Interdisciplinary Neurosurgery: Advanced Techniques and Case Management 27 (2022) 101387

Contents lists available at ScienceDirect

Interdisciplinary Neurosurgery: Advanced Techniques and Case Management

journal homepage: www.elsevier.com/locate/inat

Staged surgical treatment for a giant hypervascular extra-intracranial metastasis of thyroid cancer using preoperative embolization and total microsurgical removal

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ARTICLE INFO

Keywords:
Papillary thyroid cancer skull metastases
Hypervascular skull metastases
Preoperative embolization

ABSTRACT

Herein, we present a clinical case of a successfully staged treatment for a patient with giant hypervascular extra-intracranial metastasis of locally advanced stage IVa T3bN1aM1 (skull) papillary thyroid cancer. To treat the patient, we used tactics that included preoperative endovascular embolization and total microsurgical metastasis removal with simultaneous cranioplasty using an individual stereolithographic titanium three-dimensional implant, followed by total thyroidectomy, radiotherapy, and pharmacotherapy (^{131}I radioiodine and suppressive therapies).

The patient was diagnosed with papillary cancer after total skull metastasis removal and obtaining path histological and immunohistochemical biopsy material analysis results.

Giant hypervascular mass removal is associated with a high risk of intraoperative massive bleeding, unpredictable course of surgical intervention, and consequently, high probability of developing adverse postoperative complications.

These cases are described in isolated reports, making this article relevant.

The study describes a case when correctly planned tactics allowed operating the patient totally, safely, and with minimal blood loss and suggests the examination tactics in cranial hypervascular tumor patients.

1. Introduction and literature review

Various studies estimated that 20% of all cancer patients have brain metastases [1–3]. Note that not all cancer treatment protocols include brain magnetic resonance imaging (MRI) as a routine screening; therefore, the true incidence is likely to be higher because such estimates are limited to patients for whom the treatment is considered [4]. Although almost any type of cancer can metastasize to the brain, the most common metastases occur in lung cancer (20–56%), breast cancer (5–20%), and melanoma (7–16%) [1,3–6].

Thyroid cancer (TC) is of particular significance and is the most common endocrine malignancy. TCs are divided into four major histological subtypes: papillary, follicular, medullary, and anaplastic. Papillary thyroid cancer (PTC) is the most common subtype, followed by follicular thyroid cancer (FTC). PTC and FTC are both well-differentiated thyroid cancer (DTC), accounting for 90% of all TCs [7–9]. Despite a relatively favorable prognosis in patients with DTC, 5–23% of patients have distant metastases, which worsens their prognosis despite adequate therapy [7,9,10]. Bone is the first most common site of distant FTC metastases and the second most frequent site of PTC metastases [10]. Bone FTC metastases most commonly occur in long bones such as the femur and flat bones such as the iliac bone and sternum, whereas PTC most commonly metastasizes to the ribs, vertebrae, and sternum. Both FTC and PTC metastases to the skull bones are extremely rare and account for only 2.5% of all bone metastases. The majority of cranial bone TC metastases are caused by FTC, followed by PTC [9,11–13]. Cranial DTC metastases can occur in patients aged 18–70 years [11,14]. Cranial PTC metastases occur in patients aged

Abbreviations: TC, thyroid cancer; MRI, magnetic resonance imaging; CT, computed tomography; CTA, computed tomography angiography; PTC, papillary thyroid cancer; FTC, follicular thyroid cancer; DTC, differentiated thyroid cancer.

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https://doi.org/10.1016/j.inat.2021.101387
Received 19 June 2021; Received in revised form 10 August 2021; Accepted 20 September 2021
Available online 24 September 2021

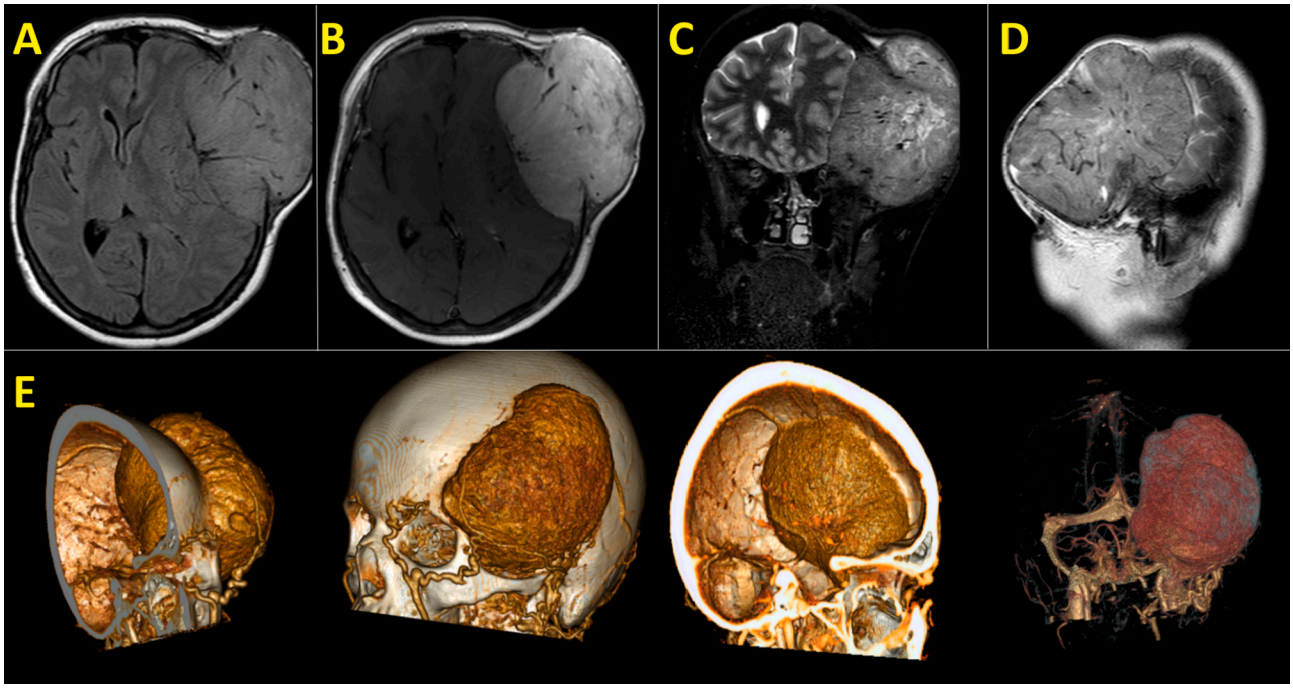

Fig. 1. A — Preoperative axial T2-weighted/FLAIR imaging. B — Preoperative axial T2-weighted imaging with gadolinium. C — Preoperative coronal short tau inversion recovery imaging. D — Preoperative sagittal T2-weighted/TSE imaging. E — Preoperative three-dimensional CTA reconstruction. A giant (105 × 102 × 75 mm) hypervascular extracerebral mass lesion with extra-intracranial growth, cerebral compression, and 10-mm right-sided axial shift.

35–73 years (mean, 53.5) [15]. The average period from thyroid carcinoma diagnosis to cranial bone metastasis is 23.3 years [11]. With regard to anatomic and topographic location, cranial vault TC metastases are most often diagnosed in frontal and parieto-occipital regions [14,16–24].

Usually, TC skull metastases are a slowly growing soft painless mass that can be palpated under the scalp. The tumor manifests itself by reaching a large size and causing symptoms, such as increased intracranial pressure, dislocation syndrome, focal and general cerebral neurological symptoms, cerebral membrane irritation, signs of dural sinus compression, seizures, cosmetic defect, and sometimes scalp ulceration or bleeding [9,11,14,16,21,25,26].

The main treatment technique of this pathology is total or nearly total thyroidectomy and removal as many metastatic foci as possible. Removal of TC skull metastases is a difficult task for a surgeon as it is accompanied by massive blood loss and unpredictable surgery course due to hypervascular mass [9,21].

Gamma knife surgery is applied as an alternative therapy for TC skull bone metastases [27]. A fractionated stereotactic radiation technique is used for patients with metastatic cranial lesions near vital structures or in previously irradiated field.

Literature has few data on PTC skull metastases, and our surgical techniques will be presented for the first time, making this article relevant.

This study presents a case of surgical treatment for a giant hypervascular extra-intracranial papillary TC metastasis with preoperative tumor embolization and simultaneous cranioplasty with a customized stereolithographic three-dimensional (3D) titanium implant.

2. Case report

A 55-year-old female visited the clinic with complaints of a large painless mass on the left side of her head, which first manifested 4 years before presentation. She finally visited due to progressive impairment of movement in her right extremities, characterized by clumsiness, and numbness. She also noted signs of increased intracranial pressure.

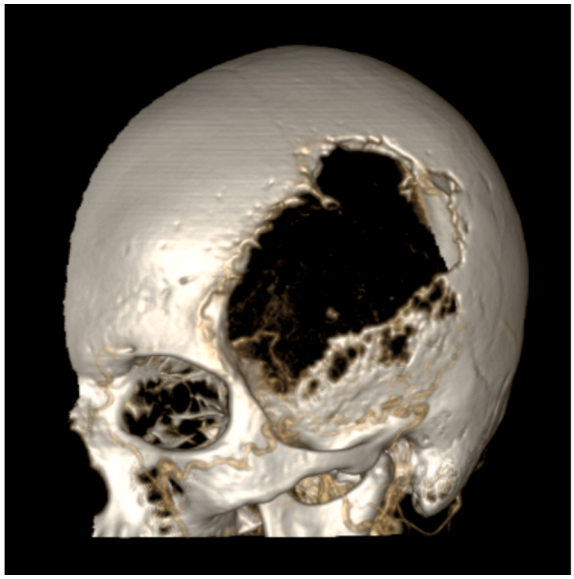

Fig. 2. Three-dimensional CT reconstruction. Left-sided frontal, temporal, and parietal bone destruction.

MRI + Gd and computed tomography (CT) angiogram showed a giant (105 × 102 × 75 mm) hypervascular extra cerebral mass lesion with extra-intracranial growth, destruction of frontal, temporal, and parietal bones on the left, brain compression, and 10-mm right-sided axial shift. The mass lesion is abundantly vascularized from the superficial temporal artery (STA), a. meningea media, middle cerebral artery (MCA), hypertrophied facial artery, and angular artery (Figs. 1 and 2).

Pre-embolization angiography shows a giant hypervascular tumor in the left frontal temporal and parietal regions with the most pronounced feeders from the left external carotid artery territory, left STA, left facial, and angular arteries, to a lesser extent from the recruited branches in the

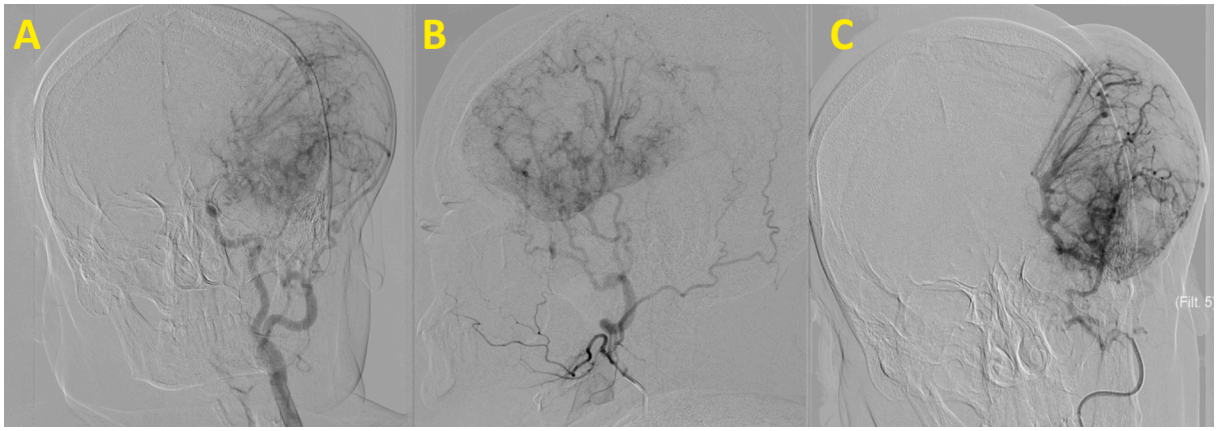

Fig. 3. A–C, Pre-embolization angiography. A giant (105 × 102 × 75 mm) hypervascular mass lesion with feeders from recruited branches of the left middle cerebral artery, a. meningea media (A), feeders from the left external carotid artery territory (B), and left superficial temporal artery (C).

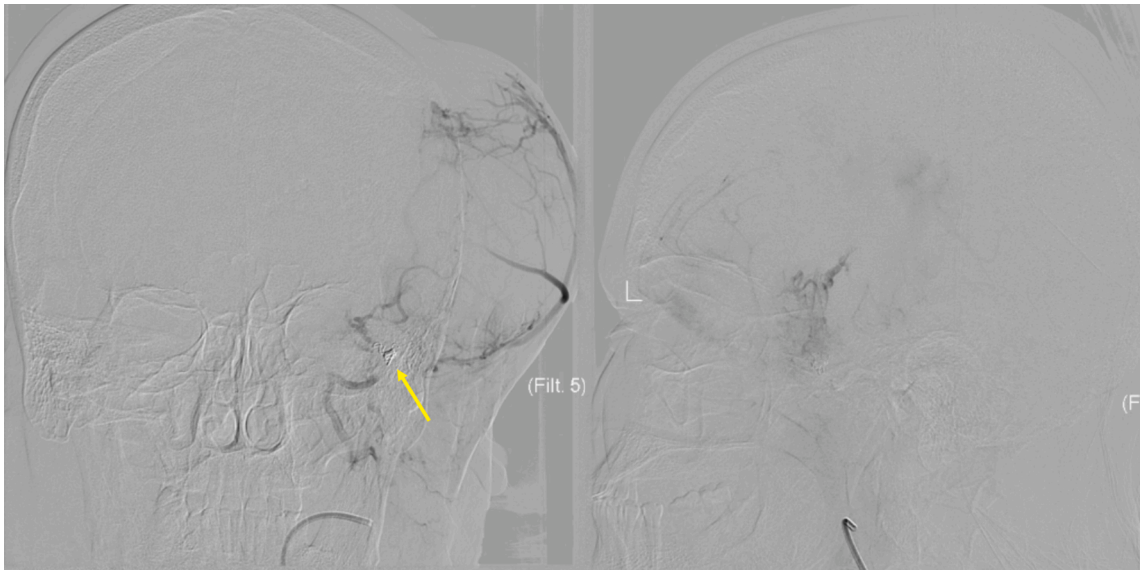

Fig. 4. Postembolization angiography. Tumor vascular tree was sub-totally embolized. Cerebral arteries are permeable. The shadow of detachable microcoil from a meningea media (yellow arrow). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

left MCA; the right external carotid artery territory has enhanced contrast (Fig. 3).

The following examinations were performed: thoracic cavity, abdominal cavity, retroperitoneal space, and pelvic cavity CT to detect primary tumor focus. No pathological mass lesions were revealed.

Given the neuroimaging data, the fact of damaged cranial vault and

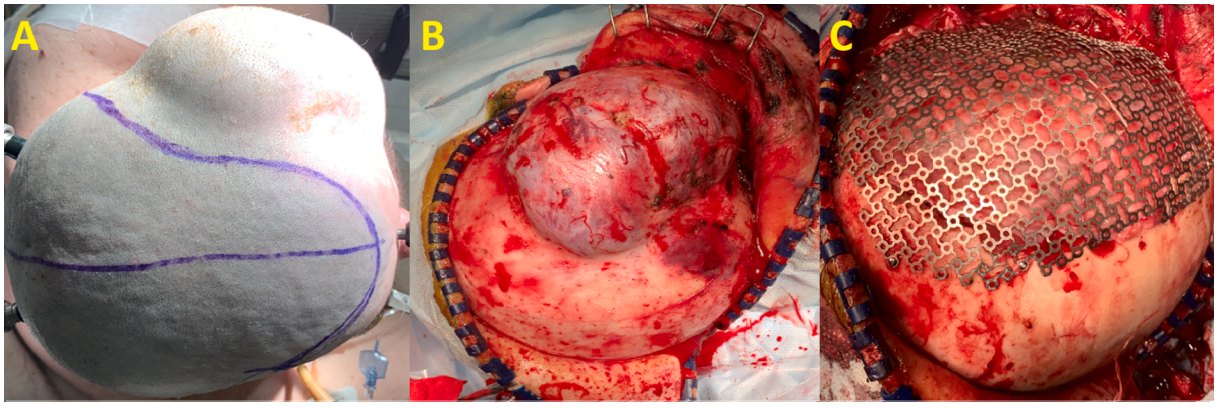

Fig. 5. Intraoperative photography. Giant (10 × 10 cm) left-sided frontotemporoparietal mass lesion (A). The tumor is separated from the soft tissue (B). Cranioplasty with a titanium individual stereolithographic 3D implant (C).

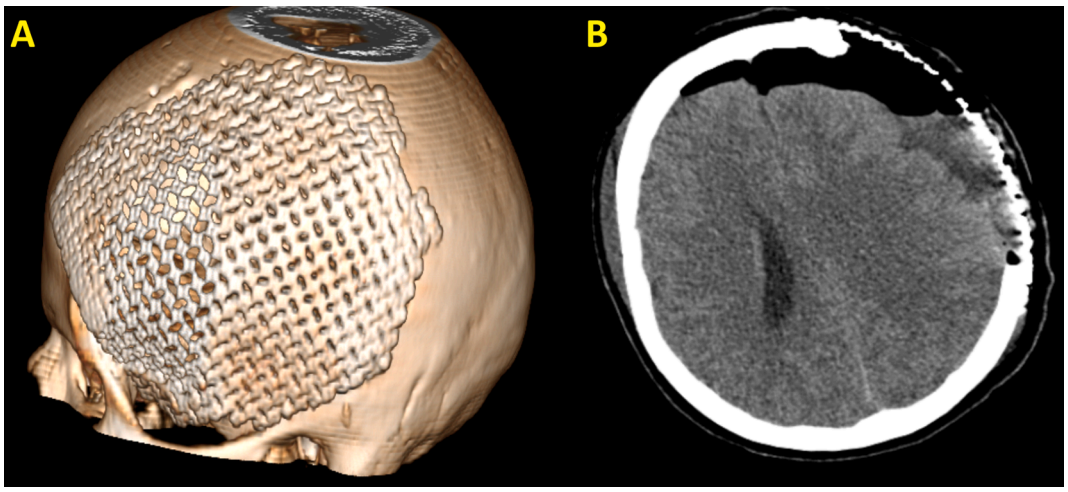

Fig. 6. A — three-dimensional CT reconstruction. Cranioplasty with a titanium individual stereolithographic 3D implant. B — postoperative axial computed tomography. The tumor is completely removed. There is no hemorrhagic component.

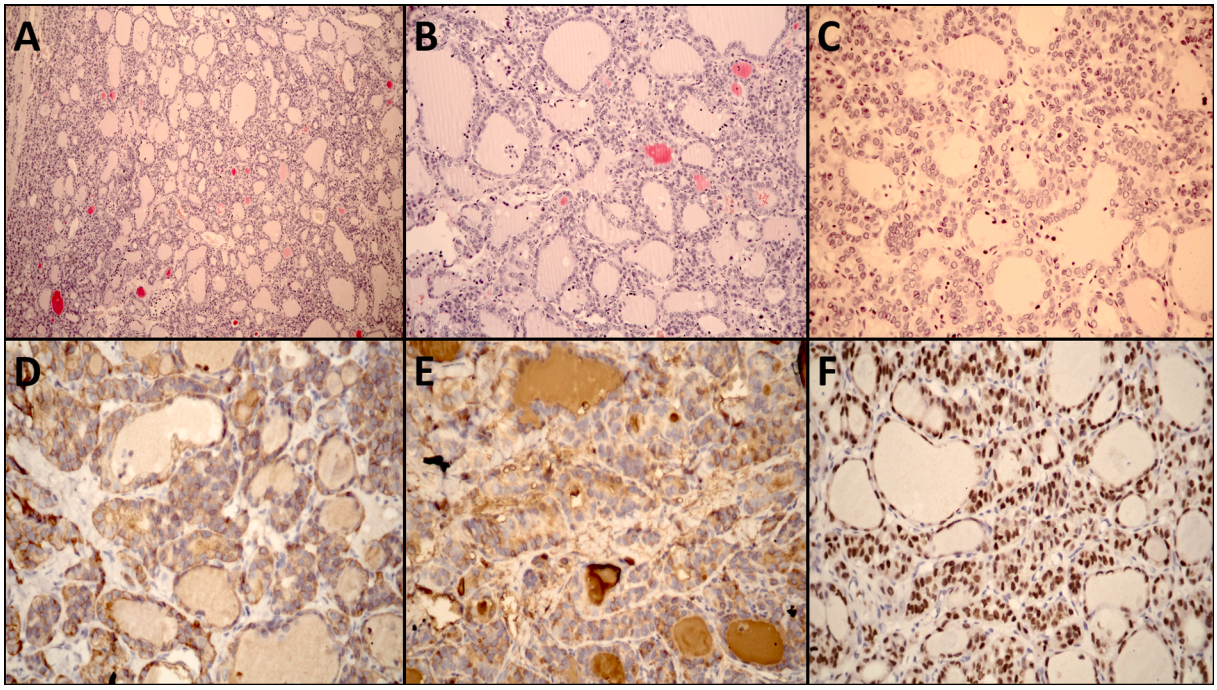

Fig. 7. Pathomorphological and immunohistochemical tumor examination. A — Hematoxylin-eosin, ×100. B — Hematoxylin-eosin, ×200. C — Hematoxylin-eosin, ×400. D — Immunohistochemical expression of pancytokeratin, ×400. E — Immunohistochemical expression of thyroglobulin, ×400. F — Immunohistochemical expression of TTF-1, ×400.

skull base bones, and internal organ CT data, the differential diagnosis between meningosarcoma and metastasis of cancer without primary focus was made.

Based on tumor peculiarities, namely, its hypervascular nature with the presence of large feeders from the external and internal carotid arteries, the following surgical techniques were suggested:

Stage 1: X-ray endovascular embolization of large tumor feeders to devascularize the tumor.

Stage 2: Surgical tumor removal with simultaneous cranioplasty with an individual stereolithographic 3D titanium implant.

The first stage involved X-ray endovascular tumor embolization. The tumor vascular tree was sequentially embolized from large feeders (bearing nsPVA V400EP, V600EP, and V800EP embolization particle). A detachable micro coil (axium of 6 × 20 mm) was placed in a. meningea media, from where the large feeders originated. About 90% of tumor vascular tree in the control angiogram series were embolized; the cerebral arteries were permeable (Fig. 4).

The second stage involved total surgical tumor removal. The tumor was first separated from MCA branches and large veins and orbital roof periosteum and was thus devascularized to the maximum extent. Then, the tumor node was fragmented and removed. The tumor was intimately fused with the cortex. The cranial dura mater was destroyed. Cranioplasty was performed with a previously prepared periosteal pedicle flap. Bone grafting with a titanium implant was performed based on an individual stereolithographic 3D model. Intraoperative blood loss was 1200 ml. Hemotransfusion was performed intraoperatively (Fig. 5). The blood loss was associated with partially preserved tumor blood supply at the base of the middle cranial fossa. This group of vessels was excluded

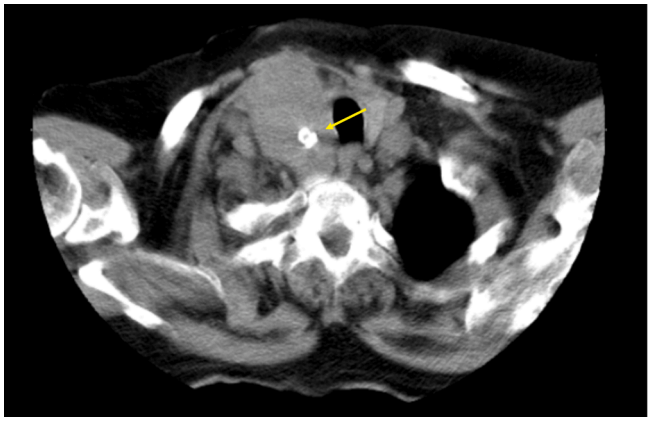

Fig. 8. Preoperative axial computed tomography. A 5.5 × 3.7-cm left-sided volumetric thyroid mass with a calcinate (yellow arrow). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

by the diathermocoagulation after the subtotal removal of the main tumor node.

Control series of CT scans show total tumor removal without evidence of hemorrhagic component, the signs of pneumocephalus. Bone defect was closed with a titanium implant prepared preoperatively using a stereolithographic 3D skull model (Fig. 6).

2.1. Histopathological evaluation

Histological examination showed tumor growth, consisting of different-sized follicle-like structures, filled with pink colloid-like content and solid areas with a small amount of stroma and foci of hemorrhages in the bones and dura mater (Fig. 7A, B). Tumor cells are medium-sized with a small amount of eosinophilic cytoplasm. The cell nuclei are round, different in size, with nuclear membrane irregularities, presence of longitudinal grooves, isolated small nuclei, and brightened central part of the nucleus (Fig. 7C).

Immunohistochemical study revealed expression of pancytokeratin (Fig. 7D), vimentin, thyroglobulin (Fig. 7E), TTF-1 (Fig. 7F), and Pax8 by tumor cells; no reaction with S100; and low proliferative activity (Ki67 expression in 4% of cells).

Based on morphological examination, metastasis of follicular variant of papillary TC (ICD-O 8340/3) was performed.

Based on the histological conclusion, the patient underwent targeted thyroid gland examination. According to CT findings, a thyroid gland mass lesion was detected (Fig. 8).

Radical surgical intervention, an extended thyroidectomy with prethyroid muscle invasion removal and modified bilateral neck dissection, was performed. An expression of histological examination confirmed the papillary cancer. The final conclusion of pathohistological and immunohistochemical examinations was follicular variant of papillary TC. One month postoperatively, in the hypothyroidism setting (TSH = 55 μIU/mL), ^{131}I radioiodine therapy was performed with a course dose of 200 mCi, followed by 1-year suppressive levothyroxine therapy to achieve the target TSH level of 0.2–0.4 μIU/ml. Subsequently, a replacement dosing therapy was performed [28–30].

The patient was discharged in satisfactory condition with improved neurological symptoms (Karnofsky performance status of 80 vs. 60 before the therapy).

Continuous screening (determining stimulated thyroglobulin level and thyroid sonography) showed no recurrence within 1 year.

3. Discussion

TC is the most common endocrine malignancy, with PTC as the most common subtype, followed by FTC. Distant metastases are diagnosed in 5–23% of patients with [7,9,10]. Cranial bone metastases are extremely rare and comprise 2.5% of all TC bone metastases [9,11–13].

Soft painless mass is increasingly observed as skull TC metastases, which can manifest by increased intracranial pressure, dislocation syndrome, focal and general cerebral neurological symptoms, cerebral membrane irritation, signs of dural sinus compression, seizures, cosmetic defect, sometimes scalp ulceration, and bleeding [9,11,14,16,21,25].

Given the tumor hypervascularization, surgical removal bears a high risk of significant blood loss and various postoperative complications due to unpredictable surgical course. Hence, our surgical techniques were based on reducing tumor blood flow volume to ensure subsequent total and safest possible mass lesion removal. Preoperative endovascular embolization is a safe and efficient method before the surgical thyroid metastasis removal.

Preoperative endovascular embolization can significantly reduce intraoperative blood loss and improve microsurgical technique, making surgical removal safer.

4. Conclusion

In our clinical case, the surgical treatment technique of a giant hypervascular metastasis of papillary TC allowed tumor removal radically and safely, which demonstrates its effectiveness. After the staged tumor "preparation," it was removed atraumatically, without significant blood loss and all preserved local anatomical structures, which made it possible to avoid numerous postoperative complications. Simultaneous cranioplasty with an individual stereolithographic 3D titanium implant was performed, which allowed prevention of postoperative complications associated with the presence of a large skull defect and cosmetic defects in the patient.

We believe that if a patient has a cranial bones mass, not only the thoracic cavity, abdominal cavity, abdominal space, and small pelvis organs should be examined, but also a comprehensive thyroid gland examination, namely, thyroid hormone level determination, sonographic thyroid gland examination, and thyroid gland CT, should be performed in order to rule out cranial bone metastasis.

Therefore, the above-mentioned methods, as well as recommendations for the examination of such patients, may be useful and applicable for the management of patients with TC in the future.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

References

[1] J.S. Barnholtz-Sloan, A.E. Sloan, F.G. Davis, F.D. Vigneau, P. Lai, R.E. Sawaya, Incidence proportions of brain metastases in patients diagnosed (1973 to 2001) in the metropolitan detroit cancer surveillance system, J. Clin. Oncol. 22 (14) (2004) 2865–2872, https://doi.org/10.1200/JCO.2004.12.149. PMID: 15254054.

[2] E. Tabouret, O. Chinot, P. Metellus, A. Tallet, P. Viens, A. Gonçalves, Recent trends in epidemiology of brain metastases: an overview, Anticancer Res. 32 (11) (2012) 4655–4662. PMID: 23155227.

[3] L. Nayak, E.Q. Lee, P.Y. Wen, Epidemiology of brain metastases, Curr. Oncol. Rep. 14 (1) (2011) 48–54, https://doi.org/10.1007/s11912-011-0203-y. PMID: 22012633.

[4] A.S. Achrol, R.C. Rennert, C. Anders, R. Soffietti, M.S. Ahluwalia, L. Nayak, S. Peters, N.D. Arvold, G.R. Harsh, P.S. Steeg, S.D. Chang, Brain metastases, Nat. Rev. Dis. Primers 5 (1) (2019) 5, https://doi.org/10.1038/s41572-018-0055-y. PMID: 30655533.

[5] P.W. Sperduto, S.T. Chao, P.K. Sneed, X. Luo, J. Suh, D. Roberge, A. Bhatt, A. W. Jensen, P.D. Brown, H. Shih, J. Kirkpatrick, A. Schwer, L.E. Gaspar, J. B. Fiveash, V. Chiang, J. Knisely, C.M. Sperduto, M. Mehta, Diagnosis-specific prognostic factors, indexes, and treatment outcomes for patients with newly diagnosed brain metastases: a multi-institutional analysis of 4,259 patients, Int. J. Radiat. Oncol. Biol. Phys. 77 (3) (2010) 655–661, https://doi.org/10.1016/j.ijrobp.2009.08.025. Epub 2009 Nov 26 PMID: 19942357.

[6] A.S. Berghoff, S. Schur, L.M. Füreder, B. Gatterbauer, K. Dieckmann, G. Widhalm, J. Hainfellner, C.C. Zielinski, P. Birner, R. Bartsch, M. Preusser, Descriptive statistical analysis of a real life cohort of 2419 patients with brain metastases of solid cancers, ESMO Open 1 (2) (2016) e000024, https://doi.org/10.1136/esmoopen-2015-000024.

[7] American Thyroid Association (ATA) Guidelines Taskforce on Thyroid Nodules and Differentiated Thyroid Cancer, Cooper DS, Doherty GM, Haugen BR, Kloos RT, Lee SL, Mandel SJ, Mazzaferri EL, McIver B, Pacini F, Schlumberger M, Sherman SI, Steward DL, Tuttle RM. Revised American Thyroid Association management guidelines for patients with thyroid nodules and differentiated thyroid cancer. Thyroid. 2009 Nov;19(11):1167-214. doi: 10.1089/thy.2009.0110. Erratum in: Thyroid. 2010 Aug;20(8):942. Hauger, Bryan R [corrected to Haugen, Bryan R]. Erratum in: Thyroid. 2010 Jun;20(6):674-5. PMID: 19860577.

[8] S. Jasim, L. Ozsari, M.A. Habra, Multikinase inhibitors use in differentiated thyroid carcinoma, Biologics 8 (2014) 281–291, https://doi.org/10.2147/BTT.S57619. PMID: 25506209; PMCID: PMC4260520.

[9] Shen J, Wang S, Zhao X, Shao X, Jiang X, Dai Y, Xu S, Pan X. Skull metastasis from follicular thyroid carcinoma: Report of three cases and review of literature. Int J Clin Exp Pathol. 2015 Nov;8(11):15285-93. PMID: 26823882; PMCID: PMC4713668.

[10] Khan SH, Hassan MU, Bhau RS. Iodine-131 avid distant metastasis in differentiated thyroid cancer: An initial institutional experience from the northern part of India. Indian J Nucl Med. 2015 Jul-Sep;30(3):227-32. doi: 10.4103/0972-3919.158530. PMID: 26170565; PMCID: PMC4479911.

[11] Y. Nagamine, J. Suzuki, R. Katakura, T. Yoshimoto, N. Matoba, K. Takaya, Skull metastasis of thyroid carcinoma. Study of 12 cases, J. Neurosurg. 63 (4) (1985) 526–531, https://doi.org/10.3171/jns.1985.63.4.0526. PMID: 4032016.

[12] G. Zettinig, B.J. Fueger, C. Passler, K. Kaserer, C. Pirich, R. Dudczak, B. Niederle, Long-term follow-up of patients with bone metastases from differentiated thyroid carcinoma – surgery or conventional therapy? Clin. Endocrinol. (Oxf.) 56 (3) (2002) 377–382, https://doi.org/10.1046/j.1365-2265.2002.01482.x. PMID: 11940050.

[13] A.L. Atkinson, A. Rosenthal, D. Nardiello, Follicular thyroid carcinoma presenting as a palpable head mass: a case report, Case Rep. Oncol. 3 (2) (2010) 106–109, https://doi.org/10.1159/000312708. PMID: 20740182; PMCID: PMC2919985.

[14] G.K. Wong, R. Boet, W.S. Poon, H.K. Ng, Lytic skull metastasis secondary to thyroid carcinoma in an adolescent, Hong Kong Med. J. 8 (2) (2002) 149–151. PMID: 11937672.

[15] Matsuno A, Murakami M, Hoya K, Yamada SM, Miyamoto S, Yamada S, Son JH, Nishido H, Ide F, Nagashima H, Sugaya M, Hirohata T, Mizutani A, Okinaga H, Ishii Y, Tahara S, Teramoto A, Osamura RY, Yamazaki K, Ishida Y. Clinicopathological and molecular histochemical review of skull base metastasis from differentiated thyroid carcinoma. Acta Histochem Cytochem. 2013 Oct;46(5):129-36. doi: 10.1267/ahc.13019. Epub 2013 Oct 25. PMID: 24194626; PMCID: PMC3813819.

[16] D. Feng, R. Rhatigan, S. Shuja, D. Wolfson, R. Makary, K. Koch, S. Masood, Papillary thyroid carcinoma with metastasis to the frontal skull, Diagn. Cytopathol. 37 (7) (2009) 522–526, https://doi.org/10.1002/dc.21070. PMID: 19459156.

[17] X. Li, G. Zhao, Y. Zhang, K. Ding, H. Cao, D. Yang, J. Zhang, Z. Duan, S. Xin, Skull metastasis revealing a papillary thyroid carcinoma, Chin. J. Cancer Res. 25 (5) (2013) 603–607, https://doi.org/10.3978/j.issn.1000-9604.2013.09.05. PMID: 24255586; PMCID: PMC3828433.

[18] N. Ozdemir, M. Senoğlu, U.D. Acar, M.S. Canda, Skull metastasis of follicular thyroid carcinoma, Acta Neurochir. (Wien) 146 (10) (2004) 1155–1158, https://doi.org/10.1007/s00701-004-0290-8, discussion 1158. PMID: 15744853.

[19] T. Hashiba, M. Maruno, Y. Fujimoto, T. Suzuki, K. Wada, T. Isaka, S. Izumoto, T. Yoshimine, Skull metastasis from papillary thyroid carcinoma accompanied by neurofibromatosis type 1 and pheochromocytoma: report of a case, Brain Tumor. Pathol. 23 (2) (2006) 97–100, https://doi.org/10.1007/s10014-006-0203-z. PMID: 18095126.

[20] Lu J, Frater JL, Kreisel FH, Marcus JN, Hassan A. Secondary lymphoma involving metastatic follicular thyroid carcinoma to the skull: a unique example of tumor-to-tumor metastasis. Head Neck Pathol. 2008 Sep;2(3):209-12. doi: 10.1007/s12105-008-0051-8. Epub 2008 Apr 22. PMID: 20614316; PMCID: PMC2807566.

[21] S. Miyawaki, R. Yamazaki, T. Harada, S. Takanashi, T. Nagashima, H. Nakaguchi, R. Okazaki, K. Yamazaki, Y. Ishida, A. Matsuno, Skull metastasis of thyroid papillary carcinoma, J. Clin. Neurosci. 14 (5) (2007) 481–484, https://doi.org/10.1016/j.jocn.2006.01.032. PMID: 17386370.

[22] Kelessis NG, Prassas EP, Dascalopoulou DV, Apostolikas NA, Tavernaraki AP, Vassilopoulos P PP. Unusual metastatic spread of follicular thyroid carcinoma: Report of a case. Surg Today. 2005 Apr;35(4):300-3. doi: 10.1007/s00595-004-2922-2. Erratum in: Surg Today. 2005;35(6):505. Evangelos, Prassas P [corrected to Prassas, Evangelos P]; Dimitra, Dascalopoulou V [corrected to Dascalopoulou, Dimitra V]; Nikiforos, Apostolikas A [corrected to Apostolikas, Nikiforos A]; Angeliki, Tavernaraki P [corrected to Tavernaraki, Angeliki P]; Pe. PMID: 15815846.

[23] J.D. Lin, E.C. Chan, T.C. Chao, K.T. Chen, C. Hsueh, Y.S. Ho, H.F. Weng, Expression of sodium iodide symporter in metastatic and follicular human thyroid tissues, Ann. Oncol. 11 (5) (2000) 625–629, https://doi.org/10.1023/a:1008340402577. PMID: 10907960.

[24] A. Matsuno, H. Katakami, R. Okazaki, S. Yamada, M. Sasaki, H. Nakaguchi, S. M. Yamada, K. Hoya, M. Murakami, K. Yamazaki, Y. Ishida, H. Iwasaki, J. Kuyama, K. Kakudo, Skull base metastasis from follicular thyroid carcinoma -two case reports-, Neurol. Med. Chir. (Tokyo) 50 (5) (2010) 421–425, https://doi.org/10.2176/nmc.50.421. PMID: 20505304.

[25] Stark AM, Eichmann T, Mehdorn HM. Skull metastases: clinical features, differential diagnosis, and review of the literature. Surg Neurol. 2003 Sep;60(3):219-25; discussion 225-6. doi: 10.1016/s0090-3019(03)00269-6. PMID: 12922038.

[26] Mitsuya K, Nakasu Y, Horiguchi S, Harada H, Nishimura T, Yuen S, Asakura K, Endo M. Metastatic skull tumors: MRI features and a new conventional classification. J Neurooncol. 2011 Aug;104(1):239-45. doi: 10.1007/s11060-010-0465-5. Epub 2010 Nov 26. PMID: 21110218; PMCID: PMC3151370.

[27] R. Kotecha, L. Angelov, G.H. Barnett, C.A. Reddy, J.H. Suh, E.S. Murphy, G. Neyman, S.T. Chao, Calvarial and skull base metastases: expanding the clinical utility of Gamma Knife surgery, J. Neurosurg. 121 (2) (2014) 91–101, https://doi.org/10.3171/2014.7.GKS141272. PMID: 25434942.

[28] S. Filetti, C. Durante, D. Hartl, S. Leboulleux, L.D. Locati, K. Newbold, M.G. Papotti, A. Berruti, ESMO Guidelines Committee. Electronic address: clinicalguidelines@esmo.org. Thyroid cancer: ESMO Clinical Practice Guidelines for diagnosis, treatment and follow-up†, Ann. Oncol. 30 (12) (2019) 1856–1883, https://doi.org/10.1093/annonc/mdz400. PMID: 31549998.

[29] Shindo ML, Caruana SM, Kandil E, McCaffrey JC, Orloff LA, Porterfield JR, Shaha A, Shin J, Terris D, Randolph G. Management of invasive well-differentiated thyroid cancer: an American Head and Neck Society consensus statement. AHNS consensus statement. Head Neck. 2014 Oct;36(10):1379-90. doi: 10.1002/hed.23619. Epub 2014 Aug 23. PMID: 24470171.

[30] Fugazzola L, Elisei R, Fuhrer D, Jarzab B, Leboulleux S, Newbold K, Smit J. 2019 European Thyroid Association Guidelines for the treatment and follow-up of advanced radioiodine-refractory thyroid cancer. Eur Thyroid J. 2019 Oct;8(5):227-245. doi: 10.1159/000502229. Epub 2019 Aug 28. PMID: 31768334; PMCID: PMC6873012.