Open Journal of Medical Images and Case Reports
ISSN: 3067-1078 | Volume 3, Issue 1, 2026
www.aytinpublications.com

Case Report

Radiation Induced Cavernous Hemangioma: Case Report with a Focus on Pathological Features and Literature Review

Dr. Abdul Rahman Al Subhi[1], Dr. Simin Laiq[1]*, Dr. Zahra Al Hajri[1], Dr. Ahmed Al Habsi[2], Dr. Ahmed Al Azri[3], Med. Phys. Fatema Al Hashmi[4], Dr. Namrata Satyapal[4]

[1]Department of Histopathology, Directorate general of Khoula Hospital, Muscat, Oman
[2]Department of Radiology, Directorate general of Khoula Hospital, Muscat, Oman
[3]Department of Neurosurgery, Directorate general of Khoula Hospital, Muscat, Oman
[4]Department of Oncology. Royal Hospital, Muscat, Oman

***Corresponding Author:** Dr Simin Laiq, Senior Specialist Pathologist, Department of Histopathology, Directorate General of Khoula Hospital, Muscat, Oman.

Abstract

Radiation-induced cavernous hemangioma (RICH) is a possible complication of brain irradiation. These lesions are rare; only a few radiation-induced cavernous hemangioma cases have been published to date. We report a case of a 41-year-old female who was diagnosed with metastatic breast cancer in 2012 and subsequently underwent metastatic brain tumor excisions followed by multiple brain irradiation sessions and stereotactic radiosurgery. In 2023, she presented with symptoms of worsening memory, left-sided weakness, and cerebellar signs. Brain imaging revealed lesions at the two sites of previous surgeries and radiation, so she underwent new brain lesion excisions to control the symptoms. Pathological inspection of the lesions showed multiple radiation-induced cavernous hemangiomas with no residual or recurrent tumor. Herein we report the details of this case and discuss the typical clinical and histological features of radiation-induced cavernous hemangiomas.

Keywords: Radiation-induced cavernous hemangioma, RICH, radiation, stereotactic radiosurgery, metastatic tumor, breast cancer.

Article Information

Recieved: 30 December 2025

Accepted: 14 January 2026

Published: 16 January 2026

Cite this article as:

Dr. Abdul Rahman Al Subhi, Dr. Simin Laiq, Dr. Zahra Al Hajri, *et al.* Radiation Induced Cavernous Hemangioma: Case Report with a Focus on Pathological Features and Literature Review. Open Journal of Medical Images and Case Reports. 2026;3(1): 01-05.

https://doi.org/10.71123/3067-1078.030101

Introduction

Radiation therapy is a very effective mode of cancer treatment; however, it is associated with myriad side effects and complications. Cavernous hemangiomas (CHs) are considered a rare complication of cranial radiation therapy. While there is an increasing move towards studying radiation therapy complications, radiation-induced cavernous hemangiomas (RICHs) remain understudied. It was not until 1994 that RICH was recognized as a complication of radiation therapy (1). The scarcity of this topic in the literature can be attributed to the low rates of RICH incidence. Evidently, a retrospective study conducted on 40 subjects by Heckel et al (2), found that 1 in 4 patients under the age of 15 who were diagnosed with cerebral cavernomas received prior radiation therapy. In adults, the evidence is scarce with regards to the exact incidence rates, and most reported cases are in the pediatric population. Another retrospective study, conducted by Koester et al, found that intracranial radiation-induced malformations made up 10 out of a total of 1662 cavernous malformation cases in a single center (3). Seldom has there been a proper assessment of the pathological characteristics of RICHs. This report will deconstruct a case of cranial RICH and compare features and findings to the evidence available in the literature.

Case Report

A 41-year-old female patient presented in 2023 with a headache, worsening memory, severe weakness in the left side causing recurrent falls, and cerebellar signs (dysdiadochokinesia, slurred speech, intention tremor, and nystagmus). In the past, she was diagnosed with triple-positive de-novo metastatic left breast cancer in 2012. She then received 12 cycles of TCH (Taxol, carboplatin and Herceptin (Trastuzumab)); and was kept on maintenance Trastuzumab, Goserelin & Tamoxifen.

In 2016, four years after the initial breast cancer diagnosis, she presented to the hospital with worsening cognitive function and motor deficit and was found to have brain metastasis on imaging with lesions in the right temporal lobe and cerebellum.

Subsequently, she underwent complete excision of the cerebellar mass followed by WBRT (whole brain radiation therapy) (30Gy/10 Fr + 9Gy/3 Fr). She remained well for two years, then motor and cognitive symptoms recurred, and imaging showed brain metastasis recurrence. She received further treatment with SRS 12Gy. Her symptoms were controlled for one year, and then she relapsed again with progression of the brain lesion. This time she received 2Gy/12 Fr for the left-brain lesion and 25Gy/5 Fr for the right-brain lesion with adjuvant chemotherapy. The patient continued to deteriorate with spine involvement, requiring spinal radiation therapy (20Gy/5Fr).

The patient was kept on chemotherapy, with regular imaging follow-ups, until her presentation in 2023. The patient was in a wheelchair unable to bear weight, and a poor historian due to slurred speech. She also exhibited ataxia, dysdiadochokinesia, nystagmus, and intention tremor. Neurological examination demonstrated generalized lower limb weakness (left side 3/5 and right side 4/5). Head CT and brain MRI were done, the latter showed an increase in the cystic component of the right temporal lesion, its solid component and the cerebellar lesion remained stable (Figure 1).

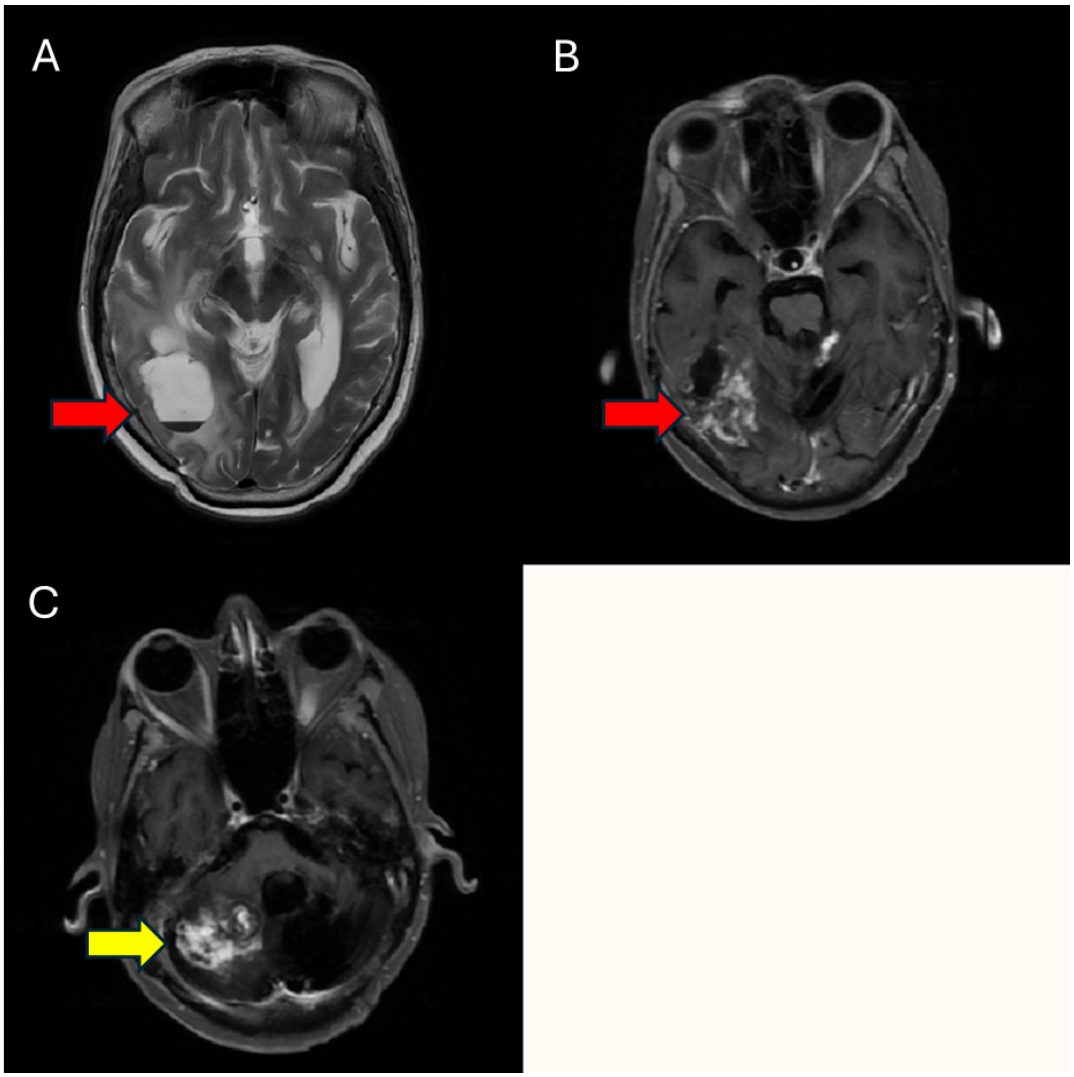

Figure 1. *Imaging findings on MRI, axial scans. (A) Shows the temporal lobe cystic lesion on contrast-enhanced MRI (red arrow). (B) Shows the same cystic lesion on flair-weighted MRI (red arrow). (C) Shows the stable cerebellar lesion on flair-weighted MRI (yellow arrow).*

Consequently, to control the disease and limit imminent deterioration, the patient underwent excision of right temporal and cerebellar lesions. Greyish firm, moderately vascular masses were seen during surgery and excised.

Histopathology reported brain parenchyma with large areas of fibrin material disposition, associated with multiple thin-walled dilated blood vessels. The blood vessels showed varying radiation-induced changes, including radiation-induced atypia in the endothelial cells, telangiectasias, vascular hyalinization and fibrinoid vascular necrosis. The rest of the brain parenchyma showed radiation-induced changes, and there was no mitotic activity or tumor identified. Special stains used supported a diagnosis of a radiation-induced cavernous hemangioma (RICH) (Figure 2).

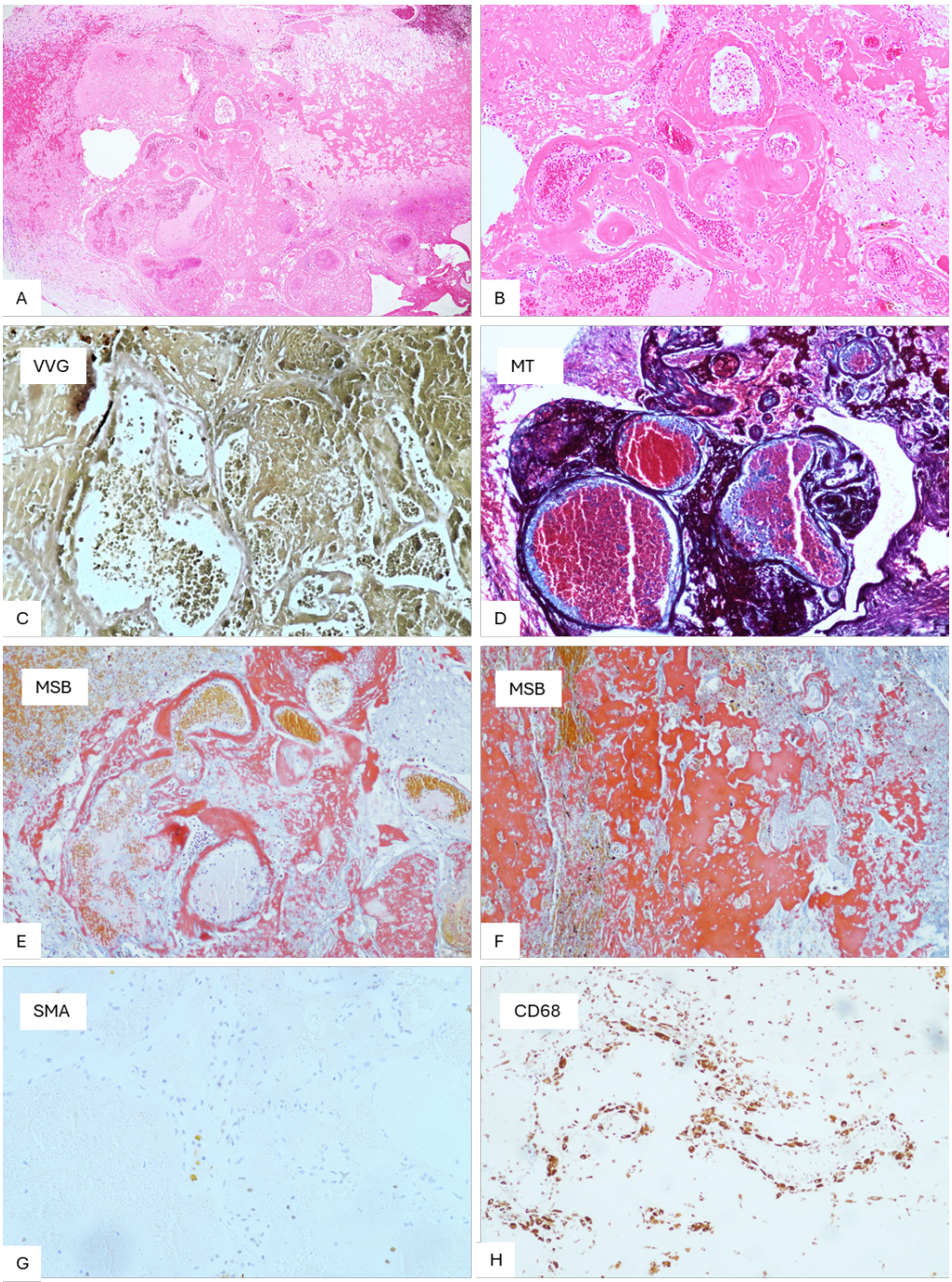

Figure 2. *Radiation-induced cavernous hemangioma (RICH). (A, B) H&E show proliferation of irregular, partly compressed capillary-sized vascular channels (Hematoxylin and Eosin, x4 and x10 magnification). (C) VVG highlights lack of elastic lamina in the walls of the vessels (Verhoff-Van Gieson, x20 magnification). (D) Mason trichrome highlights lack of prominent hyalinization (MT, x10 magnification). (E) MSB stain highlights the fibrinoid necrosis (red) in the wall of the vascular channels (Martius Scarlet Blue, x10 magnification). (F) MBS highlights the fibrin deposits in the background (Martius Scarlet Blue, x10 magnification). (G) Alpha-SMA shows the lack of smooth muscle component in the vessel wall (Smooth Muscle Actin immunostain, x10 magnification). (H) CD68 highlights infiltration of foamy macrophages into the vessel walls (CD68 macrophage immunostain, x10 magnification).*

With regards to the clinical outcome post-operation; upon follow-up, her comprehension and speech normalized, the left-sided weakness and cerebellar signs improved, she is now able to mobilize using a walking stick, and her headaches resolved.

Discussion

The etiology of radiation-induced cavernous hemangiomas (RICHs) is still not fully understood. However, it has been proposed that radiation stimulates endothelial proliferation causing fibrinoid necrosis formation and hyalinization of the vascular wall, consequently leading to narrowing of the vascular lumen and ischemia. This activates HIF-1 which induces VEGF leading to reactive angiogenesis (4). Notably, the expression of both HIF-1 and VEGF decreases with age, which can partially explain why RICHs occur more in younger patients (4). Regarding molecular pathogenesis, it has been hypothesized that a two-hit gene mutation mechanism is the basis of RICH formation (5). P53 and CCM1/KRIT-1 genes are involved, one suggested mechanism is the preexistence of a mutation in the p53 gene (common in CNS neoplasms) in the periluminal endothelial cells before irradiation. Coincidently, irradiation causes a mutation in the CCM1/KRIT-1 gene forming cavernous hemangioma. Another explanation would be a preexisting genetic mutation in CCM1/KRIT1, and subsequent radiation causes a mutation in the p53 gene leading to the formation of cavernous hemangioma. The possibility of both events being induced by irradiation alone is unknown (5,6).

Radiation amount is a determinant factor in RICHs formation, there is an inverse relationship between radiation dose and RICHs latency (7). An average of 4.75-year latency from radiation exposure (>30Gy) has been reported in the literature (8). Other studies reported a range from 2 to 21 years latency to diagnosis from irradiation in meningioma cases, depending on the radiation dose (9). In our patient's case, she received four courses of radiation therapy with an 8-year latency, which is double the average latency time reported. There is always the possibility that the patient had developed RICH earlier and exhibited the symptoms later. With regards to dose-effect correlation, she did not receive any single course exceeding 30Gy, however, considering the accumulative effect, she exceeded the 30Gy cut-off. Cases reporting patients with multiple RICHs are scant. Studies have yet to show any relationship between the development of multiple RICHs and radiation dose, age at irradiation, or latency to diagnosis (6). Radiation-induced cavernous hemangiomas in the temporal and cerebellum have been found on pathological examination in our patient. Given the current evidence, whether there is a correlation between the amount of radiation and the multiplicity of hemangiomas remains unknown, however, with further advances and research this could change.

Our patient presented with confusion, memory loss, headache, incoordination, worsening weakness, and recurrent falls. These symptoms improved post resection, however, persistent weakness remained, which can be explained by either brain dysfunction after undergoing multiple cranial surgeries, or due to muscular atrophy post prolonged immobility. A literature review was conducted by Keezer and Del Maestro in 2009. Found that 48% of 85 patients with brain RICHs, presented with the following symptoms: seizure, headache, emesis, and motor dysfunction (6). These symptoms are similar to what our patient presented with. Cavernous hemangiomas (CHs) in general are associated with hemorrhage, which is considered a serious complication. The hemorrhage risk is estimated to be 0.25-0.7% per year in de novo CHs and it is even higher in RICHs (8).

Very limited studies have looked at the pathological aspect of the disease and fewer studies have examined the differences in histology between de novo CHs and RICHs. De novo CHs are comprised of proliferating ectatic variably thickened and hyalinized blood vessels, lacking elastic tissue or significant smooth muscle cell component. Whereas, RICHs show irregular, partly compressed capillary-sized vascular channels, with capillary proliferation-like area in the center of the lesion (10). This feature was seen in our case (Figure 2). To further delineate these differences special staining was used. Upon staining with mason trichrome (MT), de novo lesions showed prominent hyalinization, while RICHs did not (10). With alpha-SMA staining, RICHs showed no uptake, whereas the endothelial-lined de novo CHs lesions were positive. The difference in infiltration by foamy macrophages into the vessel walls can be contrasted by CD68 staining, where uptake was seen in RICHs walls only (10). In our case, similar findings have been found as described in RICHs by Cha et al (10). On MT staining, no hyalinization was seen within or around the walls of the RICHs (Figure 2D), and there was no uptake on alpha-SMA staining (Figure 2G). CD68 uptake was noted in the walls upon staining (Figure 2H).

Other stains that have been used in our case, like the VVG (Verhoff-Van Gieson) stain (Figure 2C) which was used to differentiate arterio-venous malformations from hemangiomas, this was highlighted by the absence of elastic lamina within the walls of the vessels in our case. Abundant eosinophilic material deposits were also seen in the background brain parenchyma upon examination under the microscope, so both Congo-red and Martius Scarlet Blue (MSB) stains were used to determine whether these

dispositions were amyloid or fibrin. Congo-red came back negative for amyloid and MSB highlighted the deposits as fibrin material (Figure 2F). MSB also highlighted the fibrinoid necrosis in the walls of the vascular channels (Figure 2E).

Our patient underwent surgical resection of the cerebellar and right temporal lesions, for symptomatic and disease control. The treatment of RICH is symptom-dependent, around 50% of cases are asymptomatic (incidental diagnoses), these cases are followed up regularly for hemorrhagic events or neurological manifestation (11). Whereas symptomatic patients and those with hemorrhagic events may require surgical resection (11).

In conclusion, RICHs represent a distinct clinico pathological entity arising through complex vascular injury and possible two-hit genetic mechanisms. It is characterized by delayed onset after cranial irradiation, unique histological features compared to de novo cavernous hemangiomas, and a potentially higher risk of hemorrhage than de novo lesions. Our case of multiple RICHs following cumulative sub-threshold radiation exposure highlights the need for awareness of their variable latency, uncertain dose-response relationship, and the role of histopathology in accurate diagnosis. Lifelong follow-up may be warranted for at-risk patients to enable timely detection and management.

Acknowledgements

The authors would like to thank the Departments of Histopathology, Radiology, Neurosurgery, and Oncology at Khoula Hospital and Royal Hospital, Muscat, Oman, for their multidisciplinary support in the diagnosis and management of this case.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

References

1. Ciricillo, S. F., Cogen, P. H., & Edwards, M. S. (1994). Pediatric cryptic vascular malformations: presentation, diagnosis and treatment. *Pediatric neurosurgery*, *20*(2), 137-147.
2. Heckl, S., Aschoff, A., & Kunze, S. (2002). Radiation-induced cavernous hemangiomas of the brain: A late effect predominantly in children. *Cancer*, *94*(12), 3285-3291.
3. Koester, S. W., Rhodenhiser, E. G., Dabrowski, S. J., Benner, D., Rumalla, K., Scherschinski, L., ... & Lawton, M. T. (2023). Radiation-induced cerebral cavernous malformations: a single-center experience and systematic literature review. *World neurosurgery*.
4. Cacho-Díaz, B., Salmerón-Moreno, K., Lorenzana-Mendoza, N., Reyes, A., Valdés-Ferrer, S. I., Gómez-Ahumada, G., ... & Herrera-Gómez, Á. (2018). Radiotherapy induced cavernomas in adult cancer patients. *Radiotherapy and Oncology*, *127*(2), 287-291.
5. Feiz-Erfan, I., Zabramski, J. M., Herrmann, L. L., & Coons, S. W. (2006). Cavernous malformation within a schwannoma: review of the literature and hypothesis of a common genetic etiology. *Acta neurochirurgica*, *148*, 647-652.
6. Keezer, M. R., & Del Maestro, R. (2009). Radiation-induced cavernous hemangiomas: case report and literature review. *Canadian journal of neurological sciences*, *36*(3), 303-310.
7. Oishi, T., Yamasaki, T., Baba, S., Koizumi, S., Sameshima, T., & Namba, H. (2020). Acute and chronic hemorrhage from radiation-induced cavernous malformation associated with late-delayed radiation necrosis in long surviving glioma patients: A case report. *Oncology Letters*, *19*(1), 513-518.
8. Jain, R., Robertson, P. L., Gandhi, D., Gujar, S. K., Muraszko, K. M., & Gebarski, S. (2005). Radiation-induced cavernomas of the brain. *American journal of neuroradiology*, *26*(5), 1158-1162.
9. Yu, Z., Huang, B., & Liang, R. (2020). Radiation-induced cavernous malformation after stereotactic radiosurgery for cavernous sinus meningioma: a case report. *BMC neurology*, *20*, 1-7.
10. Cha, Y. J., Nahm, J. H., Ko, J. E., Shin, H. J., Chang, J. H., Cho, N. H., & Kim, S. H. (2015). Pathological evaluation of radiation-induced vascular lesions of the brain: distinct from de novo cavernous hemangioma. *Yonsei medical journal*, *56*(6), 1714-1720.
11. Park H, Kim S, Rhim JH, Park JH, Choi IS, Kim JS, Kim KH. The development of numerous radiation-induced cavernous malformations in a germ cell tumor patient: A case report. Current Problems in Cancer. 2021 Apr 1;45(2):100654.