

CLINICAL IMAGE

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Brain relapse of a mucosal melanoma

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CASE REPORT

We report a case of a 52-year-old patient, with a history of a surgically cured locally advanced anorectal melanoma with pelvic lymphadenopathies. The diagnosis was based on histology: On a macroscopical level, it was a tumor located in the anorectal margin, exophytic, blackish, and friable. Microscopy showed an ulcerated mucosa, a chorion that is massively infiltrated by a high-density proliferation made of atypical round, oval, or elongated cells with atypical hyperchromatic nuclei, often nucleolated, with the presence, in areas, of melanic pigments. Immunohistochemistry showed a diffuse cytoplasmic signal on anti-HMB45 immunomarking.

No metastases were found on the initial workup, which included a computed tomography (CT) scan of the chest-abdomen-pelvis, a brain magnetic resonance imaging (MRI), and a positron emission tomography scan. And the treatment consisted of an abdomino-perineal amputation with a pseudo-continent perineal colostomy, followed by pembrolizumab immunotherapy.

The patient had been under control for more than a year, before presenting to the emergency room of our structure, for an acute setting of a right facial palsy and a blurred vision.

A brain CT was performed, showing multiple supra and infratentorial nodular lesions, homogeneously enhanced after contrast (Figures 1 and 2).

Brain MRI showed multiple nodular lesions in the frontal, parietal, occipital, and temporal lobes, in addition to the cerebellar hemispheres, not exceeding 2 cm in size, hyperintense on T1, hypointense on T2 and T2*,

with nodular and ring-like enhancement after contrast (Figures 3–5), suggesting a melanomatous origin, in favor of a cerebral relapse of the anorectal melanoma.

We suggested stereotactic radiosurgery alone as treatment, since all the lesions were smaller than 2 cm. The patient was unfortunately lost to follow-up afterward.



Figure 1: An axial pre-contrast CT image showing a spontaneously hyperdense nodular lesion in the right caudate nucleus (arrow).



Figure 2: An axial post-contrast CT image showing a homogeneously enhanced nodular lesion in the right caudate nucleus (arrow).

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Received: 25 May 2022

Accepted: 17 September 2022

Published: 18 October 2022

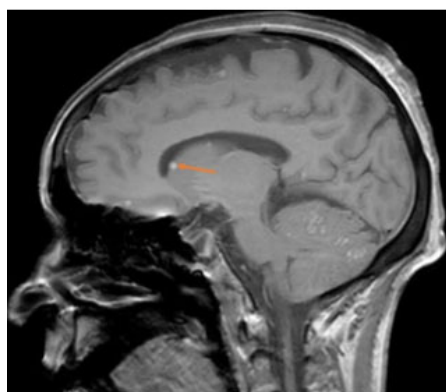


Figure 3: A sagittal T1 pre-contrast MRI image showing a hyperintense nodular lesion in the right caudate nucleus (arrow).

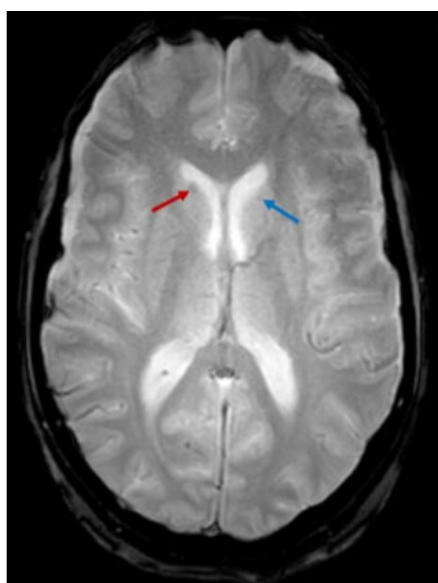


Figure 4: An axial T2* MRI image showing two hypointense nodular lesions in the right (red arrow) and left (blue arrow) caudate nuclei.

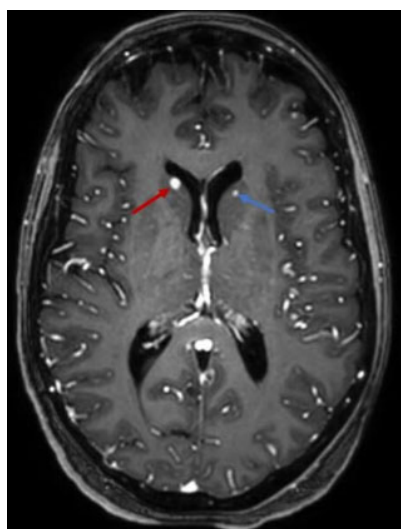


Figure 5: An axial T1 post-contrast MRI image showing a ring-like enhancing lesion in the right caudate nucleus (red arrow) and a homogeneously enhanced nodular lesion in the left caudate nucleus (blue arrow).

DISCUSSION

Extracutaneous malignant melanomas are rare tumors with a poor prognosis. Only 4–5% of all primary melanomas are noncutaneous [1].

Anorectal melanomas are rare entities accounting for 0.22% of all melanomas, 4.5% of noncutaneous melanomas, and 16.5% of mucosal melanomas [2]. It comprises 0.5–5% of all anorectal malignancies [3].

Melanomas are the second or third most common cause of cerebral metastases after lung and breast cancer, with a risk corresponding to 6.9% [4–6]. Cerebral metastases are the most ominous feature of malignant melanoma [7].

The mean time interval between the initial diagnosis of melanoma and appearance of intra-cranial metastases is estimated at 2.5 years, and the mean time interval between the initial diagnosis and development of the first symptoms of central nervous system (CNS) metastases is 3.5 years [8]. It corresponds to one year time interval in our case.

Symptoms and signs of cerebral metastases depend on the location of the tumor and its extent. There is no symptom that is specific for an intracranial metastasis. Any recently appearing neurological symptom must suggest the diagnosis. Seizures, however, are highly suspicious since melanoma is one of the primary tumors that most commonly cause seizures. Headache and mental confusion, in the other hand, are the most common symptoms of brain metastases, in addition to motor dysfunction and nerve disturbance [9].

Radiological characteristics of melanomatous metastases are distinctive due to the presence of melanin and bleeding tendencies [10].

Computed tomography scan with and without contrast can detect most metastases above 10 mm and hemorrhagic lesions [11]. Metastatic lesions found on melanoma patients can be multiple or solitary. They are frequently located in the cerebral hemispheres, usually in the frontal lobes.

They often appear spontaneously hyperdense (75%), hypodense (22%), or isodense (3%), with post-contrast homogeneous nodular enhancement or ring-like pattern [8].

Magnetic resonance imaging is far more sensitive, especially for smaller lesions, lesions in the posterior cerebral fossa, and for leptomeningeal involvement [11]:

On T1-weighted imaging (WI): Half of melanomatous metastases is hyperintense, which is distinctive, since other cerebral metastases are rarely hyperintense on T1. This hypersignal is due to the intrinsic presence of blood and melanin.

On T2 WI: They are typically hypointense.

On T2* WI: In 71% of patients, these lesions present whether a hyposignal on T2*, a hypersignal on T1, or both. Therefore, the association of T1 and T2* sequences can help suggest a melanomatous origin of intra-cranial lesions.

Post-contrast T1 WI: It is the Gold standard for the diagnosis of cerebral metastases. It demonstrates nodular or ring-like enhancement [8, 10].

Differential diagnoses include hemorrhagic metastases of non-melanomatous origin, such as choriocarcinoma, renal cell carcinoma, thyroid, lung and breast cancer. Note that lung and breast cancer have a higher overall prevalence; Hemorrhagic metastases are usually iso- or hypointense on T1, but may also demonstrate T1 signal hyperintensity, depending on the age of hemorrhage.

Primary high-grade glioma is a differential diagnosis to solitary metastases. No individual parameter can accurately distinguish the two, however, evaluation of peritumoral edema, if there is one, by spectroscopy, perfusion, and diffusion-weighted imaging (DWI), may help lead to the accurate diagnosis (e.g., an ADC in the peritumoral edema that is higher than $1.3 \times 10^{-3} \text{mm}^2/\text{s}$ is more suggestive of a metastasis) [12].

In our case, we had multiple brain lesions with a history of melanoma, which made the diagnosis easier. No peritumoral edema nor abnormality on DWI was noted.

When it comes to treatment, recent data have shown improved intracranial response with the newer molecules of systemic therapies such as BRAF/mitogen-activated protein (MAP) kinase inhibitors and immunotherapy (anti-programed cell death protein 1, anti-cytotoxic T lymphocyte-associated protein 4).

The suggestion of local therapy (surgery and stereotactic radiosurgery) would depend on patient performance, neurological symptoms, location, size, and number of brain metastasis. The advantages of surgery include rapid relief of pressure effect, evacuation of hemorrhage, and the possibilities of biopsies for diagnosis and molecular studies to select appropriate systemic therapy. Combining surgery for larger and symptomatic lesions with stereotactic radiosurgery for smaller lesions is a common practice in patients with multiple brain metastases. Following resection, radiotherapy to resection cavity is recommended to eliminate micro-metastases or residual metastases [13].

CONCLUSION

Malignant melanoma is an aggressive cancer and is a frequent cause of cerebral metastases. The latter demonstrates distinctive imaging features on brain MRI (hyperintense on T1 and hypointense on T2*). A relapse, especially intracranial, is possible even after years of control. The development of any recent neurological sign should suggest the diagnosis in patients with a history of melanoma. Systemic immunotherapy, surgery, stereotactic radiosurgery, and post-operative radiotherapy are all tools in the therapeutic arsenal to control intra-cranial metastases.

Keywords: Brain, Melanoma, Metastases, MRI

How to cite this article

Naggar A, Mohamed DA, Omor Y, Latib R. Brain relapse of a mucosal melanoma. Int J Case Rep Images 2022;13(2):160–163.

Article ID: 101351Z01AN2022

doi: 10.5348/101351Z01AN2022CI

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Author Contributions

Amine Naggar – Conception of the work, Design of the work, Acquisition of data, Analysis of data, Interpretation of data, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Daoud Ali Mohamed – Conception of the work, Design of the work, Acquisition of data, Interpretation of data, Drafting the work, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Youssef Omor – Conception of the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Rachida Latib – Conception of the work, Revising the work critically for important intellectual content, Final

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Guarantor of Submission

The corresponding author is the guarantor of submission.

Source of Support

None.

Consent Statement

Written informed consent was obtained from the patient for publication of this article.

Conflict of Interest

Authors declare no conflict of interest.

Data Availability

All relevant data are within the paper and its Supporting Information files.

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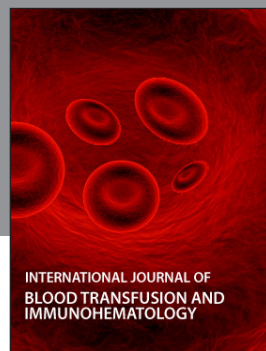
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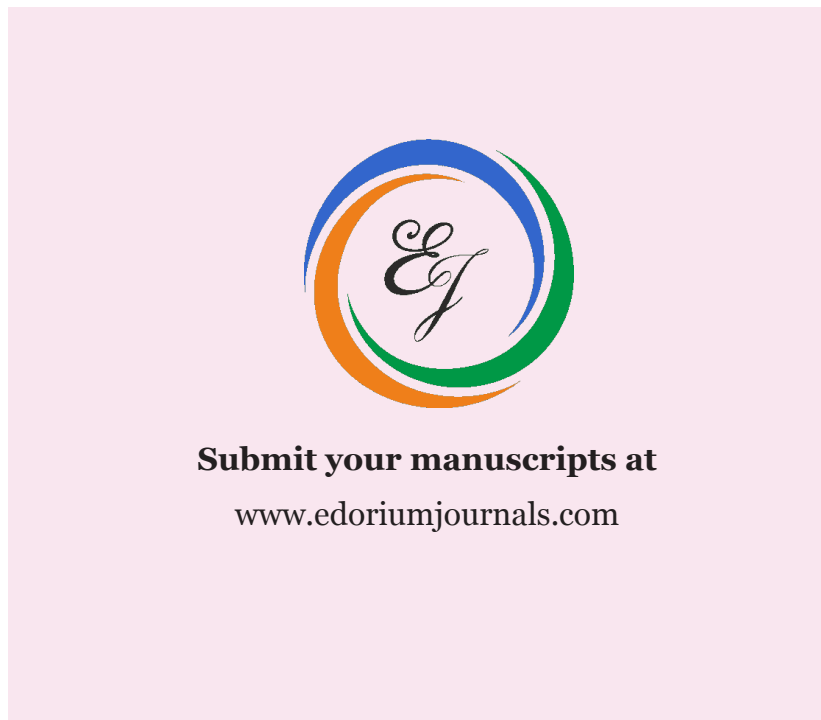
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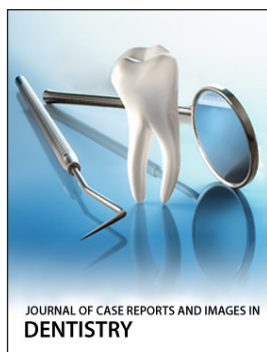
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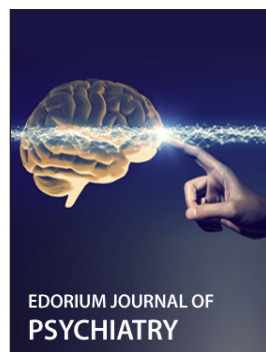
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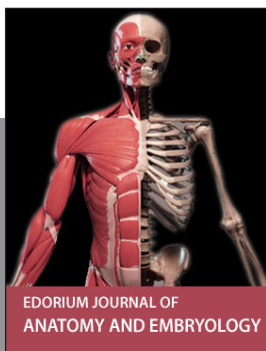
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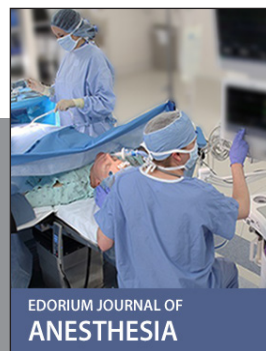
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