [11]. In our patient, the presentation was dominated by maxillary swelling associated with adjacent tooth mobility.

Imaging plays a central role in establishing the diagnosis and assessing the extent of disease. CT typically reveals an aggressive osteolytic lesion with cortical bone destruction [12]. Pelvic MRI is the reference standard for locoregional staging of rectal cancer [13]; in our case, it demonstrated a cT3N0 tumor with mesorectal fascia infiltration.

¹⁸F-FDG PET-CT represents a highly valuable tool in the staging of metastatic colorectal cancer, offering whole-body assessment with high sensitivity for osteolytic bone lesions in particular [14]. In our case, it confirmed the hypermetabolic nature of both the maxillary lesion and the rectosigmoid tumor, with no additional sites of abnormal uptake.

Definitive diagnosis relies on histopathological analysis. In our observation, the diagnosis was established without immunohistochemistry, based on a convergent body of evidence: histomorphological concordance between the maxillary biopsy and the colorectal primary, an aggressive osteolytic CT pattern, concordant FDG-PET hypermetabolism, and the absence of any other primary tumor on whole-body staging. Several authors have emphasized that in the context of a known colorectal cancer, histological concordance alone is sufficient to confirm the diagnosis of bone metastasis, with immunohistochemistry reserved for cases of diagnostic uncertainty or unknown primary [15].

From a molecular standpoint, the identification of a KRAS mutation precludes the use of anti-EGFR targeted therapies, including cetuximab and panitumumab, whose efficacy is restricted to RAS wild-type tumors [20]. This finding therefore guided the therapeutic decision toward FOLFOX chemotherapy, in

accordance with ESMO guidelines for KRAS-mutant metastatic colorectal cancer [17].

The management of bone metastases from colorectal cancer relies primarily on systemic treatment combined with local symptomatic therapy [16]. Radiotherapy plays an important role in the palliative management of painful bone metastases [18]. Our patient received FOLFOX chemotherapy in combination with palliative radiotherapy directed at the maxillary lesion.

The prognosis of bone metastases from colorectal cancer remains globally poor and generally reflects advanced systemic disease [2,16]. The rapidly fatal course observed in our case, with death occurring three months after treatment initiation, underscores the dismal prognosis of these metastatic presentations and highlights the importance of early palliative care and multidisciplinary management.

This case also illustrates that in resource-limited settings, rigorous clinical and histological reasoning can establish the diagnosis of maxillary metastasis without necessarily resorting to costly immunohistochemical techniques, provided that a sufficiently robust convergent body of evidence is available.

CONCLUSION

Maxillary metastases from colorectal cancer represent a rare entity that may present synchronously with the primary tumor. Their diagnosis rests on a convergent set of clinical, radiological, and histological arguments, with immunohistochemistry not always required in the context of a known primary malignancy. The identification of a KRAS mutation directs the therapeutic strategy toward FOLFOX chemotherapy, precluding the use of anti-EGFR agents. The rapidly fatal course observed in our case highlights the poor prognosis of these metastatic forms and underscores the importance of early multidisciplinary management focused on patient quality of life.

Conflicts of Interest

The authors declare no conflicts of interest.

Author Contributions

All authors have read and approved the final version of the manuscript.

REFERENCES

1. Besbeas S, Stearns MW Jr. Osseous metastases from carcinomas of the colon and rectum. *Dis Colon Rectum*. 1978 ;21(4) :266-8.
2. [2] Kanthan R, Loewy J, Kanthan SC. Skeletal metastases in colorectal carcinomas: a Saskatchewan profile. *Dis Colon Rectum*. 1999 ;42(12) :1592-7.

3. Nozue M, Oshiro Y, Kurata M, *et al.*, Treatment and prognosis in colorectal cancer patients with bone metastasis. *Oncol Rep*. 2002 ;9(1) :109-12.
4. Talbot RW, Irvine B, Jass JR, *et al.*, Bone metastasis in carcinoma of the rectum : a clinical and pathological review. *Eur J Surg Oncol*. 1989 ;15 :449-52.
5. Batson OV. The function of the vertebral veins and their role in the spread of metastases. *Ann Surg*. 1940 ;112(1) :138-49.
6. Libson E, Bloom RA, Husband JE, Stoker DJ. Metastatic tumours of bones of the hand and foot. *Skeletal Radiol*. 1987 ;16(5) :387-92.
7. Cama E, Agostino S, Ricci R, Scarano E. A Rare Case of Metastases to the Maxillary Sinus from Sigmoid Colon Adenocarcinoma. *ORL J Otorhinolaryngol Relat Spec*. 2002 ;64(5) :364-7.
8. Santini D, Tampellini M, Vincenzi B, *et al.*, Natural history of bone metastasis in colorectal cancer. *Ann Oncol*. 2012 ;23(8) :2072-2077.
9. Hirshberg A, Leibovich P, Buchner A. Metastatic tumors to the jawbones: analysis of 390 cases. *J Oral Pathol Med*. 1994 ;23(8) :337-341.
10. Zachariades N. Neoplasms metastatic to the mouth, jaws and surrounding tissues. *J Craniomaxillofac Surg*. 1989 ;17(6) :283-290.
11. Van der Waal RIF, Buter J, van der Waal I. Oral metastases: report of 24 cases. *Br J Oral Maxillofac Surg*. 2003 ;41(1) :3-6.
12. Galasko CSB. The anatomy and pathways of skeletal metastases. *Bone Metastasis*. 1986 :49-63.
13. Brown G, Richards CJ, Bourne MW, *et al.*, Morphologic predictors of lymph node status in rectal cancer with use of high-spatial-resolution MR imaging. *Radiology*. 2003 ;227(2) :371-377.
14. Even-Sapir E. Imaging of malignant bone involvement by morphologic, scintigraphic and hybrid modalities. *J Nucl Med*. 2005 ;46(8) :1356-1367.
15. Bayrak R, Yenidunya S, Haltas H. The value of CDX2 and cytokeratins 7 and 20 expression in differentiating colorectal adenocarcinomas. *World J Gastroenterol*. 2012 ;18(47) :6957-6963.
16. Benson AB, Venook AP, Al-Hawary MM, *et al.*, NCCN Clinical Practice Guidelines : Colon Cancer. Version 2.2023. National Comprehensive Cancer Network ; 2023.
17. Van Cutsem E, Cervantes A, Adam R, *et al.*, ESMO consensus guidelines for the management of patients with metastatic colorectal cancer. *Ann Oncol*. 2016 ;27(8) :1386-1422.
18. Lutz S, Balboni T, Jones J, *et al.*, Palliative radiation therapy for bone metastases. *Pract Radiat Oncol*. 2017 ;7(1) :4-12.
19. Riihimäki M, Hemminki A, Sundquist J, Hemminki K. Patterns of metastasis in colon and rectal cancer. *Sci Rep*. 2016 ;6 :29765.
20. Douillard JY, Oliner KS, Siena S, *et al.*, Panitumumab-FOLFOX4 treatment and RAS mutations in colorectal cancer. *N Engl J Med*. 2013 ;369(11) :1023-1034.