



Intracranial primary collision tumours: A comprehensive systematic review on preoperative radiological accuracy and neuro-oncological insights

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ARTICLE INFO

Keywords:

Collision tumours
Neurooncology
Neuroimaging
Meningioma
Glioblastoma
Sellar region

ABSTRACT

Background: Intracranial collision tumours, characterized by the coexistence of two histologically distinct neoplasms within the same anatomical region without histological transition or metastatic interaction, are rare in neuro-oncology. Their atypical imaging appearance often mimics solitary lesions, posing diagnostic challenges. **Research question:** How accurately can preoperative neuroimaging identify both components of intracranial collision tumours, and what factors influence detection?

Methods: A systematic review was conducted following PRISMA guidelines and registered with PROSPERO (CRD420251008646). Included studies were adult case reports and series, including histologically confirmed intracranial collision lesions with preoperative neuroimaging. Tumour-to-tumour metastasis, synchronous, composite, or recurrent tumours were excluded.

Results: A total of 67 published cases were analysed, with a male-to-female ratio of 28:38 and a mean age of 52.4 years (SD = 15.95), ranging from 18 to 87 years. Meningioma was the most prevalent tumour type (65.7%), commonly paired with glioblastoma (26.9%). The sellar region was the most frequent location (34.3%), followed by the two frontal lobes (31.3%). Preoperative diagnosis correctly identified both lesions in only 26.9% of cases. Detection rates varied by anatomical location ($p = 0.0095$), whereas no clear association was observed with tumour pair type ($p = 0.1351$). Surgical resection was the primary treatment, frequently combined with chemo-radiotherapy. Recurrence occurred in 17.9%, especially in high-grade tumour components such as glioblastoma. Mean survival was 8.6 months, with 11.9% mortality. No statistically significant survival differences were observed between tumour pair types ($p = 0.149$).

Conclusion: Intracranial collision tumours remain diagnostically challenging. Improved neuroimaging and molecular understanding are crucial to enhance early diagnosis and optimize clinical management.

1. Introduction

Intracranial tumours present significant diagnostic and therapeutic challenges, particularly when two distinct neoplasms coexist within the same anatomical region. Although primary brain tumours are relatively common, collision tumours, where two histologically distinct neoplasms grow adjacent to each other without histological transition or metastatic interaction, remain exceedingly rare in neuro-oncology (Ashizawa et al., 2021; Chamberlin et al., 2021).

While the exact mechanisms underlying the development of collision tumours remain unclear, several hypotheses have been proposed, including coincidental occurrence, shared oncogenic pathways, tumour

microenvironment interactions, or prior cranial irradiation (Nedeljkovic et al., 2023; Syed et al., 2018). It has been proposed that a meningioma may create a permissive microenvironment that facilitates the development of a second lesion (Syed et al., 2018). Also, chronic oncologic or inflammatory changes induced by one neoplasm may stimulate the growth of another adjacent tumour (Dietterle et al., 2020). However, such theories remain speculation without confirmation of any definitive genetic or molecular interactions.

Given their rarity, collision tumours pose a critical diagnostic and therapeutic challenge, even if they have been documented in the literature since the 70s. Radiologically, they often mimic a single heterogeneous lesion, leading to potential misdiagnosis or misclassification as

This article is part of a special issue entitled: Brain Tumor Surgery published in Brain and Spine.

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<https://doi.org/10.1016/j.bas.2026.105943>

Received 6 June 2025; Received in revised form 16 December 2025; Accepted 10 January 2026

Available online 12 January 2026

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high-grade glioma, metastatic lesion, or atypical meningioma (Chamberlin et al., 2021; Shakally et al., 2018). Very rarely, and after careful radiological inspection, the two neoplasms can be suspected pre-operatively.

Distinguishing collision tumours from other intracranial oncologic entities is necessary for the definitive diagnosis and management. Tumour-to-tumour metastasis (TTM), which involves one tumour acting as a recipient for metastatic tumour cells from a distant primary malignancy, differs fundamentally from collision lesions in pathogenesis and clinical implications (Morita et al., 2012). Similarly, synchronous tumours refer to two independent neoplasms arising in the same patient but in separate anatomical locations without direct interaction (Yaghmour et al., 2016). Composite tumours, in contrast, arise from a single progenitor cell population and develop multiple histological grades, making them distinct from collision lesions (Atallah et al., 2024). Furthermore, differentiation from previously treated tumours that recur and develop a second neoplasm simultaneously is critical (Levitus and Charitou, 2018).

Despite the increasing recognition of intracranial collision tumours, their rarity and the heterogeneous nature of previously reported cases have limited the establishment of clear diagnostic and therapeutic guidelines. To date, most published literature consists of isolated case reports and small case series, often including TTM, synchronous tumours, and recurrence cases, thereby complicating the true characterization of collision tumours as a distinct entity (Ashizawa et al., 2021; Syed et al., 2018; Harb and Moral, 2019; Zhang et al., 2018). Few comprehensive systematic reviews have exclusively focused on intracranial collision tumours. These have, however, been limited to specific histological subtypes and do not assess the accuracy of preoperative suspicion based on imaging.

This paper aims to provide the most comprehensive analysis to date by strictly evaluating only actual intracranial collision tumours, excluding TTM, synchronous or composite tumours, and cases of recurrence. By conducting a focused review, this study will better characterize intracranial collision tumours' patterns and combinations, rate of accurate radiological diagnosis, and proposed treatment strategies, facilitating a better understanding of their clinical significance in neuro-oncology.

2. Methods

2.1. Study design

A systematic review was performed to address the preoperative radiological suspicion of intracranial collision lesions and summarize the existing evidence of their diagnostic challenges, tumour pairings, and treatment approaches in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines and registered with PROSPERO: CRD420251008646 (Page et al., 2021).

2.2. Eligibility criteria

Included studies fulfilled the following criteria: 1) Case reports or case series involving patients – 2) adults (≥ 18 years old) – 3) diagnosed with intracranial collision tumours, defined as two histologically distinct neoplasms coexisting within the same anatomical region without histological transition or metastatic relationship – 4) providing preoperative radiological imaging (MRI, CT, or other neuroimaging techniques) – 5) and corresponding histopathological confirmation. – 6) published in the English language.

2.3. Exclusion criteria

Excluded studies were those 1) including patients under 18 years old, – 2) cases of tumour-to-tumour metastasis (TTM) (cases where a mass acted as a recipient for metastatic cells), – 3) synchronous tumours (two

independent lesions in separate brain regions without anatomical interaction), – 4) cases of histological transition (tumours arise from a single progenitor cell population and develop multiple histological grades), – 5) recurrent tumours with transformation, where a second tumour developed after prior treatment, – 6) lesions associated with vascular abnormalities, – 7) studies lacking preoperative imaging data or histopathological confirmation, – 8) studies focusing on autopsy-based diagnoses, – 9) cases of non-intracranial cancers, or – 10) full-text availability.

2.4. Search strategy

A comprehensive search strategy was used to identify the eligible studies published until March 2025 in PubMed, Scopus, and Web of Science. The following search terms were used in combination: collision tumour, collision neoplasm, dual tumour, coexisting neoplasms, synchronous, intracranial, brain, central nervous system, CNS. Reference lists of included studies were also screened for additional cases.

2.5. Study selection

The study selection followed a double-reviewer process. All retrieved articles were screened for duplicates, which were removed. The remaining articles underwent title and abstract screening, with irrelevant studies excluded. A full-text review was performed on eligible studies to confirm adherence to inclusion and exclusion criteria. Discrepancies in study selection were resolved by discussion between two independent reviewers.

2.6. Data extraction

Data extraction involved collecting key information from each included case report, including study and patient demographics (country, year of publication, age, and sex, presenting symptomatology, imaging modality and findings, anatomical location, pre-operative suspicion of one vs. two lesions, surgical approach, histopathological confirmation, adjuvant treatments, recurrence, and follow-up duration). These data were collected in a comprehensive table to be used for further analysis.

2.7. Outcomes

The primary outcome was the proportion of cases where preoperative radiology correctly identified both tumours compared to cases where only one mass was suspected, and the second lesion was an intraoperative surprise. Cases were distributed to the group of both tumours only if it was clearly reported that the responsible team suspected two neoplasms pre-operatively. Additionally, the study assessed the most frequently misdiagnosed tumour pairings. Secondary outcomes were the distribution of the tumour types in the different collision pairs and the frequency of collision tumour locations, recurrence, and survival rates.

2.8. Quality assessment

The quality of included studies was evaluated using the Joanna Briggs Institute (JBI) Critical Appraisal Checklist for case reports and case series (Aromataris et al., 2024). Studies were evaluated based on clear patient case descriptions, including demographic and clinical details, comprehensive radiological and histopathological findings reporting, and adequate documentation of diagnostic methods, treatment, and follow-up. The overall quality of the cases eventually included in this review was good. The quality assessment results were summarized in the supplementary material (Appendix).

2.9. Data synthesis & statistical analysis

Data synthesis and statistical analysis were performed using Python (version 3.11) with pandas and scipy libraries. Continuous variables were expressed as mean ± standard deviation (SD), and categorical variables as frequencies and percentages. Preoperative diagnostic accuracy was assessed by comparing cases where both tumours were correctly identified versus those where only one lesion was detected. Sensitivity was calculated as the proportion of cases with dual tumour detection relative to the total number of confirmed cases, while specificity could not be calculated given the absence of true negative controls. In addition, subgroup analyses were conducted to evaluate the association between tumour type, anatomical location, and preoperative detection rates. Statistical significance was set at a p-value <0.05. Survival analyses were conducted using the scikit-survival package. Follow-up time was calculated from the date of surgery to either the date of death or the last follow-up. The Kaplan-Meier method was used to estimate survival probabilities, with censoring applied at the last known follow-up date for patients who were still alive. All visualizations were generated using seaborn and matplotlib with standardized scientific formatting to ensure a precise and clear presentation of the results.

3. Results

A total of 1294 citations were initially identified (Fig. 1). After removing 80 duplicate records, 1214 citations remained for screening. Of these, 1025 were excluded as unrelated to the study scope. From the remaining 189 publications, 18 could not be retrieved, and the rest were assessed for eligibility, from which 122 were excluded due to reasons such as synchronous lesions, TTM, composite tumours, recurrent neoplasms presenting with a second tumour, lesions associated with vascular abnormalities, paediatric cases, studies focusing on autopsy-based diagnosis. After these exclusions, 49 studies remained, to which an additional 18 were added from the search to their reference lists. As a result, a final dataset of 67 cases was included in our systematic review.

3.1. Demographics and patient characteristics

The 67 included case reports were published between 1971 and 2024. The cohort included 28 males (42.4 %) and 38 females (57.6 %), with one of unknown gender. The mean age was 52.4 years (SD = 15.95), with a distribution from 18 to 87 years. The included cases of collision tumours were published in 22 different countries. Table 1

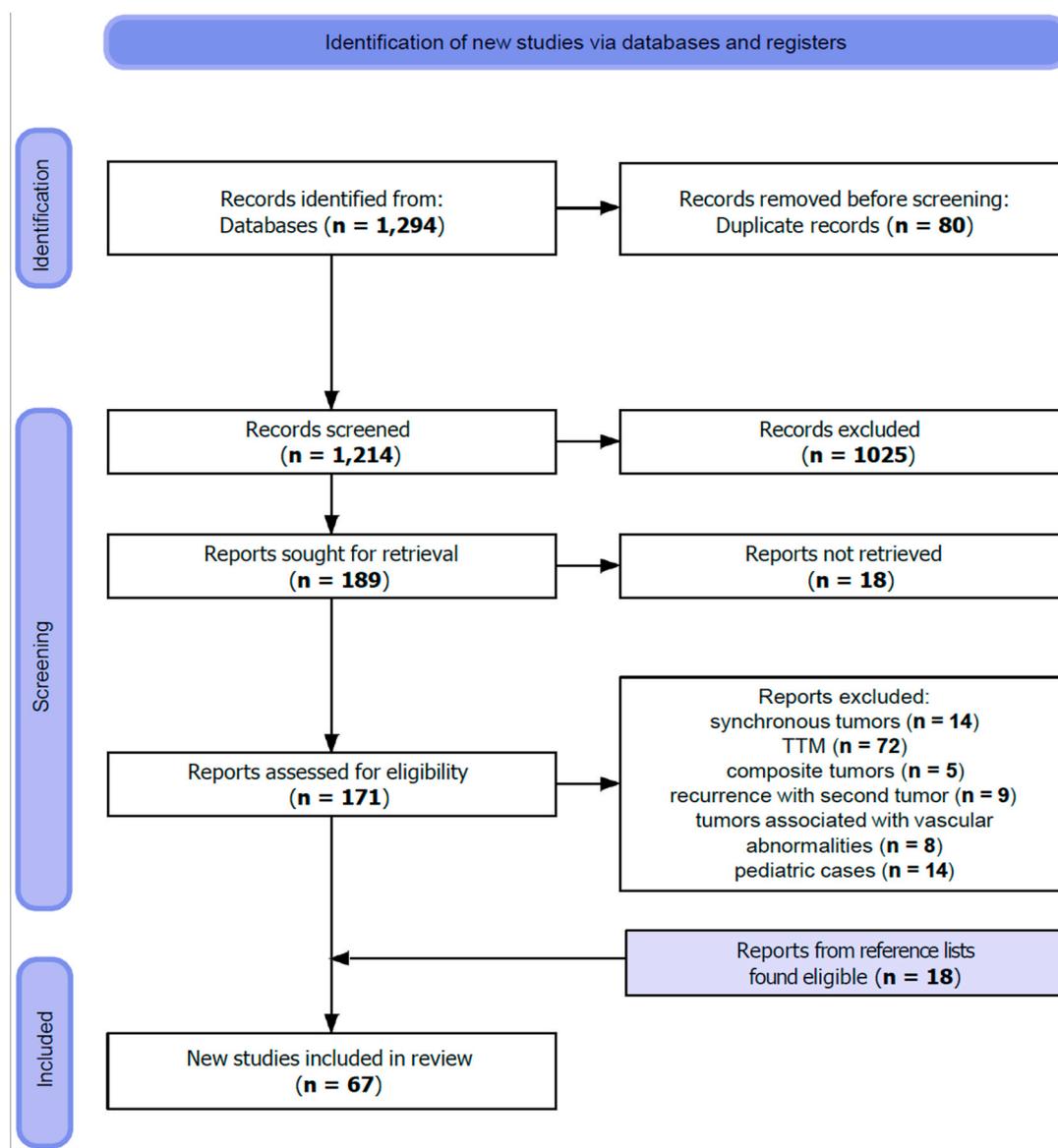


Fig. 1. “PRISMA flowchart”.

Table 1
Patients' basic characteristics.

	author	country	Age/ Gender	symptoms	location	pre-op suspicion of collision tumours	type of surgery	1st tumour	2nd tumour	adjuvant or other treatment, recurrence	f/ up (m)	mortality (in days)
1	Lobo et al., 2024	Portugal	54 F	seizures	left frontal	both	excision	Meningioma (WHO grade I)	IDH mutated with 1p/19q codeletion oligodendroglioma		N/A	
2	Kikuta et al., 2023	Japan	54 M	incidental	sella	both	endoscopic endonasal approach.	adamantinomatoustype craniopharyngioma	nonfunctioning pituitary adenoma	recurrence required re-operation	N/A	
3	Nedeljkovic et al., 2023	Serbia	18 M	seizures	right frontal	one	right frontoparietal craniotomy with a gross total resection	ganglioglioma (WHO grade 1)	ependymoma (WHO grade 3)	radiation		12
4	Sobstyl et al., 2023	Poland	64 M	speech impairment, hemiparesis.	right temporal	one	right frontotemporal craniotomy	meningothelial meningioma (WHO grade I)	glioblastoma IDH-wildtype (WHO grade IV)			2
5	Lin et al., 2022	Taiwan	56 F	weakness, dizziness, headache	left frontal	one	bi-coronal craniotomy	clear cell atypical meningioma (WHO grade II)	glioblastoma	adjuvant chemoradiation, recurrence in 6m	N/A	
6	Zacharewski et al., 2022	USA	72 F	expressive aphasia and headache	left frontal	both	pre-op MMA embolization & left craniotomy with motor mapping.	dural meningiomatosis (WHO Grade I)	glioblastoma			15
7	Ashizawa et al., 2021	Japan	46 F	headache and gait ataxia	posterior fossa	one	suboccipital craniotomy and total resection in 2 steps	meningioma (WHO grade I)	hemangiopericytoma (SFT/HPC)	radiotherapy		21
8	Chamberlin et al., 2021	USA	42 F	seizures, headaches, memory loss	right parietal	one	biopsy and right parietal craniotomy	meningioma (WHO grade I)	glioblastoma (WHO grade IV)		0.5	18
9	Gezer et al., 2020	Turkey	34 F	menstrual irregularity, weakness	sella	one	extended endoscopic endonasal approach	meningioma	pituitary corticotroph adenoma			8
10	Harb and Moral, 2019	USA	33 M	Headache, blurry vision & vomiting	left frontal	both	excision	atypical meningioma (WHO grade II)	anaplastic ependymoma (WHO grade III)	recurrence required re-operation	N/A	
11	Truong et al., 2019	Vietnam	61 F	headache and mild left hemiparesis	right frontal	both	complete macroscopical excision	fibroblastic meningioma	astrocytoma (WHO grade II)		N/A	
12	Levitus and Charitou, 2018	USA	44 F	presented post-head injury	sella	one	endoscopic transsphenoidal excision	gangliocytoma	mixed GH cell/PRL cell adenoma (WHO Grade I)			10
13	Syed et al., 2018	USA	71 F	headache, nausea, and dizziness	right temporal	one	stereotactic biopsy & right craniotomy	meningioma (WHO grade I)	glioblastoma (WHO grade IV)	radiation therapy and concomitant temozolomide		9
14	Yan et al., 2018	China	50 M	memory decline, drowsiness, aphasia, bradykinesia	left frontal	one	left frontal craniotomy	transitional meningioma	ENKTCL nasal type	polychemotherapy, recurrence in 2m	N/A	
15	Zhang et al., 2018	China	66 F	headache, nausea, and vomiting	right frontal	both	right parietal craniotomy.	meningioma (WHO grade I)	glioblastoma (WHO grade IV)		N/A	
16	Graffeo et al., 2017	USA	64 F	hearing loss and ear fullness	CPA	both	left retrosigmoid craniotomy	WHO grade I fibrous type meningioma	vestibular schwannoma			3
17	Graffeo et al., 2017	USA	42 M	incidental	CPA	both	right retrosigmoid craniotomy	WHO grade I psammomatous meningioma	vestibular schwannoma			3

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Table 1 (continued)

author	country	Age/ Gender	symptoms	location	pre-op suspicion of collision tumours	type of surgery	1st tumour	2nd tumour	adjuvant or other treatment, recurrence	f/ up (m)	mortality (in days)
18 Heng et al., 2017	China	46 F	headache with deterioration of visual acuity	sella	one	extended endoscopic endonasal approach	gangliocytoma	pituitary adenoma	gamma knife radiosurgery	8	
19 Zhao et al., 2017	China	58 F	acromegaly and snoring	sella	one	transsphenoidal pituitary resection in 2 steps	meningioma	GH-secreting pituitary adenoma		N/A	
20 Zhao et al., 2017	China	58 F	acromegaly and snoring	sella	one	transsphenoidal pituitary resection & craniotomy	meningioma	GH-secreting pituitary adenoma		N/A	
21 Ban et al., 2017	UK	74 M	retro-orbital pain, headaches, nausea - pituitary apoplexy	sella	one	Endoscopic transsphenoidal resection	diffuse large B-cell non-Hodgkin's lymphoma (DLBCL)	FSH-pituitary adenoma	chemotherapy, intrathecal methotrexate	32	
22 Shahmohammadi et al., 2016	Iran	49 F	left hemiparesis, seizures, nausea and vomiting	right frontal	both	right frontal craniotomy	meningothelial meningioma	glioblastoma	radiotherapy & chemotherapy with temozolomide	N/A	
23 Yaghmour et al., 2016	Arabia	32 M	headache	left temporal	one	partial resection	meningioma (WHO grade I)	glioblastoma (WHO grade IV)	recurrence	2,5	70
24 Jukes et al., 2016	Australia	54 F	acromegaly	sella	one	transsphenoidal resection	gangliocytoma & ganglioglioma (WHO grade I)	pituitary adenoma (WHO grade I)		N/A	
25 Karsy et al., 2015	Austria	70 F	altered mental status, mutism, and incontinence	sella	one	EVD & transsphenoidal approach to sella	fibroepithelial meningioma	pituitary macroadenoma	permanent VP-shunt	N/A	
26 Ruiz et al., 2015	Spain	86 M	drowsiness & hemiparesis.	right frontal	both	excision	secretory meningioma (WHO grade I)	glioblastoma (WHO grade IV)	radiotherapy & chemotherapy with temozolomide	15	
27 Ruiz et al., 2015	Spain	22 F	seizures	right frontal	one	excision	secretory meningioma (WHO grade I)	astrocytoma	Recurrence with 2n surgery & external beam radiotherapy, chemotherapy	44	1320
28 Verma et al., 2015	India	40 F	hearing loss	CPA	both	lateral suboccipital retrosigmoid craniectomy	meningothelial meningioma (WHO Grade I)	schwannoma	VP-shunt	11	
29 Finzi et al., 2014	Italy	75 F	sixth left nerve palsy	sella	one	endoscopic transsphenoidal approach	adamantinomatous craniopharyngioma	pituitary adenoma		10	
30 Kurdi et al., 2014	Arabia	54 F	atypical left facial pain and numbness	left temporal	one	left orbitozygomatic-temporal craniotomy	anaplastic astrocytoma (WHO grade III)	malignant trigeminal nerve sheath tumour (MTNST)	recurrence	9	270
31 Tanriover et al., 2014	Turkey	39 F	acromegaly	sella	one	endonasal transsphenoidal approach	gangliocytoma	pituitary adenoma secreting GH		N/A	
32 Yao et al., 2014	China	52 F	progressive left-side weakness and dizziness	right frontal	one	gross total excision	mixed granular cell astrocytoma (GCA)	fibrosarcoma	radiotherapy and chemotherapy	15	
33 Jin et al., 2013	China	47 F	intermittent blurred vision, headaches	sella	one	Transsphenoidal surgery(1st) + right frontal craniotomy (2nd)	adamantinomatous craniopharyngioma	nonfunctional pituitary adenoma		3	

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Table 1 (continued)

author	country	Age/ Gender	symptoms	location	pre-op suspicion of collision tumours	type of surgery	1st tumour	2nd tumour	adjuvant or other treatment, recurrence	f/ up (m)	mortality (in days)
34	Martin et al., 2013	USA	62 F	headache, neck pain, nausea, blurred vision, dizziness	left parietal	one	left parietooccipital craniotomy	meningioma	low-grade B-cell lymphoma	chemotherapy	12
35	Ghosal et al., 2012	India	24 F	motor and sensory trigeminal nerve deficits	sella	one	left frontotemporal craniotomy and tumour decompression	meningioma WHO Grade I	schwannoma		N/A
36	Jaiswal et al., 2012	India	25 M	headache, orbital pain, acromegaly, dakryrrhoea	sella	one	trans-sphenoidal approach	gangliocytoma	pituitary adenoma		N/A
37	Frassanito et al., 2011	Italy	72 M	hearing loss.	CPA	both	left retrosigmoid craniotomy	meningioma	schwannoma		N/A
38	X. Yan et al., 2011	China	45 M	seizures	right temporal	one	frontotemporal craniotomy	DNET (WHO grade I)	CPNA		156
39	Basil et al., 2011	USA	29 F	headache, nausea, vomiting, speech disturbance, cervical lymphadenopathy	left parietal	one	left parietal craniotomy	anaplastic astrocytoma	B-cell lymphoma		N/A
40	Sahli et al., 2011	Switzerland	74 M	headache, nausea, vomiting, third nerve palsy	sella	one	transseptal transsphenoidal approach	chordoma	chromophobe pituitary adenoma		N/A
41	Binello et al., 2010	USA	40 M	seizures	right frontal	one	right frontal craniotomy and gross total resection	meningioma (WHO grade I)	hemangiopericytoma (WHO grade II)	recurrence in 28m required re-operation	N/A
42	Chen et al., 2010	China	63 F	headache and right upper limb weakness.	left frontal	both	left frontal craniotomy	fibroblastic meningioma	glioblastoma		N/A
43	Grauvogel et al., 2010	Germany	46 F	hearing loss, tinnitus, and gait ataxia	CPA	one	lateral suboccipital approach	fibromatous meningioma	schwannoma		3
44	Suzuki et al., 2010	Japan	75 M	mild headache	left temporal	one	left pterional craniotomy.	meningothelial meningioma	glioblastoma	radiotherapy & chemotherapy with temozolomide	1
45	Rivera et al., 2010	Canada	58 M	diplopia - pituitary apoplexy	sella	one	transsphenoidal approach	plasma cell tumour	lactotroph pituitary adenoma	thalidomide, pamidronate & bone marrow transplant & SRS	N/A
46	Gokden and Mrak, 2009	USA	47 M	headache and visual loss	sella	one	Transsphenoidal resection	adamantinomatous craniopharyngioma	pituitary adenoma		12
47	Koutourousiou et al., 2009	Greece	38	acromegly	sella	one	sub-labial trans-sphenoidal approach	schwannoma (WHO Grade I)	sparsely granulated GHproducing adenoma		4
48	Sargis et al., 2009	USA	59 M	progressive vision loss	sella	one	subtotal transcranial resection	adamantinomatous craniopharyngioma	Pituitary gonadotroph adenoma		N/A
49	Takeuchi et al., 2009	Japan	53 F	emotional instability-confusion, drowsiness, headache, and left hemiparesis	right temporal	one	subtotal resection	gangliocytoma	anaplastic oligodendroglioma	Radiotherapy, chemotherapy with nimustine/ hydrochloride	7
50	Kutz et al., 2009	USA	43 F	headache, hearing loss, and imbalance	CPA	both	translabyrinthine craniotomy	meningioma	schwannoma		N/A

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Table 1 (continued)

author	country	Age/ Gender	symptoms	location	pre-op suspicion of collision tumours	type of surgery	1st tumour	2nd tumour	adjuvant or other treatment, recurrence	f/ up (m)	mortality (in days)
51 Yoshida et al., 2008	USA	29 M	atrial fibrillation	sella	one	endonasal endoscopic transsphenoidal resection	adamantinomatous craniopharyngioma	pituitary adenoma	transfusion & arterial embolization for recurrent epistaxis	N/A	
52 Suck et al., 2008	USA	46 M	face pain, dizziness, seizures, worsening of mobility	left frontal	one	left frontal craniotomy	syncytial meningioma	plasmacytoma	radiotherapy	24	
53 Nestler et al., 2007	Germany	49 M	weight loss, altered mental state, disorientation	left frontal	both	excision	fibrous meningioma (WHO grade I)	glioblastoma (WHO grade IV)	radiotherapy, temozolomide, recurrence in 7m required debulking developed lymphoma	14	
54 Riccioni et al., 2007	Italy	66 F	palpable left frontal-parietal lump	left frontal	one	bilateral frontal-parietal craniotomy, with complete resection	transitional meningioma	grade IIIB follicular lymphoma		12	
55 George et al., 2007	UK	71 M	headache, confusion, dizziness, lack of coordination, hemiparesis	right temporal	both	right temporal craniotomy	meningothelial meningioma (WHO grade I)	B-cell lymphoma		N/A	
56 Tugcu et al., 2006	Turkey	42 M	hemiparesis and speech disturbances	left parietal	both	craniotomy - total removal	transitional type meningioma	glioblastoma		N/A	
57 Maiuri et al., 2005	Italy	65 M	seizures	left frontal	one	one-stage removal	meningioma	Grade II astrocytoma		N/A	
58 Drlicek et al., 2004	Germany	51 M	Headache, facial palsy	left frontal	one	resection	meningothelial meningioma (WHO grade I)	glioblastoma (WHO grade IV)		N/A	
59 Goyal et al., 2003	India	72 M	headache and seizures	right temporal	one	right frontotemporal craniotomy	fibroblastic meningioma	glioblastoma	radiotherapy	N/A	
60 Prayson et al., 2002	USA	87 F	worsening mobility, urinary incontinence, and left hemiparesis	right frontal	one	right frontal craniotomy with gross total resection	meningotheliomatous meningioma (WHO grade I)	astrocytoma (WHO grade III)		N/A	
61 Lüdemann et al., 2000	Germany	59 F	hearing loss	CPA	one	right-sided suboccipital craniectomy	meningothelial meningioma	schwannoma		N/A	
62 HAKAN et al., 1988	Turkey	30 M	occipital headaches and right arm numbness	left frontal	both	left frontotemporal craniotomy	fibroblastic meningioma	glioblastoma	Radiotherapy, recurrence	5	150
63 Cannavò et al., 1993	Italy	47 F	acromegaly	sella	one	right frontotemporal craniotomy	endotheliomatous meningioma	pituitary adenoma		3	
64 Asada et al., 1990	Tokyo	52 F	headache and acromegaly.	sella	one	transsphenoidal resection	gangliocytoma	pituitary adenoma		1	
65 Vaquero et al., 1990	Spain	75 F	intermittent dysphasia and dysesthesia	left parietal	one	left parietal craniotomy	psammomatous meningioma	glioblastoma		0.5	14
66 Marra et al., 1977	Italy	63 M	left hemiparesis and bilateral severe papilledema	right parietal	one	right frontoparietal craniotomy	meningotheliomatous meningioma	glioblastoma	recurrence	8	240
67 Prabhakar et al., 1971	India	29 M	acromegaly	sella	one	right frontal craniotomy	craniopharyngioma	pituitary adenoma		0,1	4

presents the main characteristics.

Headache was the predominant presenting symptom in 26.9 % of cases, often accompanied by other neurological or systemic manifestations. Seizures and acromegaly (in sellar lesions) were the next most common presentations, each appearing in 13.4 % of cases. Notably, a significant proportion of patients presented with nausea and dizziness, hearing loss, and vomiting. However, some cases were discovered incidentally, while others manifested with acute neurological deterioration, including seizures or focal neurological deficits.

Among the total 134 tumours (67 pairs) analysed, meningioma was the most prevalent type (43 cases & 1 dural meningiomatosis case, 65.7 %), followed by pituitary adenoma (22 cases, 32.8 %), and glioblastoma (18 cases, 26.9 %). The remaining tumours each appear in 1.5 % (1 case) of the total cases. Fig. 2 summarizes the common tumour types in the collision lesions.

In this cohort, 21 different collision tumour pairs were identified. The most common pairs were meningioma-glioblastoma (18 cases, 26.9 %), followed by meningioma-schwannoma (8 cases, 11.9 %), and craniopharyngioma-pituitary adenoma (7 cases, 10.4 %). Other collision tumour pairs are illustrated in Fig. 3.

Collision tumours grew in different intracranial locations. The analysis revealed that the sellar region was the predominant site, accounting for approximately one-third (34.3 % - 23 cases) of all cases. The frontal lobes together represented the second most common location, with left frontal (17.9 % - 12 cases) and right frontal (13.4 % - 9 cases) together comprising 31.3 % of cases. The cerebellopontine angle (CPA) then follows at 10.4 % with 7 collision tumours. Other sites of collision tumour growth include the temporal lobes (right 9 % - 6 cases, and left 4.5 % - 3 cases), the parietal lobes (left 6 % - 4 cases, right 3 % - 2 cases), and 1 case in the cerebellum (1.5 %).

3.2. Preoperative diagnostic accuracy

Preoperative diagnostic accuracy revealed that both tumours were suspected in only 18 cases (26.9 %), while in 49 cases (73.1 %), only one tumour was suspected preoperatively, and the second tumour was an intraoperative finding (Fig. 4). While specificity could not be calculated due to the nature of the dataset (all cases were collision tumours), the low sensitivity (26.9 %) highlights significant challenges in preoperative detection.

The detection rates varied considerably by tumour type combination, with the meningioma and schwannoma combination showing the highest detection rate (62.5 %, 5/8 cases) among common pairs, followed by the meningioma and glioblastoma pair (44.4 %, 8/18 cases). The association between diagnostic accuracy and tumour pair type was not statistically significant ($p = 0.1351$).

Regarding anatomical location, the CPA region demonstrated the highest detection rate (71.4 %, 5/7 cases), while only 1 out of 23 sellar region tumours was detected preoperatively (4.3 %, 1/23 cases). Frontal lobe tumours showed moderate detection rates (44.4 %–50 %). Although a statistically significant association between anatomical location and detection rate ($p = 0.0095$) was observed using the Chi-square test (Fig. 5), this finding should be considered exploratory due to small subgroup sizes and the absence of correction for multiple comparisons.

3.3. Therapeutic approaches

Treatment primarily involved surgical resection, often with radiotherapy and chemotherapy. The surgical approaches employed in these cases demonstrate a diverse range of techniques, with transsphenoidal/endonasal approaches being the most common, accounting for 25.4 % (17/67), as expected due to the increased number of sella lesions. All other cases were managed with different types of craniotomies and ranged from total to subtotal resections. Some cases required staged procedures or combined approaches, such as cases where transsphenoidal surgery was followed by craniotomy. The choice of surgical approach appears to have been tailored to tumour location, size, characteristics, and specific patient factors.

Analysis of the adjuvant treatments revealed that radiation therapy was the predominant treatment modality in 63.6 % of cases (Aromataris et al., 2024), often combined with chemotherapy, which was administered in 45.5 % of cases (Levitus and Charitou, 2018). The chemotherapy protocols varied, with temozolomide being the most frequently used agent, though some cases received more complex regimens, including intrathecal methotrexate, cytarabine, or nimustine hydrochloride. VP shunts were required in 2 cases.

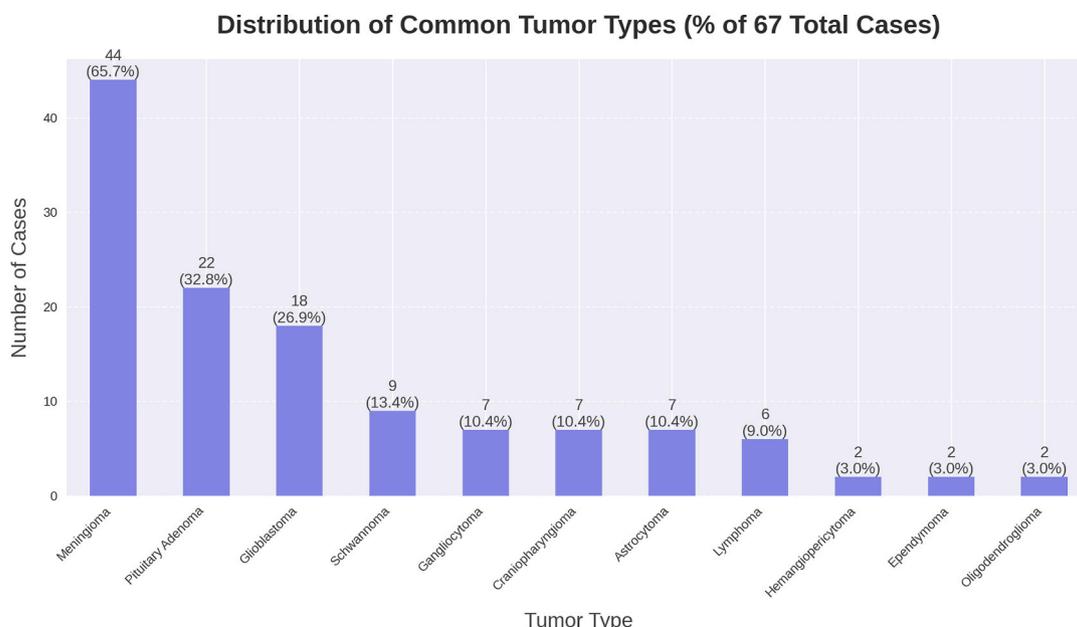


Fig. 2. “Distribution of Common Tumour types (% of 67 Total Cases)”.

Brain Tumor Pairs Distribution

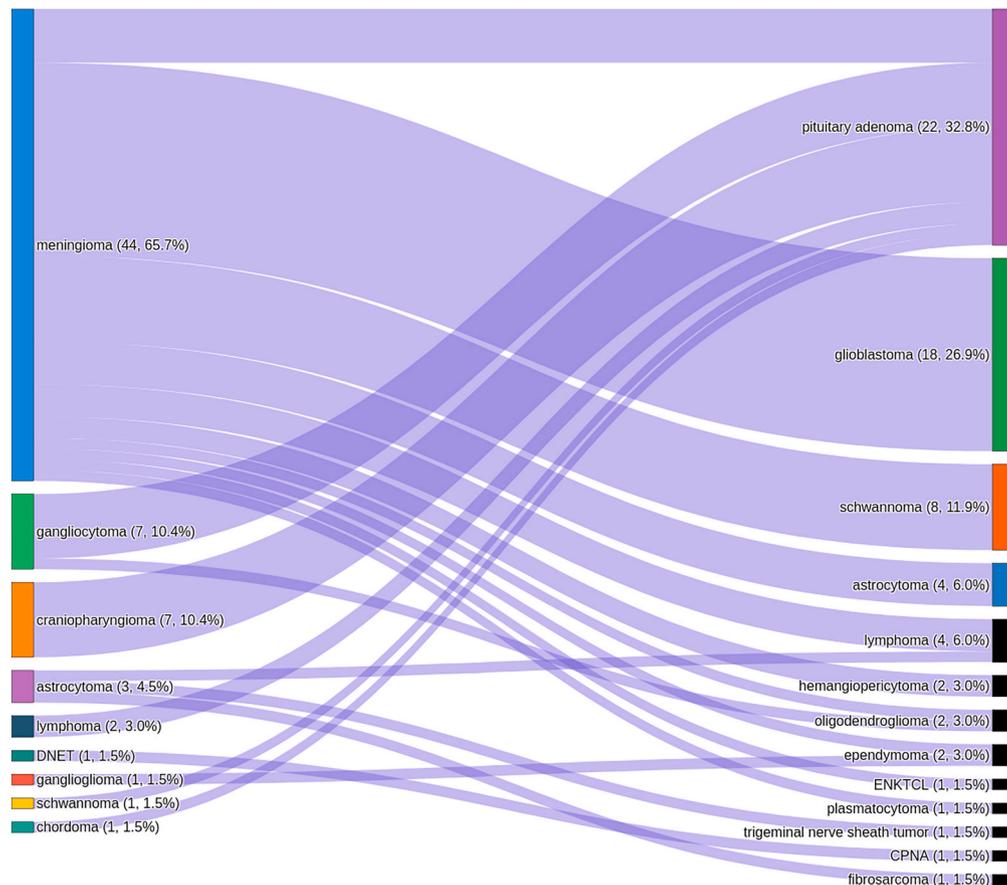


Fig. 3. "Brain tumour pairs distribution".

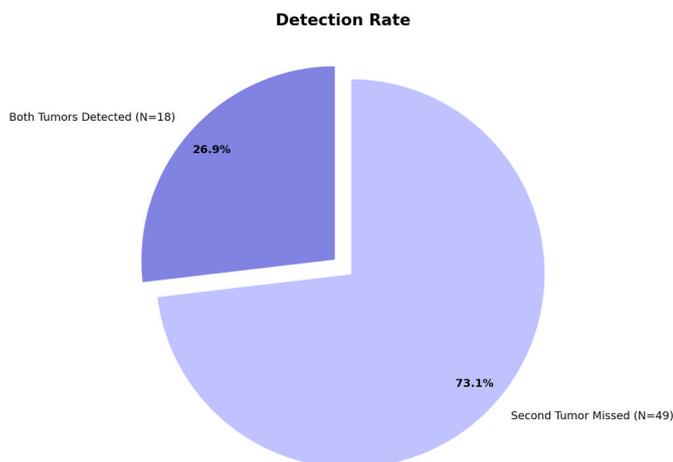


Fig. 4. "Preoperative detection rate of collision tumours".

3.4. Follow-up, recurrence rates, and survival outcomes

Out of 67 cases, 36 patients (53.7 %) were followed up for a mean of 13.8 months (SD = 26.1), ranging from a week to 156 months. Recurrence of tumours, mainly of their high-grade component, was reported in 12 cases (17.9 %). Most recurrences occurred within the first year, with a mean of 10.8 months (SD = 10.1). Revision surgery was required in 5 recurrent cases (41.7 %). Among tumour pairs with sufficient cases for analysis, meningioma and glioblastoma were the most common

combinations found to recur at 45.5 % (5/11), as expected. However, robust statistical correlations between tumour types and outcomes are limited due to the small sample sizes in most tumour pair categories and the high proportion of cases without complete follow-up data (46.3 %).

The survival data from this cohort suggest that death occurred in 8 cases (11.9 %) with a wide range in survival times, from 4 days to 1320 days (43.4 months). The mean time post-surgical operation was 260.8 days (8.6 months), while median survival was 110 days (3.6 months). Most deaths (75 %) occurred within the first 240 days (8 months), but one notable outlier at 1320 days (43.4 months) significantly affected the mean. Thus, these findings should be interpreted cautiously, given the small sample size. The cause of death was reported in 4 cases: 2 patients passed away due to recurrence, and the other 2 because of pulmonary complications and hypothalamic polydipsia. The analysis of survival outcomes across different collision tumour pairs is summarized in Fig. 6. No statistically significant differences in survival between the collision tumour pairs were found, likely due to the small sample size - Kruskal-Wallis Test (comparing all groups): H-statistic: 5.333, p-value: 0.149, Mann-Whitney U Test (comparing meningioma-glioblastoma vs. others): U-statistic: 5.000, p-value: 0.57.

4. Discussion

4.1. Overview of key results

This systematic review examines 67 cases of histologically verified intracranial collision tumours from 1971 to 2024, with a focus on pre-operative diagnostic accuracy, tumour pairings, and outcomes. Meningioma-glioblastoma was the most frequently reported pair (26.9



Fig. 5. "Detection rates across anatomical locations (p = 0.0095)".

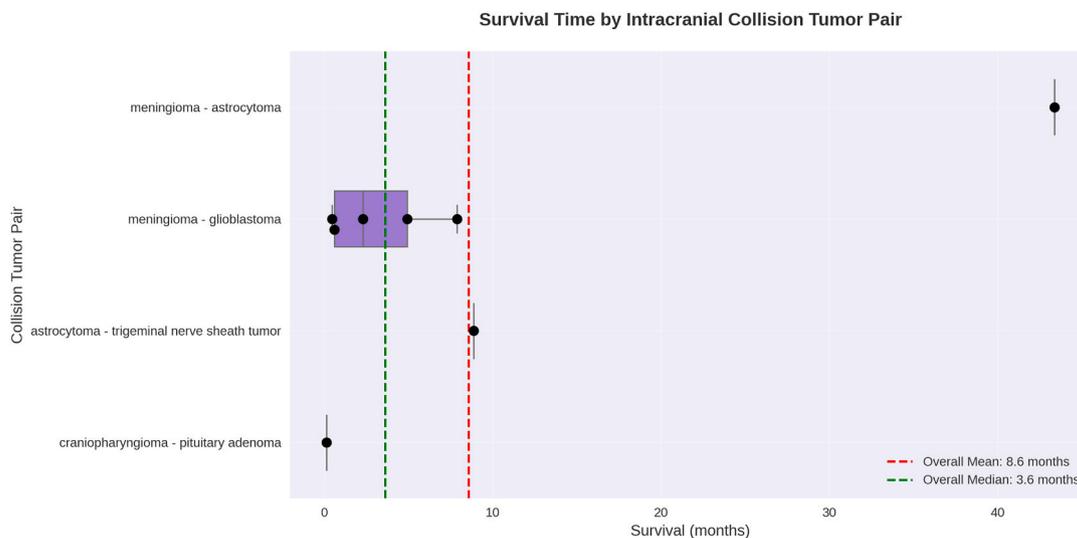


Fig. 6. "Survival time in relation to intracranial collision tumour pair".

%), while regarding location, the sellar region was the most common site (34.3 %), followed by the two frontal lobes (31.3 %). Preoperative suspicion of both lesions was low (26.9 %), and detection rates appeared to vary by anatomical location (p = 0.0095), although this finding should be interpreted cautiously given the exploratory nature of the analysis. More specifically, CPA region tumours had the highest detection rate (71.4 %), and sellar tumours had the lowest (4.3 %). Surgical resection was frequently combined with radiotherapy (63.6 %) and chemotherapy (45.5 %). Recurrence was noted in 17.9 %, especially in those including high-grade tumours such as glioblastoma. Survival analysis showed a mean survival of 8.6 months, with 11.9 % mortality within the follow-up period. Unlike prior reviews that primarily describe tumour combinations, this study systematically evaluates preoperative diagnostic accuracy and highlights location-dependent detection patterns, providing quantitative insight into why collision tumours are

frequently underrecognized.

4.2. Epidemiologic data

The reported cases of intracranial collision tumours in the literature remain limited, mainly consisting of isolated case reports or small case series. The predominance of meningiomas in collision tumours has been widely reported, likely due to their slow growth, vascularity, and interaction with adjacent brain tissue (Sobstyl et al., 2023). The high frequency of glioblastoma-meningioma combinations in this study is consistent with prior findings, which have also identified this pairing as the most common among reported cases (Chamberlin et al., 2021; Zhang et al., 2018; Lin et al., 2022; Basil et al., 2011). However, other various pairings have been reported in the literature, underscoring the diverse nature of these tumours and reinforcing the need for a broader

understanding of their incidence and behaviour (Zacharewski et al., 2022).

Most cases of collision tumours occur in middle-aged to elderly patients; a pattern also observed in previous studies (Sobstyl et al., 2023). This study found a slight female predominance (57.6 %), particularly in cases involving meningiomas, which aligns with prior research indicating a higher incidence of meningiomas in women due to hormonal influences (Levitus and Charitou, 2018). No significant geographical variations were noted, which is consistent with previous literature (Lobo et al., 2024).

4.3. Pathogenesis hypotheses

While collision tumours have been described in various locations, including the frontal, temporal, and sellar regions, their exact pathogenesis remains poorly understood (Ashizawa et al., 2021; Atallah et al., 2024). Some studies suggest that their occurrence is purely coincidental due to the relative frequency of certain intracranial tumours (Syed et al., 2018). Other theories, however, propose that specific tumour pairs may have biological interactions in ways that promote their simultaneous development. For example, meningiomas may create a highly vascular microenvironment that facilitates glioblastoma growth, which could explain the high frequency of this pairing (Lobo et al., 2024). Similarly, hormonal and inflammatory factors may contribute to the coexistence of pituitary adenoma and craniopharyngioma, a pairing that was among the most frequently observed in this study (Zhao et al., 2017).

The primary difference between collision tumours and other tumour categories, including TTMs and composite tumours, is the mechanisms of co-development. Collisions tumours develop independently and do not show metastatic seeding or histological transition, in contrast to TTM, where one tumour metastasises to an already present distinct type (Syed et al., 2018). This suggests that while some tumour combinations may form by chance, others might arise due to shared oncogenic pathways, focal microenvironmental changes, or inflammatory and hormonal variables. Despite these hypotheses, definitive molecular links between collision tumours have yet to be established. While some studies suggest the involvement of oncogenic pathways such as WNT/ β -Catenin and P53 mutations, no genetic interactions have been confirmed (Zacharewski et al., 2022).

4.4. Diagnostic challenges

Despite improvements in neuroimaging, current imaging methods often fail to detect both tumours preoperatively, which results in sub-optimal surgical planning and unexpected results. Although MRI is typically the gold standard imaging modality used in intracranial cancers, its ability to differentiate collision tumours is limited. MRI findings commonly demonstrate heterogeneous enhancement, perilesional oedema, and variable contrast uptake, which can be misleading in distinguishing tumour components (Lobo et al., 2024). Prior literature emphasizes that collision tumours often mimic single heterogeneous lesions, resulting in their misdiagnosis as high-grade gliomas, atypical meningiomas, or metastatic tumours (Nedeljkovic et al., 2023; Lobo et al., 2024). In some cases, CT scans show distinct adjacent lesions with different densities, but in others, the distinction is unclear, further complicating accurate diagnosis (Zhao et al., 2017). Advanced imaging techniques such as MR spectroscopy and perfusion MRI have been employed to differentiate between tumour components based on metabolic activity, but no definitive radiological markers have been established (Sobstyl et al., 2023). However, with a sensitivity of only 26.9 %, the current study reiterates the low preoperative detection rate, further highlighting the need for updated imaging protocols. Especially in locations such as the pituitary fossa, which had the lowest detection rate in this review (4.3 %), features suggestive of collision tumours need to be identified and employed in the preoperative imaging workup of these patients.

From a clinical standpoint, certain imaging features, such as sharply different enhancement patterns, inconsistent diffusion characteristics, or disparity between solid and cystic components that does not match with known tumour behavior, should raise suspicions for potential dual pathology. Early multidisciplinary review involving neuroradiology, skull-base specialists, and neuro-oncology may enhance preoperative suspicion and surgical planning in unclear cases.

4.5. Treatment and prognosis

The surgical approach chosen based on the lesion location, features, and the patient's characteristics remains personalized. Frequently, adjuvant therapies including radiotherapy and chemotherapy are used in the postoperative period, which are primarily dependent on the most aggressive tumour component (Zhang et al., 2018; Sobstyl et al., 2023). More complex cases require staged procedures or combined approaches, such as transsphenoidal surgery followed by craniotomy, reinforcing the need for individualized surgical planning (Sobstyl et al., 2023).

The prognosis of collision tumours remains primarily dependent on the most aggressive tumour component. In cases involving glioblastoma, survival outcomes remain poor despite multimodal treatment, whereas benign tumour pairings such as pituitary adenoma and craniopharyngioma generally have better long-term prognoses (Sobstyl et al., 2023).

4.6. Limitations and future directions

This review has several important limitations. The included cases span more than five decades (1971–2024), resulting in significant temporal heterogeneity in imaging quality, diagnostic criteria, and neurosurgical practice. Earlier cases were diagnosed using CT or early-generation MRI, whereas more recent cases underwent advanced multiparametric imaging, limiting comparability across the dataset. Then, the rarity of intracranial collision tumours introduces selection and publication bias, as unusual cases are more likely to be reported. Consequently, the available literature may overrepresent challenging presentations and underestimate scenarios in which collision tumours were either suspected preoperatively or did not significantly alter management. In addition, incomplete radiological descriptions and limited follow-up data cause reporting bias. Relatively short follow-up durations across published cases, with a mean follow-up of approximately 13 months, preclude robust conclusions regarding long-term prognosis. The low preoperative detection rate should therefore be interpreted cautiously, as it may reflect low pretest probability and diagnostic anchoring, rather than imaging inadequacy alone. Finally, statistical subgroup analyses were exploratory and conducted without correction for multiple comparisons.

Despite these limitations, this review shows how important it is to improve recognition of imaging features that suggest dual pathology and to have a structured multidisciplinary evaluation in cases where the diagnosis is not clear. Early multidisciplinary review with neuroradiologists, neurosurgeons, and neuro-oncologists may help with preoperative recognition and surgical planning. Looking forward, radiomics and artificial intelligence used with multiparametric MRI are promising tools for improving the accuracy of diagnosis in the future. These can quantify subtle heterogeneities in texture, intensity, and perfusion patterns beyond visual assessment. Machine-learning models trained to detect different radiomic signs within a single lesion, integrating diffusion, perfusion, and spectroscopy, may enable earlier suspicion of dual pathology. Although currently theoretical, these approaches represent realistic near-future.

5. Conclusion

To conclude, collision tumours pose challenges to both diagnosis and treatment because of their rarity and complex radiological appearance.

This up-to-date review highlights that, despite advances in neuroimaging, preoperative diagnosis of collision tumours remains difficult, as imaging findings often resemble a single heterogeneous lesion. Histopathological confirmation after surgical removal of collision tumours remains the gold standard for a definitive diagnosis. Future advances in imaging analysis and continued research into molecular carcinogenesis may help support improved diagnostic accuracy and clinical management.

Funding sources

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

Declaration of interests

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.

Acknowledgments

The authors declare that there are no acknowledgements to disclose.

Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.bas.2026.105943>.

References

- Aromataris, E., Lockwood, C., Porritt, K., Pilla, B., Jordan, Z. (Eds.), 2024. JBI Manual for Evidence Synthesis. JBI Manual for Evidence Synthesis [Internet]. <https://synthesismanual.jbi.global>.
- Asada, H., Otani, M., Furuhashi, S., Inoue, H., Toya, S., Ogawa, Y., 1990. Mixed pituitary adenoma and gangliocytoma associated with acromegaly—case report. *Neurol. Med.-Chir.* 30 (8), 628–632.
- Ashizawa, K., Ogura, K., Nagase, S., Sakaguchi, A., Tokugawa, J., Hishii, M., et al., 2021. A collision tumour of solitary fibrous tumour/hemangiopericytoma and meningioma: a case report with literature review. *Pathol. Int.* 71 (10), 697–706.
- Atallah, O., Almealawy, Y.F., Awuah, W.A., Conti, A., Chaurasia, B., 2024. Is simultaneous occurrence of meningioma and glioblastoma a mere coincidence? *Clin. Neurol. Neurosurg.* 236, 108099.
- Ban, V.S., Chaudhary, B.R., Allinson, K., Santarius, T., Kirolos, R.W., 2017. Concomitant primary CNS lymphoma and FSH-pituitary adenoma arising within the sella. *Entirely Coincidental Neurosurg.* 80 (1), E170–E175.
- Basil, I., Ru, K., Pu, C., Silverman, J., Jasnosc, K., 2011. A collision tumour: primary central nervous system B-Cell lymphoma and anaplastic astrocytoma. *Lab. Med.* 42 (6), 324–328. <https://doi.org/10.1309/LMR93DQ1LYHOJUQB> [Internet].
- Binello, E., Bederson, J.B., Kleinman, G.M., 2010. Hemangiopericytoma: collision with meningioma and recurrence. *Neurol. Sci.* 31 (5), 625–630.
- Cannavò, S., Curtò, L., Fazio, R., Paterniti, S., Blandino, A., Marafioti, T., et al., 1993. Coexistence of growth hormone-secreting pituitary adenoma and intracranial meningioma: a case report and review of the literature. *J. Endocrinol. Investig.* 16 (9), 703–708.
- Chamberlin, K., Chamberlin, G., Saunders, K., Khagi, S., 2021. Next-generation sequencing reveals novel mutations in a collision tumour of glioblastoma and meningioma. *CNS Oncol.* 10 (2), CNS70.
- Chen, G., Gao, X., Liao, Y., Xu, B., 2010. A glioblastoma adjacent to a meningioma. *Br. J. Neurosurg.* 24 (6), 718–719.
- Dietterle, J., Frydrychowicz, C., Müller, W., Hoffmann, K.T., Jähne, K., Meixensberger, J., 2020. Tumour-to-Tumour metastasis of multiple meningiomas and clear cell renal cell carcinoma metastasis as first clinical appearance of kidney cancer: a case report and analysis. *J. Neurol. Surg. Rep.* 81, e10–e14.
- Drlicek, M., Aichholzer, M., Wurm, G., Bodenteich, A., Fischer, J., 2004. [Collisiontumour composed of glioblastoma and meningioma—a case report]. *Pathologie* 25 (5), 402–405.
- Finzi, G., Cerati, M., Marando, A., Zoia, C., Ferrelli, F., Tomei, G., et al., 2014. Mixed pituitary adenoma/craniopharyngioma: clinical, morphological, immunohistochemical and ultrastructural study of a case, review of the literature, and pathogenetic and nosological considerations. *Pituitary* 17 (1), 53–59.
- Frassanito, P., Montano, N., Lauretti, L., Pallini, R., Fernandez, E., Lauriola, L., et al., 2011. Simultaneously occurring tumours within the same cerebello-pontine angle: refining literature definitions and proposal for classification. *Acta Neurochir.* 153 (10), 1989–1993. ; discussion 1993.
- George, B., Kumar, R., Johns, P., Bartlett, R.J.U., O'Brien, D., 2007. Contiguous synchronous occurrence of primary cerebral lymphoma and meningioma. *Br. J. Neurosurg.* 21 (1), 35–38.
- Gezer, E., Cantürk, Z., Selek, A., Çetinarslan, B., Tarkun, İ., Sözen, M., et al., 2020. Cushing's disease due to a pituitary adenoma as a component of collision tumour: a case report and review of the literature. *J. Med. Case Rep.* 14 (1), 59.
- Ghosal, N., Kumaran, S.P., Furtado, S.V., Hegde, A.S., 2012. Mixed schwannoma with meningioma - report on 2 cases of unusual tumour with review of literature. *Clin. Neuropathol.* 31 (5), 374–378.
- Gokden, M., Mrak, R.E., 2009. Pituitary adenoma with craniopharyngioma component. *Hum. Pathol.* 40 (8), 1189–1193 [Internet]. <https://www.sciencedirect.com/science/article/pii/S0046817709000641>.
- Goyal, A., Singh, A.K., Sinha, S., Tatke, M., Singh, D., Gupta, V., 2003. Simultaneous occurrence of meningioma and glioma in brain: report of two cases. *J. Clin. Neurosci. Off. J. Neurosurg. Soc. Australas* 10 (2), 252–254.
- Graffeo, C.S., Perry, A., Copeland, W.R., Giannini, C., Neff, B.A., Driscoll, C.L.W., et al., 2017. Synchronous tumours of the cerebellopontine angle. *World Neurosurg.* 98, 632–643.
- Grauvogel, J., Grauvogel, T.D., Taschner, C., Baumgartner, S., Maier, W., Kaminsky, J., 2010. A rare case of radiologically not distinguishable coexistent meningioma and vestibular schwannoma in the cerebellopontine angle - case report and literature review. *Case Rep. Neurol.* 2 (2), 111–117.
- Hakan, T., Armağan, S., Vardar Aker, F., Çelik, L., 1988. Meningioma and Glioblastoma adjacent in the Brain. *Turk Neurosurg.* 8, 57–60 [Internet]. <https://asosindex.com.tr/index.jsp?modul=articles-page&journal-id=2119&article-id=396222#cite>.
- Harb, Z., Moral, L., 2019. An unusual intracranial collision tumour of anaplastic ependymoma and atypical meningioma. *Am. J. Clin. Pathol.* 152, S53 [Internet]. <https://www.embase.com/search/results?subaction=viewrecord&id=L631018341&from=export>.
- Heng, L.J., Jia, D., Gong, L., Zhang, W., Ma, J., Qu, Y., 2017. Endoscopic endonasal resection of a mixed lesion of gangliocytoma and nonfunctioning pituitary adenoma. *World Neurosurg.* 106, 1050.e1–1050.e6.
- Jaiswal, S., Vij, M., Jaiswal, A.K., Chand, G., Behari, S., Kumarjain, V., 2012. Ossifying pituitary adenoma co-existing with astrocytoma and pituitary adenoma associated with gangliocytoma: two unusual conditions. *Turk Neurosurg.* 22 (1), 127–133.
- Jin, G., Hao, S., Xie, J., Mi, R., Liu, F., 2013. Collision tumours of the sella: coexistence of pituitary adenoma and craniopharyngioma in the sellar region. *World J. Surg. Oncol.* 11 (1), 178. <https://doi.org/10.1186/1477-7819-11-178> [Internet].
- Jukes, A., Allan, R., Rawson, R., Buckland, M.E., 2016. Growth hormone secreting pituitary adenoma with admixed gangliocytoma and ganglioglioma. *J. Clin. Neurosci.* 31, 202–204.
- Karsy, M., Sonnen, J., Couldwell, W.T., 2015. Coincident pituitary adenoma and sellar meningioma. In: *Acta Neurochirurgica. Austria*, 157, pp. 231–233.
- Kikuta, H., Jinguiji, S., Sato, T., Bakhit, M., Hiruta, R., Sato, Y., et al., 2023. A Collision Tumour of Pit-1/SF-1-positive Double Pituitary Adenoma and a Craniopharyngioma Coexisting with Graves' Disease, 10. NMC case report journal, Japan, pp. 169–175.
- Koutourousiou, M., Seretis, A., Kontogeorgos, G., 2009. Intra-sellar schwannoma co-existing with GH-secreting pituitary adenoma. *Acta Neurochir.* 151 (12), 1693–1697.
- Kurdi, M., Al-Ardati, H., Baessa, S.S., 2014. Malignant trigeminal nerve sheath tumour and anaplastic astrocytoma collision tumour with high proliferative activity and tumour suppressor p53 expression. *Case Rep. Pathol.* 2014, 153197.
- Kutz, J.W., Barnett, S.L., Hatanpaa, K.J., Mendelsohn, D.B., 2009. Concurrent vestibular schwannoma and meningioma mimicking a single cerebellopontine angle tumour. *Skull Base* 19 (6), 443–446.
- Levitus, C., Charitou, M., 2018. A case of an incidental collision tumour of the sella. *Endocr. Rev.* 39 (2) [Internet]. <https://www.embase.com/search/results?subaction=viewrecord&id=L623114220&from=export>.
- Lin, M.S., Lee, C.H., Chen, S.Y., Shen, C.C., 2022. Collision brain tumour with atypical meningioma and glioblastoma: case report. *Int. J. Surg. Case Rep.* 94, 107137.
- Lobo, G.G., Alves, J.L., Novo, A.M.F., Pereira, D.J., 2024. Collision tumours: a meningioma and not oedema, but an oligodendroglioma. *Egypt J. Radiol. Nucl. Med.* 55 (1), 29. <https://doi.org/10.1186/s43055-024-01211-4> [Internet].
- Lüdemann, W., Stan, A.C., Tatagiba, M., Samii, M., 2000. Sporadic unilateral vestibular schwannoma with islets of meningioma: case report. *Neurosurgery* 47 (2), 451–454.
- Maiuri, F., Cappabianca, P., Iaconetta, G., Esposito, F., Messina, A., 2005. Simultaneous presentation of meningiomas with other intracranial tumours. *Br. J. Neurosurg.* 19 (4), 368–375.
- Marra, A., Ramponi, G., Grimaldi, G., 1977. Simultaneous occurrence of right supratentorial meningioma and glioblastoma multiforme. Case report. *Acta Neurochir.* 36 (1–2), 83–91.
- Martin, S.E., Khalidi, H.S., Hattab, E.M., 2013. Marginal zone B-cell lymphoma involving a longstanding fibrous meningioma: an initial manifestation of systemic disease. *Hum. Pathol.* 44 (11), 2609–2613.
- Morita, K., Nakamura, F., Kamikubo, Y., Mizuno, N., Miyauchi, M., Yamamoto, G., et al., 2012. Pituitary lymphoma developing within pituitary adenoma. *Int. J. Hematol.* 95 (6), 721–724.
- Nedeljkovic, A., Ilic, R., Nedeljkovic, Z., Milicevic, M., Raicevic, S., Grujicic, D., 2023. A unique case of intracranial collision tumour composed of ganglioglioma WHO gr I and supratentorial ependymoma WHO gr III: case-based literature review. *Child's Nerv. Syst. ChNS Off. J. Int. Soc. Pediatr. Neurosurg.* 39 (9), 2407–2411.
- Nestler, U., Schmidinger, A., Schulz, C., Huegens-Penzel, M., Gamerding, U.A., Koehler, A., et al., 2007. Glioblastoma simultaneously present with meningioma—report of three cases. *Zentralbl. Neurochir.* 68 (3), 145–150.
- Page, M.J., McKenzie, J.E., Bossuyt, P.M., Boutron, I., Hoffmann, T.C., Mulrow, C.D., et al., 2021. The PRISMA 2020 statement: an updated guideline for reporting

- systematic reviews. *BMJ* 372 [Internet]. <https://www.bmj.com/content/372/bmj.n71>.
- Prabhakar, V., Rao, B.D., Subramanyam, M.V., 1971. Pituitary adenoma associated with craniopharyngioma. *J. Pathol.* 103 (3), 185–187.
- Prayson, R.A., Chowdhary, S., Woodhouse, S., Hanson, M., Nair, S., 2002. Collision of a syncytial meningioma and malignant astrocytoma. *Ann. Diagn. Pathol.* 6 (1), 44–48.
- Riccioni, L., Morigi, F., Cremonini, A.M., 2007. Follicular lymphoma of the dura associated with meningioma: a case report and review of the literature. *Neuropathology* 27 (3), 278–283.
- Rivera, J., Alves, S., Bianchi, C.C., Al-Mutawa, N., Guiot, M.C., Zeitouni, A., 2010. An unusual collision tumour comprising a prolactinoma and a plasmocytoma originating from the sellar region. *Pituitary* 13 (2), 189–193.
- Ruiz, J., Capilla, E., Díaz, J.F., Ruiz, J.A., Andrade, J., Hernández, T., et al., 2015. Secretory meningioma with KLF4 K409Q mutation in collision with glioma. *Clin. Neuropathol.* 34 (6), 322–329.
- Sahli, R., Christ, E., Kuhlen, D., Giger, O., Vajtai, I., 2011. Sellar collision tumour involving pituitary gonadotroph adenoma and chondroma: a potential clinical diagnosis. *Pituitary* 14 (4), 405–408.
- Sargis, R.M., Wollmann, R.L., Pytel, P., 2009. A 59 year-old man with sellar lesion. *Brain Pathol. (Zurich, Switzerland)*. Switzerland 19, 161–162.
- Shahmohammadi, M.R., Javadian, H., Asgari, A., Khoshnoud, R.J., 2016. Glioblastoma simultaneously present with adjacent meningioma: case report. *Int. Clin. Neurosci. J.* 3 (1), 54–58 [Internet]. <https://journals.sbm.ac.ir/neuroscience/article/view/13193>.
- Shakally, A., Seaquist, E.R., Araki, T., 2018. A case of craniopharyngioma following resection of nonfunctioning pituitary macroadenoma. *Endocr. Rev.* 39 (2) [Internet]. <https://www.embase.com/search/results?subaction=viewrecord&id=L623113980&from=export>.
- Sobstyl, M., Nagańska, E., Glinka, P., Wierzb-Bobrowicz, T., Aciewicz, A., Kuls-Ozmaniec, A., 2023. Large Haemorrhage Within Glioblastoma Mimicking Haemorrhagic Stroke and Coexistence of Meningioma: a Case of Collision Tumours, 61. *Folia neuropathologica, Poland*, pp. 433–441.
- Suck, M.L.T., Avellán, Á.M., Escudero, N.N., Cañas, S.V., Ramírez, S.G., Lara, C.S., 2008. Frontal solitary plasmacytoma and meningioma (collision tumour). Case report and literature review. *Patol. Rev. Latinoam.* 46 (2), 112–116.
- Suzuki, K., Momota, H., Tonooka, A., Noguchi, H., Yamamoto, K., Wanibuchi, M., et al., 2010. Glioblastoma simultaneously present with adjacent meningioma: case report and review of the literature. *J. Neuro Oncol.* 99 (1), 147–153.
- Syed, S., Karambizi, D.I., Baker, A., Groh, D.M., Toms, S.A., 2018. A comparative report on intracranial tumour-to-tumour metastasis and collision tumours. *World Neurosurg.* 116, 454, 463.e2.
- Takeuchi, Y., Kanamori, M., Kumabe, T., Saito, R., Sonoda, Y., Watanabe, M., et al., 2009. Collision tumour of anaplastic oligodendroglioma and gangliocytoma: a case report. *Brain Tumour Pathol.* 26 (2), 89–93.
- Tanriover, N., Aydin, O., Kucukyuruk, B., Abuzayed, B., Guler, H., Oz, B., et al., 2014. Endoscopic approach to a collision tumour of growth hormone-secreting adenoma and gangliocytoma in the pituitary gland. *J. Craniofac. Surg.* 25 (4), 1277–1279.
- Truong, V.T., Tran, D.D.T., Dang, C.T., 2019. Collision occurrence of meningioma and Astrocytoma: a case report and literature review. *Asian J. Neurosurg.* 14 (3), 938–942.
- Tugcu, B., Kepoglu, U., Gunal, M., Gunaldi, O., Karakaya, B., Demirgil, B.T., 2006. Two distinct primary brain tumours, in same region of the same patient: a case report. *J. Neuro Oncol.* 79 (2), 219–220. <https://doi.org/10.1007/s11060-006-9130-4> [Internet].
- Vaquero, J., Coca, S., Martínez, R., Jiménez, C., 1990. Convexity meningioma and glioblastoma in collision. *Surg. Neurol.* 33 (2), 139–141.
- Verma, S.K., Kumar, S., Deb, P., Yadav, K.K., 2015. Rare Case of Radiologically Distinct but Pathologically Admixed Vestibular Schwannoma and Meningioma in the Cerebellopontine Angle: a Case Report, 11. *Journal of cancer research and therapeutics, India*, p. 1029.
- Yaghmour, W., Kurdi, M.E., Baeesa, S.S., 2016. De novo glioblastoma in the territory of a recent middle cerebral artery infarction and a residual meningioma: pathogenesis revisited. *World J. Surg. Oncol.* 14, 112.
- Yan, X., Marsh, W.R., Scheithauer, B.W., 2011. Dysembryoplastic neuroepithelial tumour and calcifying pseudoneoplasms of the neuraxis: a collision of two seizure-associated lesions. *Clin. Neuropathol.* 30 (4), 197–202.
- Yan, J., Liu, W., Wang, X., Zhao, S., Gao, L., Ye, Y., et al., 2018. Primary central nervous system extranodal natural Killer/T-Cell lymphoma, nasal type colliding with meningioma. *World Neurosurg.* 120, 17–26.
- Yao, K., Wang, H., Duan, Z., Bian, Y., Xia, L., Ma, Z., et al., 2014. Mixed granular cell astrocytoma and fibrosarcoma of the brain: a case report. *Int. J. Clin. Exp. Pathol.* 7 (7), 4473–4478.
- Yoshida, A., Sen, C., Asa, S.L., Rosenblum, M.K., 2008. Composite pituitary adenoma and craniopharyngioma?: an unusual sellar neoplasm with divergent differentiation. *Am. J. Surg. Pathol.* 32 (11), 1736–1741.
- Zacharewski, N., Movahed-Ezazi, M., Song, X., Mehta, T., Manjila, S., 2022. De Novo Glioblastoma Masqueraded within a Hemispheric Dural Meningiomatosis: rare Imaging Findings and Rationale for Two-Stage Resection. *J. Neurol. Surg. Rep.* 83 (2), e44–e49.
- Zhang, Z., Yang, Y., Zhang, K., Zhuang, J., Shao, F., Liu, H., et al., 2018. Collision tumour of glioblastoma and meningioma: case report and literature review. *World Neurosurg.* 117, 137–141.
- Zhao, Y., Zhang, H., Lian, W., Xing, B., Feng, M., Liu, X., et al., 2017. Collision tumours composed of meningioma and growth hormone-secreting pituitary adenoma in the sellar region: case reports and a literature review. *Medicine (Baltim.)* 96 (50), e9139.