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Review Article

Characteristics of cranial vault lymphoma from a systematic review of the literature

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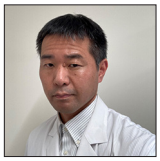

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ABSTRACT

Background: Cranial vault lymphomas are rare and their clinical features are often similar to those of cranial vault meningiomas. The objective of this review was to identify the features helpful for differentiating lymphomas of the cranial vault, from meningiomas which were the most common diagnosis before the definitive pathological diagnosis.

Methods: The inclusion criterion was a histologically proven malignant lymphoma initially appearing in the calvarium. We conducted a literature search of the electronic PubMed and Ichushi-Web databases up to June 1, 2020. Cranial vault lymphoma that was diagnosed after an original diagnosis of lymphoma in a nodal or soft-tissue site was excluded from the study. Descriptive analyses were used to present the patient characteristics.

Results: A total of 111 patients were found in 98 eligible articles. Almost all studies were case reports. The most common symptom was a growing subcutaneous scalp mass (84%) present for a mean duration of 5.9 months before the patient presented for treatment in analyzable cases; this fast growth may distinguish lymphomas from meningiomas. The tumor vascularization was often inconspicuous or poor, unlike well-vascularized meningiomas. A disproportionately small amount of skull destruction compared with the soft-tissue mass was observed in two-thirds of the analyzable cases.

Conclusion: This qualitative systematic review identified several features of cranial vault lymphomas that may be useful in differentiating them from meningiomas, including a rapidly growing subcutaneous scalp mass, poor vascularization, and limited skull destruction relative to the size of the soft-tissue mass.

Keywords: Calvarial lymphoma, Calvarium, Lymphosarcoma, Reticulum cell sarcoma, Skull

INTRODUCTION

Malignant lymphoma of the bone is uncommon and, hence, presents diagnostic and therapeutic problems.[71] Cranial vault involvement has been reported to account for 5.5% of bone lymphoma cases and the number of reported cases of lymphoma initially appearing in the calvarium is limited.[71] The diagnosis of this rare cranial vault tumor before pathological diagnosis has usually been not lymphoma but meningioma, metastatic tumor, or other mesenchymal tumors, which has tended to lead to radical operative treatments like gross total removal of the tumor.[1,25,96] If we can make a differential diagnosis of cranial vault lymphoma preoperatively, we may choose less radical procedures like biopsy and can start chemotherapy at an earlier time. This article presents

a systematic review of the literature to show demographic, clinical, and imaging characteristics, as well as treatments and outcomes of cranial vault lymphoma. We specify characteristic features of cranial vault lymphoma that may be helpful for their diagnosis, especially to distinguish them from meningiomas, which have been the most common diagnosis of cranial vault lymphomas before definitive pathological diagnosis.[1,2,4,13,35,36,37,50,54,65,66,70,75,84,86,88,96,105]

MATERIALS AND METHODS

Eligibility criteria

The inclusion criterion was a histologically proven malignant lymphoma initially appearing in the calvarium. Cranial vault lymphoma that was diagnosed after an original diagnosis of lymphoma in a nodal or soft-tissue site was excluded from the study. Skull base lymphoma and dural lymphoma were also excluded from the study. Articles whose full text was unable to be located were excluded from the study. We excluded systematic and retrospective review articles and case series articles that did not include case-specific data.

Information sources and search strategy

We conducted a literature search of the electronic PubMed and Ichushi-Web databases up to June 1, 2020, using the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. We extracted all human reports on cranial vault lymphomas, using the following terms: cranial vault lymphoma; skull lymphoma; calvarial lymphoma; transcalvarial lymphoma; dural lymphoma; and combinations of the variables of lymphoma, lymphosarcoma (an obsolete classification of non-Hodgkin lymphoma), reticulum cell sarcoma (another obsolete classification of non-Hodgkin lymphoma), cranial, vault, skull, and calvarium. The search resulted in 1492 PubMed citations and 391 Ichushi-Web citations, 113 of which were duplicated, resulting in a total of 1770 articles [Figure 1].

Selection process

Two reviewers (N.N. and T.F.) independently and in duplicate screened, reviewed, and discussed all the selected articles. The full text of all eligible articles was reviewed, and their data extracted and collated. In cases where questions regarding the inclusion of certain articles arose, this was discussed with a third reviewer, K.N.

Data collection process and data items

Data of the eligible works were obtained through careful analysis of the full text by two authors (N.N. and T.F) independently. The senior author, K.N., was available in case of a split decision. Questions arising as to pathological diagnosis were discussed with a pathologist, S.M. We analyzed the clinical and radiological characteristics of the patients, as well as their treatments and survival in these published studies. Specifically, we extracted the following items: age of the patient; sex; clinical symptoms; location of tumors; findings of palpation; skin condition; speed of growth; existence of other lesions; types of treatment; extent of resection; duration of follow-up and outcomes; laboratory data; imaging data of skull X-ray, angiogram, computed tomography (CT), magnetic resonance imaging (MRI), and others; bone images on CT; extra- and intracranial tumor extension on CT and MRI; dural tail and brain invasion on MRI; and histopathological types. Due to the heterogeneity of patient descriptions, some clinical and imaging features were not explicitly reported for each patient. We extracted and reported only unambiguously described data. Data on clinical and imaging features were also extracted from the patient imaging data. Evaluation of publication bias was not feasible because of heterogeneity and because most of the included studies were case reports and case series.

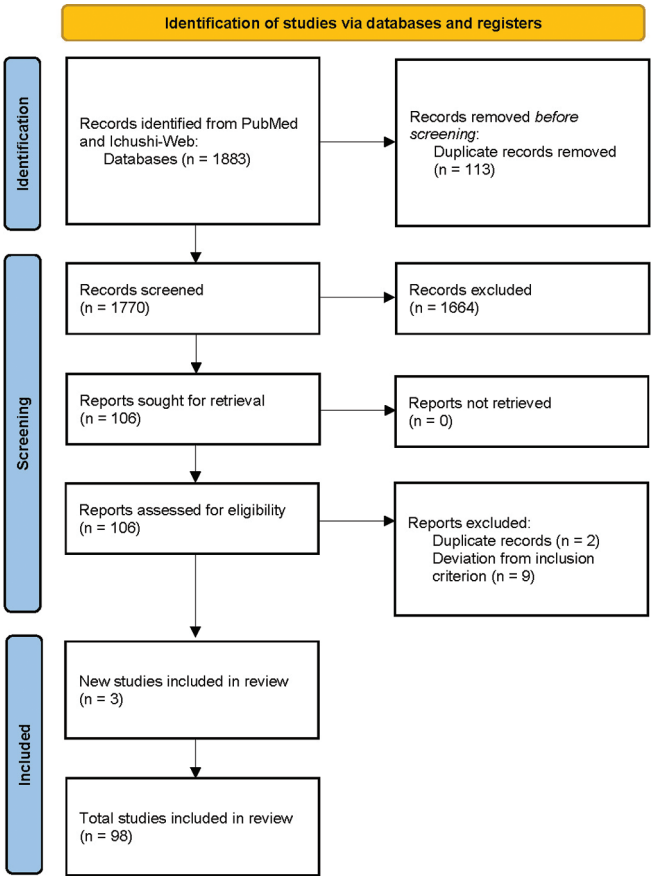

Figure 1: The PRISMA flowchart of our systematic review. New studies were those identified from the reference lists of articles identified in the initial screen.

Statistical analysis

Descriptive analyses were used to present the patient characteristics. Continuous variables were expressed as mean ± standard deviation, and categorical variables were expressed as number and percentage. Because of the limited follow-up data included in each intervention and the lack of standardization of assays and treatments across the many laboratories included in the present review, we were unable to statistically compare the findings on images and the changes in clinical outcomes. All calculations were performed with JMP 13.2.1 (SAS Institute, Inc., Cary, North Carolina, U.S.).

RESULTS

From among the articles found, we selected all studies reporting patients with cranial vault lymphoma (n = 106) without limitation of language and identified additional studies from the reference lists of the articles (n = 3). After discarding duplicate references and publications (n = 2), as well as excluding reports of secondary cranial vault lymphoma, skull base lymphoma, or dural lymphoma (n = 9), we settled on 98 articles for careful review. The number of articles retained at each stage of data acquisition is shown in a PRISMA flowchart [Figure 1].

We found 111 cases of histologically proven malignant lymphomas initially appearing in the cranial vault in the 98 articles and analyzed the data of 111 patients [Tables 1, S1, and S2].[1-11,13-16,19-25,27-56,58-70,72-76,78,80,83-89,91-109]

The average patient age was 52 ± 20 years (range, 3–85 years) [Table 1]. The male-to-female ratio was 1.09:1. Common symptoms were a growing subcutaneous mass on the scalp (84%), headache (33%), focal neurological deficit (25%), and seizure (6%) in the 110 cases with available data. The parietal (57%), frontal (54%), occipital (21%), and temporal (15%) bones were the affected sites in 110 cases with analyzable data. The subcutaneous scalp mass was firm (57%), soft (16%), nontender (56%), tender (22%), or accompanied by reddish skin or ulcer (7%) in 68 cases with a description of the scalp mass. In the 49 cases with a description of mass growth, the mean duration of growth was 5.9 ± 6.3 months before the patient presented for treatment. In 23% of 93 cases with available data, lesions outside the cranial vault were present.

Laboratory data were described in 59 cases, with human immunodeficiency virus positivity in 5 cases (8%), elevated lactate dehydrogenase in 6 cases (10%), anemia in 4 cases (7%), increased white blood cell count in 3 cases (5%), elevated erythrocyte sedimentation rate in 2 cases (3%), elevated alkaline phosphatase in 1 case (2%), increased soluble interleukin-2 receptor (sIL2R) in 1 case (2%), and decreased platelets in 1 case (2%) [Table S1]. Findings on plain skull X-ray were described in 38 cases, including osteolytic changes at the tumor site in 29 cases (76%), no change in 6 cases (16%), hyperostosis in 2 cases (5%), and periosteal reaction in 4 cases (11%). A sharp margin of the skull lesion was observed in 6 cases (16%) and an indistinct or irregular margin of the skull lesion in 15 cases (40%). In 12 cases with description of tumor vascularization on angiogram, no or poor tumor vascularization was found in 7 cases (58%). If any vascularization was present, it was mainly derived from the external carotid artery circulation. Among 32 cases with analyzable CT data, the tumor was hyperdense in 25 cases (78%), isodense in 5 cases (16%), and hypodense in 2 cases (6%). Among 42 cases with contrast-enhanced (CE) CT data, the tumor was enhanced well in 37 cases (88%) and slightly in 5 cases (12%). Most tumors were enhanced diffusely, either homogeneously or heterogeneously, whereas some extracranial components showed peripheral enhancement. On MRI, T1-weighted imaging (T1WI) with analyzable data (n = 38) showed a hyper- and isointense tumor in 1 case (3%), isointense tumor in 11 cases (29%), iso- and hypointense tumor in 4 cases (11%), and hypointense tumor in 22 cases (58%). T2-weighted imaging (T2WI) (n = 40) showed a hyperintense tumor in 21 cases (53%), hyper- and isointense tumor in 1 case (3%), isointense tumor in 12 cases (30%), iso- and hypointense tumor in 5 cases (13%), and hypointense tumor in 1 case (3%). CE-T1WI (n = 48) showed an enhancing tumor in 48 cases (100%). The tumors tended to show uptake on ^{18}F-fluorodeoxyglucose positron emission tomography (FDG-PET) and on bone scintigraphy and gallium scintigraphy, which were also used for the evaluation of lesions outside the cranial vault.

Extra- and intracranial extensions of the tumor were evaluated by CT, MRI, or both in 87 cases [Table S2]; the extracranial extension was much larger than the intracranial extension in 35 cases (40%), both were nearly the same size in 35 cases (40%), the intracranial extension was much larger than the extracranial extension in 16 cases (18%), and there was neither intra- nor extracranial extension in 1 case (1%). Bone changes of the cranial vault due to the tumor were evaluated by CT, MRI, or both in 84 cases. The images showed no skull changes and/or preserved skull contour in 11 cases (13%), osteolytic changes in 68 cases (81%), hyperostosis in 4 cases (5%), and only sclerosis at the lesion in 1 case (1%). In the 68 cases with osteolytic skull changes due to the tumor, the skull was penetrated or dissolved to less than half of the thickness in 26 cases (38%), whereas permeative dissolution with relatively preserved skull contour was observed in 35 cases (51%). Periosteal bone formation was observed in 10 cases (15%). In seven cases, there was no detailed description. A disproportionately small area of cortical destruction of the cranial vault relative to the volume of the extra- or intracranial soft-tissue mass, which we defined as cortical destruction less than one-fifth of the soft-tissue mass in diameter, was observed in 50 cases (67%) on CT, MRI, or

Table 1: Clinical features, treatment, and outcome of 111 patients with cranial vault lymphoma.

Author year (reference)	Age (years) /sex	Symptoms at onset	Lesion location	Properties of mass	Other lesions	Surgery	Adjuvant therapies	Outcomes (follow-up)
Wichtl,[106] 1949	66/F	Subcutaneous mass on the scalp	Occipital	NA	NA	Biopsy	R	Died* (55 mo)
Strange and De Lorimier,[91] 1954	50/F	Subcutaneous mass on the scalp	Frontal	Soft, growing for 6 mo, 8 cm	NA	Biopsy	R	Alive (7 y)
	47/F	Subcutaneous mass on the scalp, neurological deficit	Frontal	Soft, nontender, growing for 6 mo	NA	Biopsy	R	Alive (1 y)
	66/F	Subcutaneous mass on the scalp	Parietotemporal	Firm, nontender, growing for 8 mo, 6 cm	Preauricular LNs	Biopsy	R	Alive (10 mo)
Ullrich and Bucy,[101] 1958	53/F	Subcutaneous mass on the scalp, headache	Frontal, occipital	Soft, tender, 9 cm	None	Gross total removal	R	Alive (5.5 y)
Piendak and Alder,[80] 1959	33/M	Subcutaneous mass on the scalp, diplopia, ophthalmalgia	Frontal	Soft	None	Subtotal removal	R	Alive (9 y)
Block and Peck,[9] 1964	68/M	Subcutaneous mass on the scalp	Frontal	Firm, nontender, nonreddish, growing for 1 mo, 5 cm	Axillary LNs	Biopsy	R	NA
Topolnicki and White,[99] 1969	67/F	Subcutaneous mass on the scalp,	Frontoparietal	Soft, nontender, 12 cm	NA	Biopsy	R	Alive (4 y)
Wainwright,[104] 1973	77/M	Subcutaneous mass on the scalp, ulceration	Fronto-parietal	Ulcerated	None	Biopsy	R	Alive (22 mo)
Gawish,[27] 1976	8/M	Subcutaneous mass on the scalp	Frontal	Soft, nontender, growing for 5 mo, 17 cm	Anterior cervical LNs	Subtotal removal	C	Alive
Agbi *et al.*,[1] 1983	58/F	Subcutaneous mass on the scalp, neurological deficit, seizure, headache	Parietotemporal	Firm	None	Gross total removal	R	Alive (7 mo)
Holtås *et al.*,[30] 1985	60/F	Subcutaneous mass on the scalp	Frontal	Firm, nontender, 6 cm	None	Biopsy	Steroid	Alive (6 mo)
	20/M	Seizure	Frontal	NA	None	Surgery (NA)	R+C	Alive (5 mo)

(Contd...)

Table 1: (*Continued*).

Author year (reference)	Age (years) /sex	Symptoms at onset	Lesion location	Properties of mass	Other lesions	Surgery	Adjuvant therapies	Outcomes (follow-up)
Kinjo and Satoh,[45] 1985	23/M	Subcutaneous mass on the scalp, neurological deficit, headache, vomiting,	Frontal and occipital	Soft, 13 cm	Multifocal bone lesions	Subtotal removal	R	Died (5 mo)
Thomas and Kennedy,[97] 1986	28/F	Subcutaneous mass on the scalp	Occipital	Firm, tender, 10 cm	Supraclavicular LNs	Biopsy	R	Alive (14 mo)
Howat *et al.*,[32] 1987	3/M	NA	Frontal	NA	None	NA	R+C	Alive (13 y)
Maiuri *et al.*,[56] 1987	51/F	Neurological deficit, headache	Parieto-occipital	NA	None	Gross total removal	R	Alive (2 y)
Tagawa *et al.*,[94] 1987	65/F	Subcutaneous mass on the scalp	Frontal	Growing for 3 mo, 9 cm	None	Biopsy	R+C	Alive (1 y)
Kawakami *et al.*,[42] 1988	52/F	Subcutaneous mass on the scalp	Parietal	Firm, 5 cm	None	Gross total removal	R	Alive (6 y)
Morgello *et al.*,[63] 1989	56/M	Subcutaneous mass on the scalp	Occipital	Growing for 5 mo	NA	Biopsy	R+C	Died (20 mo)
Herkes *et al.*,[29] 1991	73/F	Seizure	Frontal	Firm, nontender, 5 cm	Retroperitoneal LNs, multifocal bone lesions	Biopsy	R+C	Alive (3 mo)
	55/M	Neurological deficit	Parietal	Ulcerated	None	Biopsy	R	Died (3 mo)
Kumon *et al.*,[49] 1991	74/F	Subcutaneous mass on the scalp	Frontal	Firm, nontender, 6 cm	None	Subtotal removal	C	Alive (8 mo)
Natsuda *et al.*,[68] 1991	41/F	Subcutaneous mass on the scalp	Temporal	Ulcerated, growing for 4 mo, 15 cm	None	Biopsy	R+C	Alive (6 mo)
Lonjon *et al.*,[53] 1993	71/F	Subcutaneous mass on the scalp	Occipital	Growing for 3 mo, 11 cm	None	Subtotal removal	C	Alive (6 mo)
Parekh *et al.*,[75] 1993	65/F	Neurological deficit, headache	Parietal	Firm, nontender, 3 cm	None	Gross total removal	R	Died* (6 y)
Sato *et al.*,[89] 1993	65/M	Subcutaneous mass on the scalp, neurological deficit	Parietal	Firm, nontender, growing for 1 mo, 13 cm	None	Subtotal removal	R+C	Alive (20 mo)
Kelleher *et al.*,[43] 1994	30/F	Headache	Frontotemporal	Tender	None	Biopsy	C	Alive (27 mo)
Loembe *et al.*,[52] 1994	32/M	Subcutaneous mass on the scalp	Frontoparietal	Firm, nontender, growing for 6 mo	None	Subtotal removal	Refused	Died* (6 wk)

(Contd...)

Table 1: (*Continued*).

Author year (reference)	Age (years) /sex	Symptoms at onset	Lesion location	Properties of mass	Other lesions	Surgery	Adjuvant therapies	Outcomes (follow-up)
Morioka *et al.*,[64] 1994	69/M	Subcutaneous mass on the scalp	Frontal	Firm, nontender, growing for 1 mo	None	Biopsy	R+C	Died (18 mo)
Vigushin *et al.*,[103] 1994	68/M	Neurological deficit	Parieto-temporal	NA	None	Partial removal	C	Alive (2 y)
Wittram *et al.*,[107] 1994	31/M	Subcutaneous mass on the scalp, seizure	Parietal	Soft, 8 cm	LNs in the abdomen	Removal	NA	NA
Aslan *et al.*,[6] 1995	12/M	Subcutaneous mass on the scalp, neurological deficit, neck pain	Occipital	Firm, nontender, growing for 0.5 mo, 8 cm	None	Biopsy	R+C	Alive (2 mo)
Landys *et al.*,[50] 1995	62/M	Neurological deficit, malaise, abdominal pain	Fronto-parietal	Firm, nontender, 16 cm	Retroperitoneal lesion, spleen	Biopsy	C	Alive (5 y)
Paige and Bernstein,[72] 1995	51/M	Subcutaneous mass on the scalp, headache	Parieto-occipital	Firm, growing for 3 mo, 10 cm	None	Biopsy	R+C	NA
	78/M	Subcutaneous mass on the scalp	Temporal	Nontender, growing for 12 mo, 5 cm	None	Removal	R+C	NA
Isla *et al.*,[34] 1996	75/F	Seizure	Frontal	NA	None	Removal	R+C	Alive (3 y)
Bhatia *et al.*,[7] 1997	50/M	Headache	Parietal	Firm, tender, 3 cm	None	Biopsy	R+C	Died (7 mo)
Curty *et al.*,[14] 1997	19/F	Subcutaneous mass on the scalp, neurological deficit, headache, vomiting	Parietal	NA	Multifocal bone lesions	Gross total removal	C	NA
Muin *et al.*,[65] 1997	60/M	Neurological deficit, headache	Parietal	NA	None	Gross total removal	R+C	Alive (8 mo)
Jamjoom *et al.*,[37] 1998	25/M	Subcutaneous mass on the scalp	Parietal	Firm, nontender, growing for 12 mo, 5 cm	None	Gross total removal	R	Alive (5 mo)
Jiménez Moragas *et al.*,[38] 1999	38/M	Incidentally found	Whole of the cranial vault	NA	Hepatomegaly	Biopsy	None	Died* (5 d)
Dai *et al.*,[16] 2000	21/M	Subcutaneous mass on the scalp	Frontal	Firm, nontender, growing for 4 mo, 5 cm	Preauricular LNs	Gross total removal	R+C	Alive (6 mo)

(Contd...)

Table 1: (*Continued*).

Author year (reference)	Age (years) /sex	Symptoms at onset	Lesion location	Properties of mass	Other lesions	Surgery	Adjuvant therapies	Outcomes (follow-up)
Pardhanani *et al.*,[74] 2000	77/M	Subcutaneous mass on the scalp, neurological deficit	Frontotemporal	Firm	NA	Biopsy	R	Died (3 wk)
Parker *et al.*,[76] 2001	11/M	Subcutaneous mass on the scalp	Frontal	Growing for 1 mo	None	Removal	C	Alive (1 y)
Thurnher *et al.*,[98] 2001	36/M	Subcutaneous mass on the scalp	Frontoparietal	Tender, 8 cm	A lesion in the mouth	Partial removal	R+C	Died (8 mo)
Duyndam *et al.*,[19] 2002	71/F	Subcutaneous mass on the scalp	Frontal	Firm, tender, 7 cm	None	Biopsy	C	Alive (6 mo)
Pernot *et al.*,[78] 2002	40/M	Subcutaneous mass on the scalp, headache	Frontoparietal	NA	None	Gross total removal	R	Alive (1 y)
Kanai *et al.*,[39] 2003	71/F	Subcutaneous mass on the scalp	Frontoparietal	Firm, nontender, growing for 3 mo, 8 cm	None	Subtotal removal	C	Died (3.5 y)
Kantarci *et al.*,[41] 2003	65/M	Subcutaneous mass on the scalp	Fronto-parietal	Firm, nontender, ulcerated, 10 cm	Cervical LNs	Biopsy	NA	NA
Koral *et al.*,[47] 2003	15/M	Subcutaneous mass on the scalp	Temporo-occipital	Growing for 24 mo, 7 cm	None	Biopsy	C	Alive (2.5 y)
Mongia *et al.*,[62] 2003	25/M	Subcutaneous mass on the scalp	Fronto-temporo-parietal	Firm, tender, growing for 6 mo, 15 cm	None	Subtotal removal	R+C	Alive (2.5 y)
Nishimoto *et al.*,[69] 2003	63/M	Subcutaneous mass on the scalp	Frontoparietal	Firm, nontender, growing for 2 mo, 4 cm	None	Biopsy	C	Alive (NA)
Aquilina *et al.*,[5] 2004	72/F	Subcutaneous mass on the scalp, seizure, headache, fatigue	Frontotemporal	Firm, nontender	None	Biopsy	C	Alive (6 mo)
Horstman *et al.*,[31] 2004	80/F	Subcutaneous mass on the scalp	Parieto-occipital	Growing for 6 mo, 18 cm	A neck lesion	Biopsy	C	Died (NA)
Madan *et al.*,[55] 2004	70/F	Subcutaneous mass on the scalp, weight loss	Parieto-occipital	Nontender, reddish, growing for 12 mo, 7 cm	None	NA	C	Alive (NA)
Szucs-Farkas *et al.*,[93] 2005	42/F	Subcutaneous mass on the scalp, malaise, nausea, and headache	Parieto-occipital	NA	None	Biopsy	R	Alive (NA)

(Contd...)

Table 1: (*Continued*).

Author year (reference)	Age (years) /sex	Symptoms at onset	Lesion location	Properties of mass	Other lesions	Surgery	Adjuvant therapies	Outcomes (follow-up)
Tanimura *et al.*,[95] 2005	85/F	Subcutaneous mass on the scalp	Parietal	4 cm	None	Refused (autopsy)	Refused	Died (4 mo)
Evliyaoglu *et al.*,[21] 2006	78/F	Subcutaneous mass on the scalp, neurological deficit, headache	Parietal	Firm, growing for 0.25 mo, 5 cm	None	Subtotal removal	R+C	Alive (4 y)
Galarza *et al.*,[25] 2006	61/M	Headache	Parietal	NA	None	Gross total removal	R+C	Alive (23 mo)
Palled *et al.*,[73] 2006	13/M	Subcutaneous mass on the scalp, proptosis, neck swelling	Frontal	Nontender, growing for 2 mo, 9 cm	Cervical LNs	Biopsy	C	Alive (NA)
Agrawal *et al.*,[2] 2007	43/F	Subcutaneous mass on the scalp	Frontal	Firm, nontender, growing for 8 mo, 4 cm	None	Gross total removal	R	NA
Fukushima *et al.*,[23] 2007	60/F	Subcutaneous mass on the scalp	Parietal	Firm, nonpulsatile, 6.5 cm	None	Removal	R+C	Alive (3 y)
Mohindra *et al.*,[61] 2007	30/M	Subcutaneous mass on the scalp, headache	Frontal	Growing for 2 mo	NA	Removal	R+C	Alive (6 mo)
Uff and Shieff,[100] 2007	76/F	Subcutaneous mass on the scalp, neurological deficit	Parietal	NA	None	Biopsy	R	Died* (2 wk)
Gaitonde *et al.*,[24] 2008	70/F	Subcutaneous mass on the scalp	Frontal	Growing for 12 mo, 3.4 cm	None	Gross total removal	R+C	Alive (NA)
Gonzalez-Bonet *et al.*, [28] 2008	84/F	Subcutaneous mass on the scalp, seizure	Frontoparietal	Growing for 0.75 mo, 2.5 cm	None	Gross total removal	Refused	Died (5 mo)
Yoon *et al.*,[109] 2008	53/M	Neurological deficit	Frontoparietal	NA	Retrobulbar lesion	Removal	R	Alive (3 y)
Renard *et al.*,[84] 2009	67/F	Subcutaneous mass on the scalp	Frontal	Tender	None	Biopsy	C	NA
da Rocha *et al.*,[15] 2010	13/M	Subcutaneous mass on the scalp	Parietal	NA	NA	NA	C	NA
	66/F	Subcutaneous mass on the scalp	Frontoparietal	NA	NA	NA	C	NA
	72/F	Subcutaneous mass on the scalp	Parieto-occipital, frontal	NA	NA	NA	R+C	NA

(Contd...)

Table 1: (*Continued*).

Author year (reference)	Age (years) /sex	Symptoms at onset	Lesion location	Properties of mass	Other lesions	Surgery	Adjuvant therapies	Outcomes (follow-up)
Khalid *et al.*,[44] 2010	19/M	Subcutaneous mass on the scalp	Parietal, occipital	Firm, nontender, growing for 6 mo, 6 cm	None	Biopsy	C	Alive (2 y)
Ochiai *et al.*,[70] 2010	72/M	Subcutaneous mass on the scalp	Temporoparietal	Firm, nontender, 7 cm	None	Gross total removal	R+C	Alive (1 y)
Castro-Bouzas *et al.*,[10] 2011	59/F	Subcutaneous mass on the scalp	Occipital	Firm, tender, 3 cm	None	Gross total removal	R+C	Alive (30 mo)
Fadoukhair *et al.*,[22] 2011	42/F	Subcutaneous mass on the scalp	Parietal	Firm, tender, 8 cm	None	Biopsy	R+C	Alive (9 mo)
Ciarpaglini and Otten,[13] 2012	74/F	Subcutaneous mass on the scalp	Temporoparietal	Firm, tender, growing for 36 mo, 15 cm	None	Gross total removal	R+C	Alive (2 y)
El Asri *et al.*,[20] 2012	52/M	Subcutaneous mass on the scalp, seizure, proptosis	Frontotemporal	Firm, tender, growing for 7 mo	None	Biopsy	R	Alive (18 mo)
Martin *et al.*,[58] 2012	50/M	Subcutaneous mass on the scalp	Parieto-occipital	Nontender, growing for 4 mo, 10 cm	None	Partial removal	R+C	NA
Rezaei-Kalantari *et al.*,[85] 2012	42/M	Subcutaneous mass on the scalp, headache	Frontal, parietal	Soft, nontender, growing for 4 mo, 10 cm	None	Removal	R+C	NA
Ko *et al.*,[46] 2013	81/F	Subcutaneous mass on the scalp	Frontoparietal	Firm, nontender, 6 cm	None	Removal	C	Alive (9 mo)
Kosugi *et al.*,[48] 2013	82/F	Subcutaneous mass on the scalp	Parietal	Firm, 3 cm	Ileocecal lesion	Biopsy	R+C	Alive (30 mo)
Mishra *et al.*,[60] 2013	34/M	Subcutaneous mass on the scalp	Frontal	Nontender, growing for 12 mo, 7 cm	None	Removal	R+C	Alive (6 mo)
Salunke *et al.*,[86] 2013	30/M	Subcutaneous mass on the scalp, headache	Frontal	Firm, nontender, growing for 3 mo	None	Removal	R+C	Alive (6 mo)
Sanjayan *et al.*,[88] 2013	29/M	Subcutaneous mass on the scalp, headache, and vomiting	Parieto-occipital	NA	Axillary LNs	Gross total removal	R+C	Alive (7 y)
Rasouli *et al.*,[83] 2014	48/F	Subcutaneous mass on the scalp	Frontoparietal	Firm, growing for 4 mo, 5.5 cm	None	Removal	R	Alive (NA)
Sugimoto *et al.*,[92] 2014	39/F	Subcutaneous mass on the scalp	Frontal	Tender	NA	Biopsy	C	Alive (2 y)

(Contd...)

Table 1: (*Continued*).

Author year (reference)	Age (years) /sex	Symptoms at onset	Lesion location	Properties of mass	Other lesions	Surgery	Adjuvant therapies	Outcomes (follow-up)
Tashiro *et al.*,[96] 2015	63/F	Subcutaneous mass on the scalp	Frontal	Growing for 4 mo	None	Subtotal removal	C	Alive (9 y)
	53/M	Subcutaneous mass on the scalp	Frontal	Growing for 2 mo	Cervical LNs	Subtotal removal	R+C	Alive (2 y)
Wang *et al.*,[105] 2015	45/M	Subcutaneous mass on the scalp, neurological deficit	Parietal	Growing for 6 mo, 9 cm	None	Gross total removal	R+C	Alive (12 mo)
Akamatsu *et al.*,[4] 2016	76/F	Subcutaneous mass on the scalp	Frontal	Soft, nontender, growing for 1 mo, 10 cm	Multifocal bone lesions, retroperitoneal lesion	Biopsy	C	Alive (9 mo)
Bhatoe and Ambastha,[8] 2016	14/F	Subcutaneous mass on the scalp	Frontoparietal	Soft, nontender, growing for 6 mo, 5 cm	NA	Gross total removal	NA	NA
Issara *et al.*,[35] 2016	56/F	Enlarging lacuna image of skull	Multiple	NA	None	Removal	R+C	Alive (4 mo)
Jaiswal *et al.*,[36] 2016	40/F	Subcutaneous mass on the scalp, headache	Frontoparietal	Firm, tender, growing for 12 mo	None	Gross total removal	NA	NA
Lv *et al.*,[54] 2016	56/M	Neurological deficit, headache	Parietal	NA	None	Removal	C	Alive (50 mo)
Mascolo *et al.*,[59] 2016	58/M	Subcutaneous mass on the scalp	Parietal, frontal	Growing for 3 mo, 12 cm	None	Biopsy	C	Alive (6 mo)
Kanaya *et al.*,[40] 2017	65/M	Subcutaneous mass on the scalp, neurological deficit	Parietal	NA	Multifocal LNs	Removal	C	Alive (18 mo)
Naama *et al.*,[66] 2017	67/F	Subcutaneous mass on the scalp	Parietal	Nontender, growing for 2 mo, 6 cm	None	Biopsy	R+C	Alive (20 mo)
Chan *et al.*,[11] 2018	49/F	Subcutaneous mass on the scalp	Frontal	Growing for 12 mo, 11 cm	None	Biopsy	C	Alive (6 mo)
Lee and Yun,[51] 2018	50/M	Subcutaneous mass on the scalp, headache	Frontal	Nontender, 3 cm	None	Gross total removal	C	Alive (6 mo)
Salvo *et al.*,[87] 2018	74/F	Neurological deficit	Parietotemporal	NA	None	Removal	C	Alive (6 mo)
Huang *et al.*,[33] 2019	31/M	Subcutaneous mass on the scalp	Parietooccipital	Tender, growing for 2 mo	None	Gross total removal	C	Alive (12 mo)

(Contd...)

Table 1: (*Continued*).

Author year (reference)	Age (years) /sex	Symptoms at onset	Lesion location	Properties of mass	Other lesions	Surgery	Adjuvant therapies	Outcomes (follow-up)
Umemura *et al.*,[102] 2019	72/F	Subcutaneous mass on the scalp	Parietal	Nontender, 10 cm	None	Removal	R	NA
	63/M	Subcutaneous mass on the scalp, neurological deficit	Occipital	Nontender, 4 cm	None	Partial removal	C	NA
Xing *et al.*,[108] 2019	53/M	Subcutaneous mass on the scalp, headache	Parietal	8.2 cm	NA	NA	NA	NA
	28/M	Subcutaneous mass on the scalp	Occipital	5 cm	NA	NA	NA	NA
	68/F	Subcutaneous mass on the scalp	Fronto-temporo-parietal	6.3 cm	NA	NA	NA	NA
	39/M	Subcutaneous mass on the scalp	Frontoparietal	11.2 cm	NA	NA	NA	NA
	45/M	Subcutaneous mass on the scalp, neurological deficit, headache	Parieto-occipital	10.4 cm	NA	NA	NA	NA
Ahmad *et al.*,[3] 2020	37/M	Subcutaneous mass on the scalp, neurological deficit	Frontoparietal	Growing for 3 mo, 5 cm	NA	Biopsy	R+C	NA
Nasim *et al.*,[67] 2020	69/M	Neurological deficit	Parietal	NA	None	Gross total removal	None	Alive (1 y)

The size shown in "Properties of mass" is the greatest diameter of the scalp mass. C: Chemotherapy, Dead*: Died of a cause unrelated to the skull lesion, F: Female, LN: Lymph node, M: Male, NA: Not available, R: Radiotherapy, y: Year, mo: Month

both (n = 75). On MRI (n = 57), a dural tail was observed in 42 cases (74%), and invasion of the brain was observed in 15 cases (26%).

Surgery was detailed in 98 cases, of which 45 (46%) involved a biopsy or partial removal and 53 (54%) involved subtotal or gross total removal [Table 1]. Adjuvant therapy was reported for 96 cases. The breakdown was radiotherapy alone for 24 cases (25%), chemotherapy alone for 34 cases (35%), and both for 38 cases (40%).

The type of lymphocyte was described in 80 cases: 75 (94%) were B-cell lymphomas and 5 (6%) were T-cell lymphomas [Table S2]. Diffuse large B-cell lymphoma (DLBCL) was the most common, being reported in 34 cases.

To determine the rates of survival, we excluded cases in which the patients died of a cause unrelated to the skull lesion and included cases with follow-up periods of more than 6 months (n = 74) or 1 year (n = 54). In the included cases, 67 (90.5%) and 45 (83.3%) patients were alive at the follow-up of 6 months and 1 year, respectively [Table 1].

DISCUSSION

We reviewed the demographic, clinical, and imaging characteristics of cranial vault lymphoma to specify the features that might be helpful for differential diagnosis, especially between lymphomas and meningiomas of the cranial vault.

When a tumor with intra- and extracranial extension sandwiching the skull is seen, meningioma with extracranial extension is often first suspected. In many cases, the subcutaneous scalp mass was firm and nontender, which is also similar to meningioma with extracranial extension. However, in our review, the subcutaneous scalp mass grew very rapidly before the patient presented for treatment, for a mean duration of 5.9 months, which is atypical for meningioma, which is generally slow growing.[18,79]

Laboratory data were unremarkable in many cases. Kosugi *et al*.[48] reported elevated sIL2R, a marker of lymphoproliferative neoplasms,[81] in a patient with cranial vault lymphoma, suggesting that elevated sIL2R associated with a cranial vault tumor might indicate lymphoma rather than meningioma. Another finding that could be used to differentiate the two tumor types is poor or no tumor vascularization on angiograms, which differs from the rich tumor vascularization from the external carotid artery circulation observed in many cases of meningioma.[90] The tumor also showed high uptake on FDG-PET.[4,11,40,86,92,96] Because meningiomas are mostly slow-growing tumors and their glucose metabolism might be only moderately elevated, the high physiological glucose uptake of the normal cerebral cortex leads to a low meningioma-to-background ratio.[26] Hence, a high tumor-to-background ratio may indicate a lymphoma and contribute to ruling out a meningioma.

The extracranial component tended to be at least as large as the intracranial component in cranial vault lymphoma. Because meningioma usually originates from the meninges, theoretically, it tends to grow intracranially rather than extracranially. Extracranial-dominant extension might, therefore, also contribute to distinguishing cranial vault lymphoma from meningioma.

However, other findings are unlikely to clarify the diagnosis. For example, the tumors tended to be hyper- to isodense on CT, iso- to hypointense on T1WI, and hyper-to isointense on T2WI, which are nonspecific features of skull tumors. Although a dural tail was observed in many cases, as reported by Xing *et al*.,[108] this finding is also observed in meningiomas.[79]

It has been suggested that lymphoma cells infiltrate the spaces within the diploe and extend along the emissary veins to infiltrate the soft tissues on either side of the bone.[5,20,30,77] Osteolytic changes were most frequently observed on CT and MRI (68 cases) followed by no changes and/or preserved skull contour (11 cases). In the cases with osteolytic skull changes, permeative dissolution with relatively preserved skull contour was observed in 35 cases. Although the destruction of the cranial vault tended to be small, the extracranial and intracranial components of the tumor tended to be large. These data support the characteristic permeating growth pattern of cranial vault lymphoma involving a large soft-tissue component and slight bony destruction, as reported previously.[20,62,90,108]

After histological confirmation of the diagnosis, patients were usually treated with adjuvant chemotherapy, radiotherapy, or both. Although the best treatment for cranial vault lymphoma has not been elucidated because of the paucity of cases and lack of clinical studies, treatments based on those for systemic malignant lymphoma tended to be adopted.

B-cell lymphomas accounted for approximately 94% of the cranial vault lymphomas, whereas T-cell lymphomas roughly accounted for the remaining 6% of cases. Peripheral T-cell lymphomas account for 6–10% of all cases of non-Hodgkin lymphoma[57] and T-cell lymphomas account for 5–6% of cases of primary lymphoma of bone.[12,82] However, T-cell lymphomas account for only 2% of all primary lymphomas in the central nervous system (CNS),[17] suggesting that cranial vault lymphomas are more similar to extracranial lymphomas than they are to CNS lymphomas. We could not determine the subtypes of lymphoma of all cases using the present classification because our study included some very old reports from before the discovery of lymphocyte markers. Nonetheless, DLBCL accounted for the majority of cases of cranial vault lymphoma in the available data, which is also true for cases of bone lymphoma.[82]

In analyzable follow-up data, 67 and 45 patients were alive after 6 months (n = 74) and 1 year (n = 54), respectively [Table 1]. Although our review does not allow for predicting prognosis due to the limited numbers and the limited duration of follow-up data, the 6-month survival rate and 1-year survival rate are not <60.4% and 40.5%, respectively.

In the present review, we have provided objective data from patients reported in published studies. The difficulty in systematically reviewing the data reported in the literature is the heterogeneity of the data. In particular, almost all reports reviewed for this study were case reports. Because of the limited number and the heterogeneity of described data of preoperative findings and treatments, we could perform only descriptive analyses. Another weakness of the present review resulted from biased reporting in the published studies, with likely underreporting of recent cases in the United States and European countries because of decreased novelty in inverse proportion to the accumulation of reported cases. The data that were more likely to be reported, if present, included findings on MRI and CT. Findings of plain skull X-ray and angiography and long-term follow-up data were less likely to be reported.

The strength of the present review is the comprehensive nature of the literature search. We analyzed cases from all over the world, reported not only in English but also in four non-English languages.

CONCLUSION

Cranial vault lymphoma is a rare entity among skull tumors. To the best of our knowledge, this is the largest reported pooled database describing cranial vault lymphoma patients. The most common symptom was a rapidly growing subcutaneous scalp mass. The tumor was poorly vascularized on angiography. Skull destruction on images was mild and disproportionately small despite the large size of the extracranial and/or intracranial component in two-thirds of the cases. These features should help to distinguish lymphoma from meningioma.

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Declaration of patient consent

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Conflicts of interest

There are no conflicts of interest.

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SUPPLEMENTARY TABLES

Table S1: Laboratory data and features on images of patients with cranial vault lymphoma.

Author year (reference)	Labo data	Plain skull X-ray	Angiogram	Density on CT	Enhancement on CECT	Intensity on T1WI	Intensity on T2WI	Enhancement on CET1WI	Additional
Wichtl,[106] 1949	N/A	Destruction of bones	NA	NA	NA	NA	NA	NA	NA
Strange and De Lorimier,[91] 1954	Unremarkable	Dissolution of the tables, periosteal reaction	NA	NA	NA	NA	NA	NA	NA
	Unremarkable	Dissolution and erosion of the tables, periosteal reaction	NA	NA	NA	NA	NA	NA	NA
	N/A	Bone destruction, erosion of the inner table	NA	NA	NA	NA	NA	NA	NA
Ullrich and Bucy,[101] 1958	Unremarkable	Patchy decalcification, progressive bone destruction	NA	NA	NA	NA	NA	NA	NA
Piendak and Alder,[80] 1959	Mild increase in WBCs	Irregular lytic lesion	NA	NA	NA	NA	NA	NA	NA
Block and Peck,[9] 1964	N/A	Periosteal reaction, the diploic space, and inner table were normal	NA	NA	NA	NA	NA	NA	NA
Topolnicki and White,[99] 1969	N/A	Destruction of the outer table	NA	NA	NA	NA	NA	NA	NA
Wain wright,[104] 1973	Unremarkable	Marked erosion	NA	NA	NA	NA	NA	NA	NA
Gawish,[27] 1976	Lymphocytosis, eosinophilia	Rarefaction of the bone with erosion	Mass effect with no blood circulation to the tumor	NA	NA	NA	NA	NA	NA
Agbi *et al.*,[1] 1983	Unremarkable	An area of hyperostosis	Mass effect with no pathological circulation	Hyper	Well, homogeneous	NA	NA	NA	NA
Holtås *et al.*,[30] 1985	N/A	NA	NA	NA	Well, diffuse	NA	NA	NA	NA
	Unremarkable	NA	NA	NA	Well, diffuse	NA	NA	NA	NA
Kinjo and Satoh,[45] 1985	Dehydration, hypercalcemia	Multiple punched out lesions	No abnormalities	Iso	Well, diffuse	NA	NA	NA	Uptake of 99m Tc-MDP
Thomas and Kennedy,[97] 1986	N/A	A large lytic lesion	NA	Hypo	Well, diffuse	NA	NA	NA	NA

(Contd...)

Table S1: (*Continued*).

Author year (reference)	Labo data	Plain skull X-ray	Angiogram	Density on CT	Enhancement on CECT	Intensity on T1WI	Intensity on T2WI	Enhancement on CET1WI	Additional
Howat *et al.*,[32] 1987	N/A	NA	NA	NA	NA	NA	NA	NA	NA
Maiuri *et al.*,[56] 1987	Unremarkable	Osteolytic area with irregular margins	Mass effect with no pathological vascularization	Hyper	Slight, diffuse	NA	NA	NA	NA
Tagawa *et al.*,[94] 1987	Increase in LDH	NA	NA	NA	Well, diffuse	NA	NA	NA	Uptake of Ga-67
Kawakami *et al.*,[42] 1988	Unremarkable	Sharp bone defect	An avascular mass	NA	Well, diffuse	NA	NA	NA	NA
Morgello *et al.*,[63] 1989	N/A	NA	NA	NA	NA	NA	NA	NA	NA
Herkes *et al.*,[29] 1991	Unremarkable	A permeative lytic lesion	NA	NA	NA	NA	NA	Marked	NA
	Hyperglycemia, increase in Cre and urea	NA	Partial obstruction of the sinus	NA	NA	NA	NA	NA	NA
Kumon *et al.*,[49] 1991	Unremarkable	No abnormalities	Vascularization from ophthalmic artery branches	Iso	Slight, diffuse	Iso	Iso	NA	NA
Natsuda *et al.*,[68] 1991	HTLV-1 positive	Multiple lytic lesions with sharp margin, no sclerosis	NA	NA	NA	NA	NA	Enhanced	Uptake of Ga-67
Lonjon *et al.*,[53] 1993	N/A	Osteolysis	Vascularization from occipital artery circulation	NA	Well, diffuse	NA	NA	NA	NA
Parekh *et al.*,[75] 1993	Unremarkable	Hyperostosis	NA	Hyper	Well, diffuse	NA	NA	NA	NA
Sato *et al.*,[89] 1993	Unremarkable	An osteolytic lesion with an irregular margin	NA	Iso to hyper	Well, diffuse	Hypo	Hyper	Marked	NA
Kelleher *et al.*,[43] 1994	HIV positive	NA	NA	NA	NA	NA	NA	NA	Uptake on bone scintigraphy
Loembe *et al.*,[52] 1994	Anemia, accelerated ESR	Osteolysis with intratumoral calcifications	Vascularization from the meningeal and scalp vessels	NA	NA	NA	NA	NA	NA
Morioka *et al.*,[64] 1994	N/A	NA	NA	NA	Well, diffuse	NA	NA	NA	NA
Vigushin *et al.*,[103] 1994	Monoclonal κ chains in urine	NA	NA	NA	NA	Hypo	NA	NA	Uptake of 123I-SAP
Wittram *et al.*,[107] 1994	N/A	NA	NA	Hyper	Well, diffuse (intracranial), peripheral (extracranial)	NA	NA	NA	NA

(Contd...)

Table S1: (*Continued*).

Author year (reference)	Labo data	Plain skull X-ray	Angiogram	Density on CT	Enhancement on CECT	Intensity on T1WI	Intensity on T2WI	Enhancement on CET1WI	Additional
Aslan *et al.*,[6] 1995	Unremarkable	An osteolytic soft tissue mass	NA	NA	NA	Hypo	Hyper	Marked	NA
Landys *et al.*,[50] 1995	N/A	Destruction of bones	NA	Hyper	Well, diffuse	NA	NA	NA	NA
Paige and Bernstein,[72] 1995	N/A	NA	Vascularization from ICA and ECA circulation	NA	Well, diffuse	Iso to hypo	Iso to hypo	Marked	NA
	N/A	NA	Poor vascularization from ICA and ECA circulation	NA	NA	NA	Iso	Marked	NA
Isla *et al.*,[34] 1996	Unremarkable	NA	NA	NA	Well, diffuse	NA	NA	NA	NA
Bhatia *et al.*,[7] 1997	HIV positive	Ill-defined serpentine bone resorption	NA	NA	NA	NA	NA	Enhanced	Uptake of 99Tc
Curty *et al.*,[14] 1997	N/A (HIV negative)	NA	NA	Hyper	Well, diffuse	NA	NA	NA	Uptake on bone scintigraphy
Muin *et al.*,[65] 1997	N/A	NA	NA	NA	Well, diffuse	NA	NA	NA	NA
Jamjoom *et al.*,[37] 1998	N/A (HIV negative)	NA	Slight peripheral vascularization from ECA circulation.	NA	Well, diffuse	NA	NA	NA	NA
Jiménez Moragas *et al.*,[38] 1999	HIV positive, decreased PLTs,	NA	NA	NA	NA	NA	NA	NA	NA
Dai *et al.*,[16] 2000	Increase in LDH	An osteolytic lesion with an irregular margin	NA	NA	NA	NA	NA	Marked	NA
Pardhanani *et al.*,[74] 2000	N/A	NA	NA	NA	NA	NA	NA	NA	NA
Parker *et al.*,[76] 2001	Unremarkable	An osteolytic lesion	NA	NA	NA	NA	NA	NA	NA
Thurnher *et al.*,[98] 2001	HIV positive	NA	NA	Hyper	Well, diffuse (intracranial), peripheral (extracranial)	NA	NA	NA	NA
Duyndam *et al.*,[19] 2002	Unremarkable	No abnormalities	NA	NA	NA	Hypo	Iso	Heterogeneous	NA
Pernot *et al.*,[78] 2002	N/A	NA	NA	NA	NA	Iso	Iso	Homogeneous	NA

(Contd...)

Table S1: (*Continued*).

Author year (reference)	Labo data	Plain skull X-ray	Angiogram	Density on CT	Enhancement on CECT	Intensity on T1WI	Intensity on T2WI	Enhancement on CET1WI	Additional
Kanai *et al.*,[39] 2003	Increase in LDH and ALP	NA	NA	Hyper	Well, diffuse	Hypo	Hyper	Enhanced	Uptake on bone and Ga scintigraphy
Kantarci *et al.*,[41] 2003	Increase in LDH	NA	NA	NA	NA	Iso	Iso	Marked	NA
Koral *et al.*,[47] 2003	N/A	NA	NA	NA	Well, diffuse	NA	NA	NA	NA
Mongia *et al.*,[62] 2003	N/A	No bony erosion	NA	NA	NA	NA	NA	NA	NA
Nishimoto *et al.*,[69] 2003	Unremarkable	NA	NA	Hypo	Well, diffuse	Hypo	Hypo	Homogeneous	NA
Aquilina *et al.*,[5] 2004	N/A (HIV negative)	NA	NA	NA	Well, diffuse	NA	NA	NA	NA
Horstman *et al.*,[31] 2004	N/A	NA	NA	NA	Well, diffuse	NA	NA	NA	NA
Madan *et al.*,[55] 2004	N/A	NA	NA	Hyper	NA	NA	NA	NA	NA
Szucs-Farkas *et al.*,[93] 2005	HIV positive	NA	NA	Hyper	Well, diffuse	NA	NA	NA	NA
Tanimura *et al.*,[95] 2005	Anemia, increase in LDH and ferritin	Ill-defined serpentine bone resorption	NA	Iso	NA	Iso to hypo	Iso to hypo	Homogeneous	NA
Evliyaoglu *et al.*,[21] 2006	N/A	No abnormalities	NA	Hyper	Slight, diffuse	NA	NA	NA	NA
Galarza *et al.*,[25] 2006	N/A	NA	NA	Hyper	Well, diffuse	Hypo	Hyper	Enhanced	NA
Palled *et al.*,[73] 2006	N/A	NA	NA	NA	Well, diffuse	NA	NA	Enhanced	NA
Agrawal *et al.*,[2] 2007	Accelerated ESR	NA	NA	NA	Well, diffuse	NA	NA	NA	NA
Fukushima *et al.*,[23] 2007	Unremarkable	An osteolytic lesion	NA	NA	Slight, diffuse	Hypo	Hyper	Homogeneous	Uptake on bone scintigraphy
Mohindra *et al.*,[61] 2007	N/A	NA	NA	NA	Well, diffuse	NA	NA	Marked	NA
Uff and Shieff,[100] 2007	N/A (HIV negative)	NA	NA	NA	Well, diffuse	NA	Hyper	Enhanced	NA
Gaitonde *et al.*,[24] 2008	N/A	NA	NA	NA	NA	NA	NA	NA	NA
Gonzalez-Bonet *et al.*,[28] 2008	Hypogammaglobulinemia	NA	NA	NA	NA	NA	NA	Marked	NA

(Contd...)

Table S1: (*Continued*).

Author year (reference)	Labo data	Plain skull X-ray	Angiogram	Density on CT	Enhancement on CECT	Intensity on T1WI	Intensity on T2WI	Enhancement on CET1WI	Additional
Yoon *et al.*,[109] 2008	Unremarkable	NA	NA	Hyper	NA	Hypo	Hyper	NA	NA
Renard *et al.*,[84] 2009	N/A	NA	NA	Hyper	Well, diffuse	Iso	Iso	Marked, heterogeneous	NA
da Rocha *et al.*,[15] 2010	N/A	NA	NA	NA	NA	Hypo	Hyper	Enhanced	NA
	N/A	NA	NA	NA	NA	NA	NA	NA	NA
	N/A	NA	NA	NA	NA	NA	NA	NA	NA
Khalid *et al.*,[44] 2010	Unremarkable	NA	NA	Hyper	Well, diffuse	NA	NA	NA	NA
Ochiai *et al.*,[70] 2010	N/A	No destruction or periosteal reaction	NA	NA	NA	Hypo	Hyper	Homogeneous	NA
Castro-Bouzas *et al.*,[10] 2011	N/A	NA	NA	NA	NA	NA	NA	NA	NA
Fadoukhair *et al.*,[22] 2011	N/A	NA	NA	NA	NA	NA	NA	NA	NA
Ciarpaglini and Otten,[13] 2012	Unremarkable	NA	NA	NA	NA	Hypo	Hyper	Homogeneous	NA
El Asri *et al.*,[20] 2012	Unremarkable	NA	NA	Hyper	Slight, diffuse	Iso	Iso	Marked	Uptake of 99 Tc MDP
Martin *et al.*,[58] 2012	HBV positive	NA	NA	NA	NA	NA	NA	NA	NA
Rezaei-Kalantari *et al.*,[85] 2012	Unremarkable	Neither obvious bone erosion nor sclerosis	NA	Hyper	NA	Iso	Iso	Marked, homogeneous	NA
Ko *et al.*,[46] 2013	Unremarkable	An irregular inner cortical bone margin	Vascularization from ECA circulation.	Iso	Well, diffuse	Iso to hypo	Iso to hypo	Enhanced	A choline peak on MRS
Kosugi,[48] 2013	HCV positive, increase in sIL2R	NA	NA	NA	NA	Iso to hypo	Iso to hypo	NA	Uptake on bone scintigraphy
Mishra *et al.*,[60] 2013	N/A	Moth-eaten lytic lesion	NA	NA	Well, diffuse	NA	NA	NA	NA
Salunke *et al.*,[86] 2013	Unremarkable	NA	NA	NA	NA	Hypo	Hyper	Enhanced	Uptake on PET-CT
Sanjayan *et al.*,[88] 2013	N/A	NA	NA	NA	NA	NA	NA	Enhanced	NA
Rasouli *et al.*,[83] 2014	N/A	NA	NA	NA	NA	NA	NA	NA	NA

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Table S1: (*Continued*).

Author year (reference)	Labo data	Plain skull X-ray	Angiogram	Density on CT	Enhancement on CECT	Intensity on T1WI	Intensity on T2WI	Enhancement on CET1WI	Additional
Sugimoto *et al.*,[92] 2014	Anemia	Osteolytic bone destruction	NA	NA	NA	NA	NA	NA	Uptake on bone scintigraphy and FDG-PET
Tashiro *et al.*,[96] 2015	N/A (HIV negative)	NA	NA	NA	NA	Hypo	Hyper	Homogeneous	Uptake on Ga-67 scintigraphy
	N/A (HIV negative)	NA	NA	NA	NA	Hypo	Hypre	Heterogeneous	Uptake on FDG-PET
Wang *et al.*,[105] 2015	N/A	NA	NA	Hyper	NA	Hypo	Hyper	Homogeneous	NA
Akamatsu *et al.*,[4] 2016	N/A	NA	NA	NA	NA	NA	NA	NA	Uptake on FDG-PET
Bhatoe and Ambastha,[8] 2016	Unremarkable	Erosion of the calvarium	NA	NA	NA	NA	NA	Enhanced	NA
Issara *et al.*,[35] 2016	N/A	NA	NA	NA	NA	NA	NA	Slight, heterogeneous	NA
Jaiswal *et al.*,[36] 2016	Unremarkable	NA	NA	NA	Well, diffuse	NA	NA	Enhanced	NA
Lv *et al.*,[54] 2016	Unremarkable	NA	NA	Hyper	NA	Hypo	Iso	Marked	NA
Mascolo *et al.*,[59] 2016	Unremarkable	NA	NA	NA	NA	NA	NA	Heterogeneous	NA
Kanaya *et al.*,[40] 2017	N/A	NA	NA	NA	NA	NA	NA	NA	Uptake on FDG-PET
Naama *et al.*,[66] 2017	Unremarkable	NA	NA	Hyper	Well, diffuse	Hypo	Hyper	Heterogeneous	NA
Chan *et al.*,[11] 2018	Increase in LDH	NA	NA	NA	NA	NA	Hyper	NA	Uptake on FDG-PET
Lee and Yun,[51] 2018	Unremarkable	NA	NA	NA	NA	Hyper to iso	Hyper to iso	Homogeneous	NA
Salvo *et al.*,[87] 2018	Unremarkable	NA	NA	NA	NA	Hypo	Iso to hypo	Homogeneous	Hypointense on ADC map
Huang *et al.*,[33] 2019	Increase in WBCs	NA	No vascular malformations	NA	NA	Iso	Iso	Heterogeneous	Hyperintense on DWI
Umemura *et al.*,[102] 2019	Chronic renal failure	NA	NA	Hyper	NA	Iso	Iso	NA	NA
	Unremarkable	NA	NA	NA	NA	Hypo	Hyper	Homogeneous	NA
Xing *et al.*,[108] 2019	N/A	NA	NA	Hyper	NA	Iso	Hyper	Heterogeneous	NA
	N/A	NA	NA	Hyper	NA	Iso	Hyper	Homogeneous	NA

(Contd...)

Table S1: (*Continued*).

Author year (reference)	Labo data	Plain skull X-ray	Angiogram	Density on CT	Enhancement on CECT	Intensity on T1WI	Intensity on T2WI	Enhancement on CET1WI	Additional
	N/A	NA	NA	NA	NA	Hypo	Hyper	Homogeneous	NA
	N/A	NA	NA	Iso	NA	Iso	Iso	Homogeneous	NA
	N/A	NA	NA	Hyper	NA	Hypo	Hyper	Homogeneous	NA
Ahmad *et al.*,[3] 2020	N/A	NA	NA	NA	NA	NA	NA	Heterogeneous	NA
Nasim *et al.*,[67] 2020	Increase in WBCs	NA	NA	NA	NA	NA	NA	NA	NA

ADC: Apparent diffusion coefficient, ALP: Alkaline phosphatase, CE: Contrast enhanced, Cre: Creatinine, CT: Computed tomography, DWI: Diffusion-weighted imaging, ECA: External carotid artery, ESR: Erythrocyte sedimentation rate, FDG: Fluorodeoxyglucose F 18, Ga: Gallium, HBV: Hepatitis B virus, HCV: Hepatitis C virus, HIV: Human immunodeficiency virus, HTLV-1: Human T-cell leukemia virus type 1, I: Iodine, ICA: Internal carotid artery, LDH: Lactate dehydrogenase, MDP: Methylene diphosphonate, MRI: Magnetic resonance imaging, MRS: Magnetic resonance spectroscopy, N/A: Not available, PET: Positron emission tomography, PLT: Platelet, SAP: Serum amyloid P, sIL2R: Soluble interleukin 2 receptor, T1WI: T1-weighted imaging, T2WI: T2-weighted imaging, Tc: Technetium, WBC: White blood cell

Table S2: Bone imaging, dural tail, brain invasion, and pathological features of patients with cranial vault lymphoma.

Author year reference	Predominance in tumor extension	Findings of skull bone on CT and/or MRI	Disproportionately small amount of cortical destruction	Dural tail	Invasion of brain	Histology	Lymphocyte subtype
Wichtl,[106] 1949	NA	NA	NA	NA	NA	Reticulum cell sarcoma	NA
Strange and De Lorimier,[91] 1954	NA	NA	NA	NA	NA	Reticulum cell lymphosarcoma	NA
	NA	NA	NA	NA	NA	Reticulum cell sarcoma	NA
	NA	NA	NA	NA	NA	Reticulum cell sarcoma	NA
Ullrich and Bucy,[101] 1958	NA	NA	NA	NA	NA	Reticulum cell sarcoma	NA
Piendak and Alder,[80] 1959	NA	NA	NA	NA	NA	Reticulum cell sarcoma	NA
Block and Peck,[9] 1964	NA	NA	NA	NA	NA	Lymphosarcoma	NA
Topolnicki and White,[99] 1969	NA	NA	NA	NA	NA	Reticulum cell sarcoma	NA
Wainwright,[104] 1973	NA	NA	NA	NA	NA	Reticulum cell sarcoma	NA
Gawish,[27] 1976	NA	NA	NA	NA	NA	Burkitt lymphoma	NA
Agbi *et al.*,[1] 1983	Intra	No change	Yes	NA	NA	Diffuse, non-Hodgkin malignant lymphoma with cleaved and notched nuclei	NA
Holtås *et al.*,[30] 1985	None	Permeative dissolution with preserved skull contour	Yes	NA	NA	Undifferentiated large cell malignant lymphoma	NA
	None	No change	Yes	NA	NA	Poorly differentiated non-Hodgkin lymphoma	NA
Kinjo and Satoh,[45] 1985	None	Osteolytic skull defect	No	NA	NA	Malignant non-Hodgkin lymphoma, diffuse pleomorphic type	NA
Thomas and Kennedy,[97] 1986	Extra	Osteolytic skull defect	No	NA	NA	Nodular sclerosing Hodgkin disease	NA
Howat *et al.*,[32] 1987	NA	NA	NA	NA	NA	Malignant lymphoma of mixed large and medium-sized cells type, some with cleaved nuclei	NA
Maiuri *et al.*,[56] 1987	Intra	Osteolytic skull defect	No	NA	NA	Lymphoblastic lymphoma	NA
Tagawa *et al.*,[94] 1987	None	Permeative dissolution with preserved skull contour	No	NA	NA	Diffuse lymphoma, mixed cell type	NA

(Contd...)

Table S2: (*Continued*).

Author year reference	Predominance in tumor extension	Findings of skull bone on CT and/or MRI	Disproportionately small amount of cortical destruction	Dural tail	Invasion of brain	Histology	Lymphocyte subtype
Kawakami *et al.*,[42] 1988	None	Osteolytic skull defect	No	NA	NA	Malignant lymphoma of diffuse medium-sized cell type	NA
Morgello *et al.*,[63] 1989	NA	NA	NA	NA	NA	Large cell lymphoma	T-cell
Herkes *et al.*,[29] 1991	Extra	Permeative dissolution	No	Yes	Yes	Malignant non-Hodgkin lymphoma, diffuse large cell type, noncleaved subtype	NA
	NA	Demineralization of the intervening bone	NA	NA	NA	Malignant non-Hodgkin lymphoma, diffuse mixed cell type	NA
Kumon *et al.*,[49] 1991	Extra	Permeative dissolution with preserved skull contour	Yes	NA	No	Malignant lymphoma of large- or medium-sized cell type with noncleaved nuclei	B-cell
Natsuda *et al.*,[68] 1991	Extra	NA	NA	NA	Yes	Malignant non-Hodgkin lymphoma, diffuse large or medium-sized cell type with cleaved nuclei.	T-cell
Lonjon *et al.*,[53] 1993	Extra	Osteolytic skull defect	No	NA	NA	B centroblastic lymphoma	B-cell
Parekh *et al.*,[75] 1993	Intra	Preserved skull contour	Yes	NA	NA	Malignant B-cell non-Hodgkin lymphoma	B-cell
Sato *et al.*,[89] 1993	None	NA	No	Yes	No	Diffuse large B-cell type	B-cell
Kelleher *et al.*,[43] 1994	Extra	NA	NA	NA	NA	High-grade, Ber H2/Ki-1-positive, diffuse large-cell anaplastic lymphoma	B-cell
Loembe *et al.*,[52] 1994	NA	NA	NA	NA	NA	Diffuse malignant lymphoma: pleomorphic small and large lymphocytes	NA
Morioka *et al.*,[64] 1994	Extra	Osteolysis with preserved skull contour	Yes	NA	NA	Malignant lymphoma with thick fibrous stroma	B-cell
Vigushin *et al.*,[103] 1994	Intra	NA	NA	NA	NA	Lymphoplasmacytic lymphoma	B-cell
Wittram *et al.*,[107] 1994	None	Permeative dissolution with preserved skull contour	Yes	NA	NA	Centrocytic/centroblastic low-grade non-Hodgkin lymphoma	NA

(Contd...)

Table S2: (*Continued*).

Author year reference	Predominance in tumor extension	Findings of skull bone on CT and/or MRI	Disproportionately small amount of cortical destruction	Dural tail	Invasion of brain	Histology	Lymphocyte subtype
Aslan *et al.*,[6] 1995	Extra	Permeative dissolution with preserved skull contour and periosteal reaction	Yes	Yes	NA	Undifferentiated Burkitt lymphoma	B-cell
Landys *et al.*,[50] 1995	None	Osteolytic defect	No	NA	NA	High-grade, malignant non-Hodgkin lymphoma of centroblastic type	NA
Paige and Bernstein,[72] 1995	None	Permeative dissolution with preserved skull contour	Yes	Yes	Yes	Large-cell malignant lymphoma	NA
	Extra	Permeative dissolution with preserved skull contour	Yes	Yes	No	Large-cell malignant lymphoma	NA
Isla *et al.*,[34] 1996	None	Permeative dissolution with preserved skull contour and periosteal reaction	Yes	NA	NA	Low-grade, malignant non-Hodgkin lymphoma, centroblastic-centrocytic type with follicular pattern	B-cell
Bhatia *et al.*,[7] 1997	Extra	NA	NA	NA	No	Large B-cell lymphoma	B-cell
Curty *et al.*,[14] 1997	Intra	Osteolytic skull defect	No	NA	NA	High-grade B-cell lymphoma, centroblastic type	B-cell
Muin *et al.*,[65] 1997	Intra	Hyperostosis	Yes	NA	NA	High-grade B-cell non-Hodgkin lymphoma	B-cell
Jamjoom *et al.*,[37] 1998	Intra	Osteolytic skull defect	No	NA	NA	Large T-cell lymphoma, immunoblastic type	T-cell
Jiménez Moragas *et al.*,[38] 1999	NA	Multiple osteolytic lesions	NA	NA	NA	Large B-cell non-Hodgkin lymphoma	B-cell
Dai *et al.*,[16] 2000	None	NA	No	Yes	No	Diffuse large B-cell non-Hodgkin lymphoma	B-cell
Pardhanani *et al.*,[74] 2000	None	Osteolytic skull defect	No	NA	NA	Diffuse large B-cell lymphoma of follicular center cell origin	B-cell
Parker *et al.*,[76] 2001	NA	NA	NA	NA	NA	Anaplastic large-cell lymphoma	NA
Thurnher *et al.*,[98] 2001	Extra	Permeative dissolution with preserved skull contour	Yes	NA	NA	Large B-cell lymphoma	B-cell

(Contd...)

Table S2: (*Continued*).

Author year reference	Predominance in tumor extension	Findings of skull bone on CT and/or MRI	Disproportionately small amount of cortical destruction	Dural tail	Invasion of brain	Histology	Lymphocyte subtype
Duyndam *et al.*,[19] 2002	Extra	NA	Yes	Yes	No	Malignant B-cell non-Hodgkin lymphoma	B-cell
Pernot *et al.*,[78] 2002	Extra	NA	Yes	Yes	No	B-cell lymphoma with small, cleaved cells	B-cell
Kanai *et al.*,[39] 2003	Extra	Permeative dissolution with preserved skull contour	Yes	NA	No	Diffuse, medium-sized B-cell non-Hodgkin lymphoma	B-cell
Kantarci *et al.*,[41] 2003	None	NA	Yes	Yes	Yes	Diffuse, large-cell-type lymphoma	B-cell
Koral *et al.*,[47] 2003	None	Osteolysis of more than half of the skull thickness	No	NA	NA	Diffuse, histiocytic, large-cell NHL	NA
Mongia *et al.*,[62] 2003	Extra	NA	NA	NA	NA	Non-Hodgkin lymphoma	NA
Nishimoto *et al.*,[69] 2003	Extra	Permeative dissolution with preserved skull contour	Yes	Yes	No	Large B-cell lymphoma	B-cell
Aquilina *et al.*,[5] 2004	None	Permeative dissolution with preserved skull contour	Yes	NA	NA	Small-to-intermediate B-cell lymphoma	B-cell
Horstman *et al.*,[31] 2004	Extra	Osteolytic skull defect	Yes	NA	NA	Diffuse large B-cell non-Hodgkin lymphoma	B-cell
Madan *et al.*,[55] 2004	Extra	Permeative dissolution with preserved skull contour	Yes	NA	NA	Diffuse B-cell lymphoma	B-cell
Szucs-Farkas *et al.*,[93] 2005	Extra	Osteolytic skull defect	No	Yes	No	Peripheral B-cell lymphoma, Burkitt-like	B-cell
Tanimura *et al.*,[95] 2005	Extra	Osteolytic skull defect	No	NA	No	Large B-cell non-Hodgkin lymphoma	B-cell
Evliyaoglu *et al.*,[21] 2006	Intra	Permeative dissolution with preserved skull contour	Yes	NA	NA	Diffuse, small B-cell non-Hodgkin lymphoma	B-cell
Galarza *et al.*,[25] 2006	Intra	No change	Yes	Yes	No	Diffuse, large B-cell lymphoma	B-cell
Palled *et al.*,[73] 2006	None	Osteolysis with periosteal reaction	NA	NA	No	High-grade B-cell Burkitt-type non-Hodgkin lymphoma	B-cell
Agrawal *et al.*,[2] 2007	Intra	Hyperostosis	Yes	NA	NA	Diffuse, large-cell non-Hodgkin lymphoma (MALToma type)	NA

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Table S2: (*Continued*).

Author year reference	Predominance in tumor extension	Findings of skull bone on CT and/or MRI	Disproportionately small amount of cortical destruction	Dural tail	Invasion of brain	Histology	Lymphocyte subtype
Fukushima *et al.*,[23] 2007	None	Osteolytic skull defect	No	No	No	Diffuse medium-sized B-cell non-Hodgkin lymphoma	B-cell
Mohindra *et al.*,[61] 2007	None	Preserved skull contour	Yes	Yes	Yes	B-cell lymphoma	B-cell
Uff and Shieff,[100] 2007	Extra	Osteolysis of more than half of the skull thickness	Yes	Yes	Yes	Diffuse, large B-cell lymphoma	B-cell
Gaitonde *et al.*,[24] 2008	None	Osteolysis	NA	NA	NA	Follicular lymphoma, grade 2	B-cell
Gonzalez-Bonet *et al.*,[28] 2008	None	Osteolytic skull defect	No	No	No	B-cell immunoblastic lymphoma	B-cell
Yoon *et al.*,[109] 2008	Intra	Permeative dissolution with preserved skull contour	Yes	NA	No	Small lymphocytic B-cell lymphoma	B-cell
Renard *et al.*,[84] 2009	None	Osteolysis of more than half of the skull thickness with periosteal bone formation	No	Yes	No	Diffuse, malignant, large B-cell non-Hodgkin lymphoma	B-cell
da Rocha *et al.*,[15] 2010	None	No change	NA	Yes	No	Diffuse, large B-cell lymphoma	B-cell
	NA	No change	NA	NA	NA	Mantle cell lymphoma	B-cell
	NA	No change	NA	NA	NA	Small lymphocytic lymphoma	NA
Khalid *et al.*,[44] 2010	None	Permeative dissolution with preserved skull contour	Yes	NA	NA	Low-grade B-cell non-Hodgkin lymphoma	B-cell
Ochiai *et al.*,[70] 2010	Intra	No change	Yes	Yes	Yes	Diffuse, large B-cell lymphoma	B-cell
Castro-Bouzas *et al.*,[10] 2011	Extra	Permeative dissolution	NA	NA	NA	ALK-negative, T-cell non-Hodgkin lymphoma	T-cell
Fadoukhair *et al.*,[22] 2011	Extra	Osteolytic skull defect	No	NA	NA	Diffuse, large B-cell non-Hodgkin lymphoma	B-cell
Ciarpaglini and Otten,[13] 2012	Extra	Osteolysis	NA	Yes	Yes	B-cell GC-like non-Hodgkin lymphoma	B-cell
El Asri *et al.*,[20] 2012	None	Sclerosis and hyperostosis	Yes	Yes	NA	Non-Hodgkin lymphoma (MALT type)	B-cell
Martin *et al.*,[58] 2012	Extra	Permeative dissolution with preserved skull contour and periosteal bone formation	Yes	NA	NA	Diffuse, B-cell lymphoma	B-cell

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Table S2: (*Continued*).

Author year reference	Predominance in tumor extension	Findings of skull bone on CT and/or MRI	Disproportionately small amount of cortical destruction	Dural tail	Invasion of brain	Histology	Lymphocyte subtype
Rezaei-Kalantari *et al.*,[85] 2012	None	Hyperostosis	Yes	Yes	No	Diffuse, large B-cell lymphoma	B-cell
Ko *et al.*,[46] 2013	None	Permeative dissolution with preserved skull contour and periosteal bone formation	Yes	Yes	No	Diffuse, large B-cell lymphoma	B-cell
Kosugi,[48] 2013	None	NA	No	NA	NA	Diffuse, large B-cell lymphoma	B-cell
Mishra *et al.*,[60] 2013	None	Permeative dissolution with preserved skull contour and periosteal bone formation	Yes	NA	NA	Diffuse, large B-cell lymphoma	B-cell
Salunke *et al.*,[86] 2013	None	Permeative dissolution with preserved skull contour, periosteal bone formation and sclerosis	Yes	Yes	Yes	Diffuse, large B-cell lymphoma	B-cell
Sanjayan *et al.*,[88] 2013	Intra	NA	Yes	Yes	No	Mixed small and large B-cell lymphoma	B-cell
Rasouli *et al.*,[83] 2014	Extra	Permeative dissolution with preserved skull contour	Yes	NA	NA	Extranodal marginal zone lymphoma (EMZL)	B-cell
Sugimoto *et al.*,[92] 2014	NA	Osteolysis of more than half of the skull thickness	NA	NA	NA	Spindle-shaped, diffuse, large B-cell lymphoma with a storiform pattern (germinal center type)	B-cell
Tashiro *et al.*,[96] 2015	Extra	Permeative dissolution with preserved skull contour	Yes	Yes	No	Diffuse, large B-cell lymphoma	B-cell
	Extra	No change	Yes	Yes	No	Diffuse, large B-cell lymphoma	B-cell
Wang *et al.*,[105] 2015	Intra	NA	Yes	Yes	No	Diffuse, large B-cell lymphoma	B-cell
Akamatsu *et al.*,[4] 2016	None	Osteolytic skull defect	No	NA	NA	Diffuse, large B-cell lymphoma	B-cell
Bhatoe and Ambastha,[8] 2016	Extra	Osteolytic skull defect	Yes	Yes	Yes	ALK-positive, anaplastic large-cell lymphoma	T-cell
Issara *et al.*,[35] 2016	No extension	Permeative dissolution with preserved skull contour	NA	No	No	Small B-cell lymphoma	B-cell

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Table S2: (*Continued*).

Author year reference	Predominance in tumor extension	Findings of skull bone on CT and/or MRI	Disproportionately small amount of cortical destruction	Dural tail	Invasion of brain	Histology	Lymphocyte subtype
Jaiswal *et al.*,[36] 2016	Extra	Osteolysis of more than half of the skull thickness	Yes	Yes	Yes	Diffuse, large B-cell lymphoma	B-cell
Lv *et al.*,[54] 2016	Intra	Permeative dissolution with preserved skull contour and periosteal bone formation	Yes	Yes	Yes	Diffuse, large B-cell lymphoma	B-cell
Mascolo *et al.*,[59] 2016	Extra	Permeative dissolution with preserved skull contour	Yes	Yes	No	Diffuse, large B-cell lymphoma	B-cell
Kanaya *et al.*,[40] 2017	NA	Osteolytic skull defect	No	NA	NA	Diffuse, large B-cell lymphoma	B-cell
Naama *et al.*,[66] 2017	None	Permeative dissolution with preserved skull contour	Yes	Yes	Yes	Diffuse, large B-cell lymphoma	B-cell
Chan *et al.*,[11] 2018	Extra	Preserved skull contour	Yes	Yes	NA	Diffuse, large B-cell lymphoma	B-cell
Lee and Yun,[51] 2018	None	Sclerosis and osteolysis	Yes	Yes	No	Diffuse, large B-cell lymphoma	B-cell
Salvo *et al.*,[87] 2018	None	Sclerosis	Yes	Yes	Yes	Diffuse, large B-cell lymphoma	B-cell
Huang *et al.*,[33] 2019	Extra	Osteolysis	No	Yes	No	Plasmablastic non-Hodgkin lymphoma (a rare subtype of DLBCL)	B-cell
Umemura *et al.*,[102] 2019	Extra	Permeative dissolution with preserved skull contour	Yes	NA	NA	Diffuse, large B-cell lymphoma	B-cell
	None	Osteolysis of more than half of the skull thickness	No	Yes	No	Diffuse, large B-cell lymphoma	B-cell
Xing *et al.*,[108] 2019	NA	Permeative dissolution with preserved skull contour	NA	Yes	NA	B-cell non-Hodgkin lymphoma	B-cell
	NA	Permeative dissolution with preserved skull contour	NA	Yes	NA	B-cell non-Hodgkin lymphoma	B-cell
	NA	Permeative dissolution with preserved skull contour	NA	Yes	NA	B-cell non-Hodgkin lymphoma	B-cell
	NA	Permeative dissolution with preserved skull contour	NA	Yes	NA	B-cell non-Hodgkin lymphoma	B-cell

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Table S2: (*Continued*).

Author year reference	Predominance in tumor extension	Findings of skull bone on CT and/or MRI	Disproportionately small amount of cortical destruction	Dural tail	Invasion of brain	Histology	Lymphocyte subtype
	None	Permeative dissolution with preserved skull contour	Yes	Yes	NA	Diffuse, large B-cell lymphoma	B-cell
Ahmad *et al*.,[3] 2020	Extra	Osteolysis	NA	Yes	Yes	Diffuse, large B-cell lymphoma	B-cell
Nasim *et al*.,[67] 2020	Intra	NA	NA	NA	NA	Low-grade B-cell lymphoma	B-cell

CT: Computed tomography, Extra: Extracranial extension is much larger than intracranial extension, Intra: Intracranial extension is much larger than extracranial extension, Both: Intra- and extracranial extensions are of similar size, MRI: Magnetic resonance imaging, NA: Not available