

## Castleman's Disease in a Child Presenting with a Partly Mineralized Solitary Meningeal Mass

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### Key Words

Castleman's disease · Intracranial disease · Childhood · Meningioma · Myeloid and plasmacytoid dendritic cells · Human herpes virus-8

### Abstract

We report a case of solitary intracranial childhood Castleman's disease (CD) presenting with a sudden onset of partial seizures due to a meningeal and cortical mass lesion. The patient was a previously healthy 8-year-old girl who developed a new onset of simple partial seizures with motor signs. On physical examination, she was neurologically intact. Other findings included low-grade fever, mild microcytic anemia and lymphopenia. Magnetic resonance imaging (MRI) of the brain revealed a left posterior parietal, partly mineralized, contrast-enhancing meningeal mass with cortical invasion and adjacent white matter edema. A complete surgical resection of the dural-based component and a subtotal resection of the adherent, invasive cortical lesion were performed. Pathohistology and flow cytometry of the dural-based lesion disclosed a hyaline-vascular type of CD with striking proliferation of polyclonal B lymphocytes, scattered plasma cells and extensive multifocal cortical mineralization. At the 6-month follow-up, the patient was sei-

zure free on antiepileptics and had returned to normal daily activities. MRI showed no residual lesion, and a workup for systemic disease was negative.

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### Introduction

Castleman's disease (CD) is a rare, pathologically and clinically well-defined, albeit heterogenous, nonneoplastic reactive lymphoproliferative disorder of uncertain pathogenesis [1–4]. Originally reported in 1956 as mediastinal giant lymph node hyperplasia [5], and synonymously known as angiofollicular lymph node hyperplasia, CD has since emerged as having two distinctive pathohistological and clinical forms [1–4]. One is a more common, hyaline-vascular type of CD (HVCD), usually presenting as a surgically curable, single-lymph nodal or (rarely) extranodal, contrast-enhancing mass [1–3]. The other, plasma cell type of CD (PCCD), is more frequently a multicentric lymph nodal proliferative disorder that may be associated with significant morbidity and mortality requiring multifaceted treatment [1–4]. Systemic findings may include fever, weight loss, anemia, elevated sedimentation rate, dysgammaglobulinemia, propensity to infections [2] and amyloidosis [6]. Malignancy, such as Hodgkin's disease

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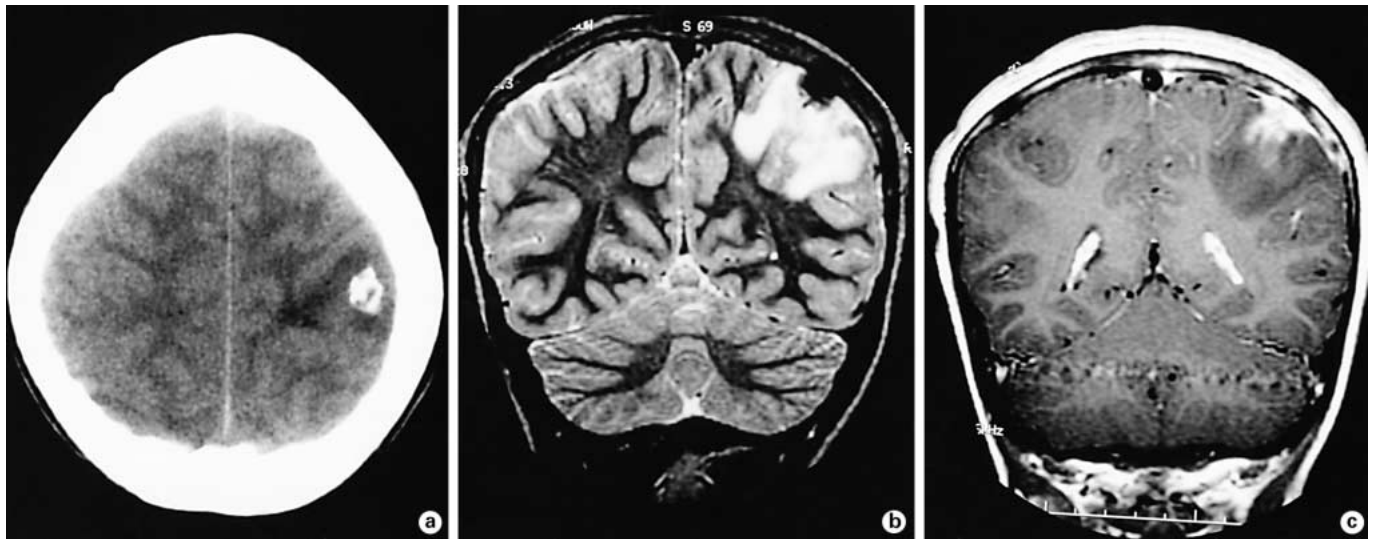


Fig. 1. a Unenhanced axial CT. There is a mineralized nodule within the left parietal cortex with hypodense vasogenic edema in the adjacent white matter. b T2-weighted coronal MRI. The dural-based mass is hypointense, with an associated hyperintense signal in the adjacent brain. c T1-weighted contrast-enhanced coronal MRI. There is extensive meningeal and adjacent focal cortical enhancement with associated hypointense brain edema.

and non-Hodgkin's lymphoma [2, 7], or vascular-mesenchymal neoplasia [8], may occur in association with both types of CD; this is exceptionally rare in HVCD in general [1, 2, 8], and in childhood CD in particular [1, 9, 10].

Primary extranodal, monocentric systemic CD is rare [1–4, 9, 10]. Cases of solitary intracranial-meningeal [11–13] and spinal-epidural [14–16] CD have been reported in a few adults. To the best of our knowledge, there are no similar published cases in children, with the possible exception of a spinal, epidural mass in a 10-year-old boy [17] and CD-like intracranial and systemic findings associated with a chordoid meningioma [18]. Ours may be the first well-documented, combined clinical, radiological and pathological report of localized, meningeal-cortical HVCD occurring in a child.

## Case Report

### *Clinical Findings*

A previously healthy 8-year-old girl presented with a recent onset of repeated brief episodes of abnormal, uncontrollable movements of her right upper extremity, occasionally accompanied by urinary incontinence. On admission, her general and neurologic examinations were normal. There was a mild fever of up to 100.4 °F that responded to Tylenol. Abnormal laboratory tests included: low hemoglobin of 9.0–9.5 g/dl and hematocrit of 28.9–29.5%; likewise, mean corpuscular volume (MCV), mean corpuscular hemoglobin

(MCH), and mean corpuscular hemoglobin concentration (MCHC) were all low at 65.8  $\mu\text{m}^3$ , 20.4 pg and 31.0%, respectively. A white blood cell count range was 6.7–11.7  $\times 10^3$  cells/ml with as many as 89.9% neutrophils, unusually low lymphocyte (6.2%) and monocytes counts (3.9%) and a somewhat lower than normal mean platelet volume, although their number was in the normal range. The patient was placed on Decadron, Tegretol, Zantac, Tylenol and morphine.

### *Imaging*

Computed tomography (CT) (fig. 1a) and magnetic resonance imaging (MRI) (fig. 1b, c) of the brain demonstrated an enhancing, partly mineralized intra- and extraaxial left parietal mass, with associated cerebral edema.

### *Surgery*

Left parietal craniotomy was performed with an intraoperative EEG and somatosensory evoked potential (SSEP) monitoring. The dural-based mass could be grossly visualized upon removal of the bone flap; this was excised entirely and separated from the underlying leptomeningeal-cortical component of the lesion. Under the guidance of a microscope, the cortical lesion was subtotaly resected in a piecemeal fashion using a microsurgical technique, and the visible remnants were bipolarized. Intraoperative EEG and SSEP monitoring showed no changes during or after resection.

### *Postoperative Course*

Immediately following surgery, the patient remained neurologically intact and seizure free. The chest and abdominal CT scans failed to disclose any evidence of a systemic disease. She was discharged home 2 days after surgery on Tegretol and a Decadron taper. After 6 months of follow-up, the patient remains seizure free on

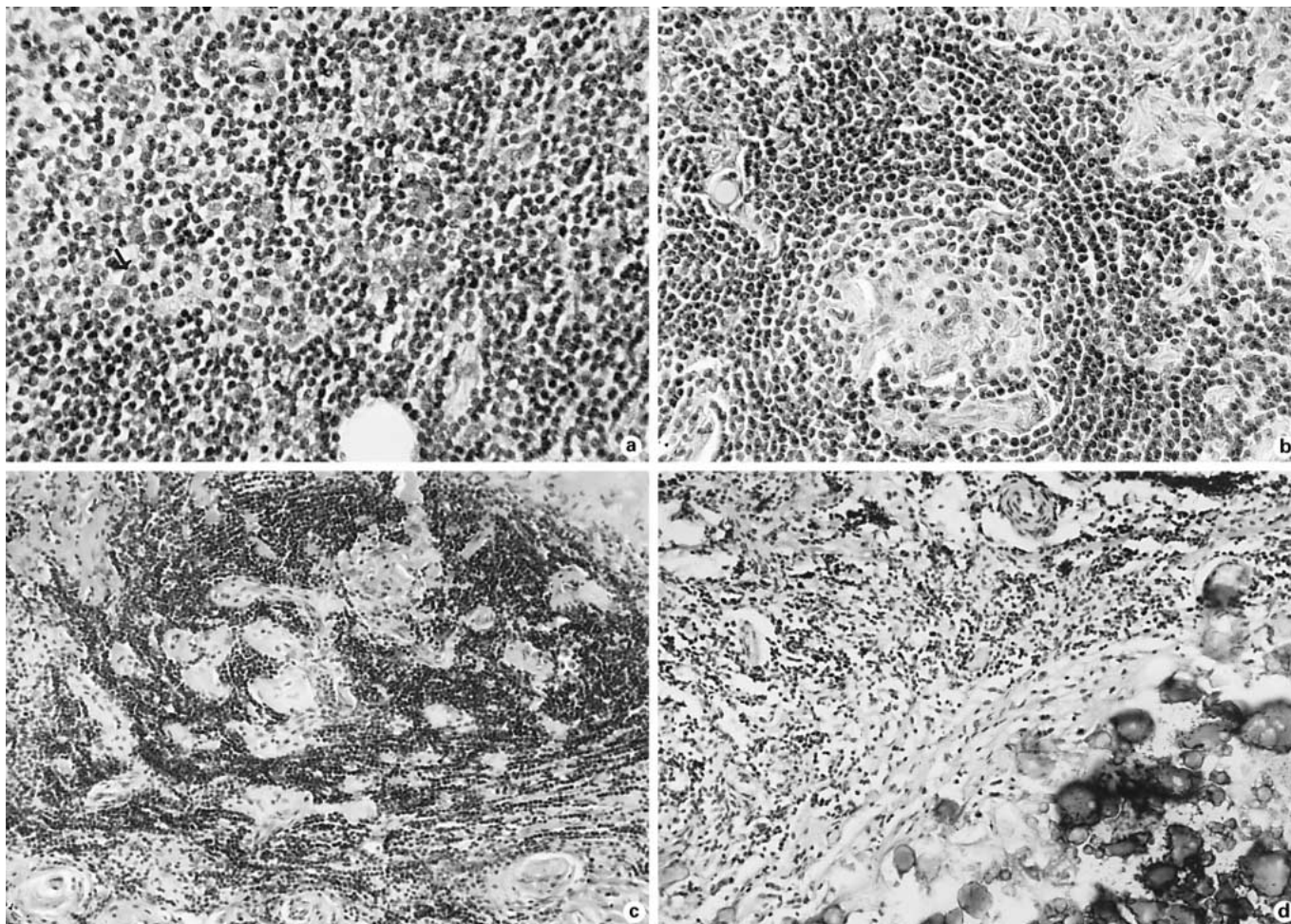


Fig. 2. Hematoxylin and eosin-stained dural-based (a–c) and leptomeningeal-brain tissue (d) sections. a Large solid sheaths of well-differentiated lymphoid cells; in contrast, plasma cells (arrow) appear singly or in small clusters. b Small, partly fibrosed germinal center, surrounded by multiple concentric layers of mature lymphocytes. c Lymphoid cells intertwined with bundles of collagen and thick-walled hyalinized blood vessels. d Fibrous-lymphoid tissue obliterating the subarachnoidal space and leptomeninges merges imperceptibly with multifocal mineralization of the underlying cortex (lower right). Original magnifications: A, B:  $\times 400$ ; C, D:  $\times 250$ .

Tegretol 100 mg twice a day. She has normal strength and has resumed her normal daily activities. MRI revealed no residual mass.

#### Pathology

The dural lesion contained the brunt of the diagnostic pathology; it was a grossly lobulated whitish-tan, rubbery-hard plaque-like mass ( $2.5 \times 2.0 \times 0.7$  cm) that merged imperceptibly with the surrounding 0.5- to 1.0-cm-wide segment of grossly normal dural membrane. The intraoperative hematoxylin and eosin-stained frozen section, multiple permanent sections obtained from paraffin-embedded tissue, as well as Giemsa-stained touch preparations were all dominated by the same densely cellular sheaths of well-differentiated, small lym-

phoid cells, scattered plasmacytoid cells (fig. 2a) and rare clusters of cells with large irregular nuclei, delicate chromatin and inconspicuous cytoplasm. Also present were haphazardly disposed small, partly fibrosed germinal centers, surrounded by multiple concentric layers of mature lymphocytes arranged in a pattern reminiscent of 'onion skinning' (fig. 2b). The cellular elements were focally intertwined with bundles of hyalinized collagen and numerous thick-walled hyalinized blood vessels (fig. 2c). An exhaustive light microscopic examination of the entire specimen, and epithelial membrane antigen immunostaining of the representative tissue sections, failed to demonstrate the presence of proliferating meningeothelial cells.

The cortical lesion consisted of nonspecific, fibrous-lymphoid tissue obliterating the subarachnoidal space, blood vessels and leptome-

ninges. This was associated with focal, extensive dystrophic mineralization of the underlying cortex (fig. 2d), neuronal loss, reactive astrocytosis and perivascular inflammatory infiltrates.

#### *Special Studies*

Flow cytometry of a small fragment of the dural-based specimen disclosed a predominance (70%) of distinctly polyclonal B lymphocytes and plasma cells (kappa:lambda ratio 2:1); the remaining 30% of cells were T lymphocytes with an adequate CD4+/CD8+ ratio of 2:1. Further analysis of T and B cell gene rearrangement revealed no monoclonal population of any cell types. Frozen tissue samples were negative for the presence of Epstein-Barr virus and cytomegalovirus DNA by ultraquant technique; polymerase chain reaction (PCR) analysis did detect human herpes virus-8 (HHV-8) DNA in unspecified cell types (viral tests were performed by Specialty Laboratories, Santa Monica, Calif., USA).

#### Discussion

Our comprehensive study of a contrast-enhancing meningeal mass, surgically removed from an 8-year-old girl with a recent onset of partial seizures, demonstrated typical HVCD. The brunt of the lesion was within the dura and to a lesser extent the leptomeninges; adjacent cerebral cortex contained extensive mineralization. The principal cellular constituents, lymphocytes and plasma cells, were universally polyclonal. Such absence of monoclonal neoplastic cells is indicative of a reactive lymphoplasmacellular proliferative process, and this has previously been substantiated in most adult and childhood cases of CD [10, 19].

Furthermore, our patient's lesion tested positive for HHV-8 DNA, synonymously known as Kaposi's sarcoma-associated herpesvirus. Viral particles of uncertain type have been ultrastructurally demonstrated in one case of meningeal CD [12]; in contrast, PCR was negative for HHV-8 sequences in this [12] and nine other cases of solitary extracranial HVCD [20]. However, varied viruses are known to occur more commonly in association with multicentric PCCD [21]. This is especially true of HHV-8, whose DNA sequences have been demonstrated in a substantial number of CD cases [21–24] and in all tested patients with CD occurring in the setting of HIV positivity [23, 25]. The intracellular presence of a viral genome does not necessarily establish its causal relationship with the underlying disease [22]; nevertheless, some suggest that HHV-8 could play an etiological role in multicentric PCCD [23, 24].

The feasibility of a viral etiology of CD notwithstanding, the pathogenesis of this disorder is yet to be fully elucidated; it seems complex, presumably multifactorial and likely different in HVCD from that of PCCD [1–4, 9, 10,

24, 26]. For instance, there are some indications that overproduction of the cytokine interleukin-6, associated with a global activation of the immune system, could play a significant role in PCCD [27, 28]. Moreover, chromosomal abnormalities that were detected in a small number of PCCD cases have been attributed to the lymphoid cells [29]; this may be one of the reasons for a sporadic association of PCCD with lymphoid tissue malignancies [2, 4, 7, 30]. In contrast, a clonal karyotypic aberration, demonstrated in a single case of solitary HVCD, was thought to have originated from the stromal cells [20]. This, coupled with a repeatedly documented proliferative activity of the follicular dendritic and other stromal cells in a significant percentage of HVCD cases [20, 31], could explain the rare occurrence of angiomyoid and follicular dendritic cell proliferative and malignant [32–34] lesions in association with HVCD.

Until recently, the central nervous system (CNS) and meninges were considered immunoprivileged and thus devoid of antigen-presenting dendritic cells (DC). In 1999, McMenamin [35] demonstrated a large number of DC in the normal rat dura, leptomeninges and choroid plexus. Subsequently, both myeloid and plasmacytoid DC were detected in the CSF of humans with primary inflammatory disorders of the CNS [36], and in the brain tissue of mice with infectious and autoimmune encephalitis [37].

Both systemic HVCD and PCCD, be they lymph nodal, extranodal, mono- or multicentric mass lesions, with and without hematologic abnormalities, occur at all ages [1]. Although pediatric CD is monocentric in 87% of cases, it is otherwise clinically and pathologically indistinguishable from that in adults [1, 9]. This includes the following: rare solely extra-lymph nodal location, relatively common association with fever, elevated sedimentation rate, anemia, hypergammaglobulinemia and growth retardation in children. Additionally, there is usually complete and permanent recovery, including normalization of all hematologic abnormalities, after gross total resection of solitary lesions [1–3, 9, 10].

Intracranially, lymphoid tissue aggregates, morphologically similar to those of CD of unspecified type, have occasionally been observed in association with chordoid subtype or classical meningioma [18, 38–40]. Although chordoid and other types of meningiomas are rare in childhood, at least nine youngsters below 20 years of age have been reported to have had a chordoid meningioma admixed with massive polyclonal lymphoplasmacellular infiltrates and compounded with systemic manifestations of CD, most commonly anemia that responded favorably

Table 1. Clinical and radiographic findings in solitary intracranial and spinal CD cases

Case No.	Age, years gender	Clinical presentation	CT/MRI location	BI-SCI/surgical procedure	Report
1	30/F	seizures and ICP	right posterior fossa DL	BI = no GTR, XRT	Lacombe et al. [43], 1983
2	25/M	partial motor seizures	CT: right parietal DL	BI = ? GTR	Severson et al. [11], 1988
3	82/F	partial motor seizures	CT: right parietal DL mass and focal brain edema	BI = no GTR	Severson et al. [11], 1988
4	63/F	right hemianopsia and generalized seizures	CT: left occipital DL mass and focal brain edema	BI = no ? GTR	Severson et al. [11], 1988
5	63/F	focal neurological signs	CT: bilateral frontal enhancing DL mass and focal brain edema	BI = ? PR, XRT	Gianaris et al. [44], 1989
6	10/M	leg weakness, headache and fever	MRI: enhancing epidural mass at C6–T2	SCI = no PR	Alper et al. [17], 1996
7	47/F	headache and seizures	CT: left frontal enhancing DL mass and focal brain edema	BI = yes PR, XRT	Gulati et al. [12], 1998
8	62/F	focal neurological signs	MRI: left temporal DL enhancing mass and focal brain edema	BI = no GTR	Hashimoto et al. [13], 1999
9	34/M	leg weakness	MRI: T2-hypointense mass at T2–T3	SCI = no GTR	Lee et al. [14], 2002
10	44/F	leg weakness	MRI: enhancing epidural mass at T3–T5	SCI = no GTR	Kachur et al. [15], 2002
11	54/M	leg weakness and back pain	MRI: enhancing epidural mass at T8–T11	SCI = no GTR	Eisenstat et al. [16], 2002
12	8/F	partial motor seizures	MRI: left parietal DL enhancing/calcified mass and focal brain edema	BI = yes GTR	present case

ICP = Increased intracranial pressure; DL = dural-leptomeningeal; GTR = gross total removal; BI-SCI = brain invasion-spinal cord invasion; XRT = X-ray treatment; PR = partial removal.

to the surgical removal of intracranial lesions [18, 41, 42].

Even though our patient's anemia subsided after surgery, her meningeal lesion contained neither morphologically typical meningioma nor epithelial membrane antigen-immunoreactive cells (a marker for meningotheial-arachnoidal fibroblasts). Its morphology, however, was highly characteristic of a solitary intracranial CD, not unlike that previously reported in adult patients [11–13, 43, 44]. Moreover, with the exception of paralesional mineralization and hematologic abnormalities, our patient's clinical and radiographic findings are shared by other intracranial CD cases as listed in table 1. Jointly, they had a supratentorial meningioma-like lesion, with varied clinical manifestations of underlying brain damage, including partial seizures, focal neurological signs and/or peritumoral brain edema. Mineralization, which was confined to the underlying brain tissue in our patient,

has not been observed in any other intracranial cases, although it was said to have been abundant enough in at least three HVCD systemic cases as to be visible by a roentgenogram [1]. Most importantly, as previously reported in systemic cases of solitary CD [1–3, 9, 10], all but one of the intracranial CD lesions were amenable to gross total resection (table 1), which led to complete patient recovery. Long-term follow-up, however, is not available for any of the intracranial [11–13, 43, 44] or spinal epidural CD cases [14–17].

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