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# CEREBELLAR LIPONEUROCYTOMA IN THE LAST DECADE: A COMPREHENSIVE CASE REVIEW

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**ABSTRACT**

**Introduction.** Tumors of the central nervous system (CNS) are relatively uncommon when compared with neoplasms arising in other organs and tissues of the human body. Among these rare entities, cerebellar liponeurocytoma represents one of the least frequently encountered tumor types. This neoplasm was originally described in 1978 by Bechtel and colleagues, who referred to it as a “mixed mesenchymal and neuroectodermal tumor” due to its unusual histological characteristics combining neuronal and lipomatous differentiation. Since that time, only a limited number of cases have been reported in the literature, underlining its exceptional rarity. In subsequent decades, advances in neuropathology and molecular diagnostics have allowed for more precise recognition of this lesion as a distinct clinicopathological entity. Within the World Health Organization (WHO) classification of tumors of the central nervous system published in 2016, cerebellar liponeurocytoma was formally designated as a grade II tumor. This reflects its generally indolent biological behavior but also acknowledges the significant potential for local recurrence following treatment.

**Aim of the Study.** The aim of the study was to collect data about symptoms and immunoreactivity of cerebellar liponeurocytoma from published cases between 2014 and 2024.

**Materials and Methods.** PubMed and Scopus databases were used to search for cerebellar liponeurocytoma cases.

**Result.** Analyzing data from 32 cases of cerebellar liponeurocytoma, 11 men (34.4%) and 21 women (65.6%) were affected. The mean age was  $42.9 \pm 18.17$  years. The youngest person was 6 years old and the oldest was 77. Two cases concerned patients under 18 years old. The most common tumor location was the posterior fossa (25/32 cases, 78.12%), followed by the ventricle area (4 cases, 12.5%) and the frontal lobe (1 case). The most common symptoms were headache (26/32, 81.25%), vomiting and nausea (15/32, 46.9%), gait disturbance (14/32, 44%), ataxia (3/32, 9.4%), paresthesia (3/32, 9.4%), and urge incontinence (1 case).

**Conclusion.** Cerebellar liponeurocytoma is a rare but distinctive tumor of the central nervous system. The usual symptoms arise from the tumor's typical location in the cerebellum. Its clinical course, with atypical features, may pose significant management challenges. Further research into the tumor's molecular and genetic characteristics is needed to provide insights into optimizing management strategies and improving patient outcomes.

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**KEYWORDS**

Cerebellar Liponeurocytoma, Liponeurocytoma, Neurocytoma

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**Introduction**

Tumors occurring in the central nervous system are uncommon in comparison to other types of human tumors. They are associated with high mortality, particularly among patients under 18 years old.[1] Data from the Central Brain Tumor Registry of the United States indicate that, in the period from 2016 to 2020, the incidence rate of central nervous system tumors was 24.83 per 100,000 population.[2]

Cerebellar liponeurocytoma is among the rarest of these CNS tumors. It was first described in 1978 by Bechtel et al. as a “mixed mesenchymal and neuroectodermal tumor.”[3] This tumor was previously incorrectly defined as lipidized medulloblastoma, medulloctoma, lipomatous medulloblastoma, or lipidized mature neuroectodermal tumor.[39,40,41] In 2000, the WHO classified cerebellar liponeurocytoma as a grade I glioneuronal tumor.[4] According to the 2016 WHO classification, cerebellar liponeurocytoma was reclassified as grade II due to the risk of local recurrence.[5]

**Epidemiology**

This tumor usually appears in the fifth decade of life, although it has been documented in patients as young as 4 and as old as 77.[17,42] Analyzing data from 32 cases of cerebellar liponeurocytoma, 11 men (34.4%) and 21 women (65.6%) were affected, with a mean age of  $42.9 \pm 18.17$  years. The youngest person was 6 years old and the oldest was 77; 2 cases involved patients under 18 years old.[Table 1]

### Symptoms

The usual symptoms arise from the tumor's typical location in the cerebellum. Patients report symptoms including headache, vomiting, nausea, dizziness, ataxia, loss of coordination, walking difficulties, and falls. The flow of cerebrospinal fluid may be reduced by the tumor, leading to hydrocephalus[15,26,28]. Analyzing data from 32 cases, the most common symptoms were headache (26/32, 81.25%), vomiting and nausea (15/32, 46.9%), gait disturbance (14/32, 44%), ataxia (3/32, 9.4%), paresthesia (3/32, 9.4%), and urge incontinence (1 case).[Table 1]

**Table 1.**

	Author	Age	Sex	Location	Symptoms
1	Karabagli et al.[11] 2014	34	M	Third ventricle	Headache
2	Takami et al.[8] 2015	59	M	Vermis	Headaches, Dizziness
3	Kakkar et al.[12] 2015	35	M	Left cerebellar hemisphere	Headaches, Vomiting, Gait imbalance
4	Wang et al.[13] 2016	45	F	Right cerebellar hemisphere	Headaches, Gait disturbance, Paresthesia
5	Wolf et al.[14] 2016	37	F	Superior cerebellar hemisphere and vermis	Headaches, Paresthesia
6	Nzegwu et al.[15] 2016	6	F	Left cerebellar hemisphere	Headaches and vomiting, Gait disturbance
7	Pikis et al.[16] 2016	72	F	Right cerebellar hemisphere	Headaches, Gait disturbance
8	Hermann et al.[17] 2017	77	F	Posterior fossa	Headaches, Dizziness, Nausea
9	Sivaraju et al.[18] 2017	37	M	Left Cerebellar hemisphere	Headaches, Vomiting, Gait unsteadiness
10	Tucker et. Al[19] 2017	41	F	Right cerebellar hemisphere	Headaches, Nausea
11	Xu et al.[20] 2017	29	M	Right lateral ventricle	Headaches, Vomiting
		48	F	Left cerebellar hemisphere	Headaches, Paresthesia
12	Cai et al.[21] 2018	11	M	Right frontal lobe	Headaches
13	Chiaromonte et al.[22] 2018	35	F	Right cerebellar hemisphere	Headaches, Vomiting, Dizziness
14	Gembruch et al.[23] 2018	39	M	Right cerebellar hemisphere	Headache, Nausea
15	Hamzaoglu et al.[24] 2018	55	F	Posterior fossa	Headaches
16	Khatri et al.[25] 2018	36	F	Left cerebellar hemisphere + Right cerebellar hemisphere multifocal	Headache, Dizziness, Gait disturbance
17	Deora et al.[26] 2019	55	M	Fourth ventricle	Headache, Vomiting, Visual blurring
18	Linsenmann et al.[27] 2019	59	F	Vermis	Headache, Vertigo, Gait disturbance
19	Mousavinejad et al.[28] 2019	27	F	Right CPA	Hearing loss, Ataxia
20	Abuzneid et al.[29] 2021	50	F	Left cerebellar hemisphere	Gait disturbance, Vomiting
21	Hirono et al.[30] 2021	44	F	Vermis	Headache, Nausea, Gait disturbance, Unsteadiness, Low back pain
22	Wang et al.[31] 2021	70	F	Vermis + Temporal lobe	Nausea, Vomiting, Hear loss, Gait disturbance
23	Al-Umran et al.[32] 2021	24	M	Left lateral ventricle	Headache
24	Shen et al.[33] 2021	29	F	Both lateral ventricles	Headache
25	Dong et al.[34] 2021	5	M	Fourth ventricle and vermis	Headache, Nausea, Vomiting, Dizziness, Dysphoria, Blurring vision
26	Hadelsberg et al.[35] 2022	61	F	Fourth ventricle	Dizziness, Urge incontinence
27	Borni et al.[36] 2022	44	F	Vermis	Vomiting, Headache, Blurry vision, Gait disturbance
28	Ghedira et al.[10] 2023	59	F	Left cerebellar hemispere and right cerebellar hemisphere	Headache, Gait disturbance
		68	F	Left cerebellar hemisphere and vermis	Dizziness, Gait disturbance
29	Mulone et al.[37] 2024	31	F	Left cerebellar hemisphere	Headache, Dizziness
30	Chaouche et al.[38] 2024	52	M	Left cerebellar hemisphere	Headache, Nausea, Vomiting, Gait disturbance

CPA - Cerebellopontine angle ; M- male ; F - female

### Pathohistology

Histologically, cerebellar liponeurocytomas are characterized by the presence of both neuronal and adipose tissue components. Ki-67/MIB-1 was described in 30 cases, with a mean value of  $4.05\% \pm 3.05$ . Neoplasm cells were immunopositive for synaptophysin (26/32 cases, 81.25%), glial fibrillary acidic protein (GFAP) (15/24 cases, 62.5%), Neuron-Specific Enolase (NSE) (10/10 cases, 100%), Microtubule-Associated Protein 2 (MAP-2) (8/8 cases, 100%), chromogranin A (2/4 cases, 50%), S-100 (6/8 cases, 75%), and Hexaribonucleotide Binding Protein-3 (NeuN) (10/11 cases, 90.9%). Tumor cells were mostly immunonegative for neurofilament (NF) (2/9 cases, 22.2%), P53 (2/7 cases, 28.6%), Epithelial Membrane Antigen (EMA) (1/10 cases, 10%), desmin (2/5 cases, 40%), Oligodendrocyte Transcription Factor 2 (OLIG-2) (3/11 cases, 27.3%), and Isocitrate Dehydrogenase 1 (IDH-1) (1/4 cases, 25%). [Table 2 is included in Appendix 1.]

Neuron-Specific Enolase(NSE)	10/10(100%)
Microtubule Associated Protein 2(MAP 2)	8/8(100%)
Hexaribonucleotide Binding Protein-3(NeuN)	10/11 (90.9%)
Synaptophysin	26/32 (81.25%)
S-100	6/8(75%)
Glial fibrillary acid	15/24(62.5%)
Chromogranin A	2/4(50%)
Desmin	2/5 (40%)
P53	2/7(28.6%)
Oligodendrocyte Transcription Factor 2 (OLIG-2)	3/11(27.3%)
Isocitrate Dehydrogenase 1 (IDH-1)	1/4(25%)
Neurofilament(NF)	2/9(22.2%)
Epithelial Membrane Antigen(EMA)	1/10(10%)

### Radiological Characteristics

The most common tumor location was the posterior fossa (25/32 cases, 78.12%), followed by the ventricle area (4 cases, 12.5%) and the frontal lobe (1 case).[Table 1] Magnetic resonance imaging (MRI) and computed tomography (CT) play an important role in diagnosing cerebellar liponeurocytoma. CT images show a hypodense or isodense mass with focal areas of marked hypodensity corresponding to fat. The absence of fat makes diagnosis more challenging. In addition, MRI T1 images show neoplasm cells that are isointense or hypointense with areas of hyperintensity. By contrast, tumor cells in the T2 projection are slightly hyperintense with focal areas of hyperintensity. Lesions reveal heterogenous contrast enhancement after the application of a contrast agent.[6,9]

### Treatment

Surgical resection represents the most effective treatment for cerebellar liponeurocytoma. Total resection of the tumor is preferred in every case if possible. Postoperative radiotherapy is a point of discussion,[9] but a study by Ghedira et al. suggests that this treatment should be reserved for multifocal lesions or those with a high Ki-67/MIB-1 index.[10]

### Discussion

Cerebellar liponeurocytoma is a rare, benign brain tumor typically located in the posterior fossa. This tumor manifests in patients with a mean age of  $42.9 \pm 18.17$  years. Women seem to be affected more often than men, with a ratio of 1.9:1. The usual symptoms arise from the tumor's typical location in the cerebellum. The most common symptoms were headache (26/32, 81.25%), vomiting and nausea (15/32, 46.9%), and gait disturbance (14/32, 44%). Ataxia and paresthesia were less common. Only one patient suffered from urge incontinence. [Table 1] At times, the tumor can reduce the flow of cerebrospinal fluid, resulting in symptoms resembling obstructive hydrocephalus. Histologically, cerebellar liponeurocytoma consists of small, round tumor cells and adipocytes. In our study, the tumor was mostly immunoreactive to synaptophysin (81.25%), glial fibrillary acidic protein (GFAP) (62.5%), microtubule-associated protein 2 (MAP-2) (100%), Hexaribonucleotide Binding Protein-3 (NeuN) (90.9%), and S-100 (75%). Ki-67/MIB-1 was mainly low, with a mean value of  $4.05\% \pm 3.05$ , but in 2 cases, this parameter was 12%. [Table 2, Appendix 1.] A low Ki-67/MIB-1 index suggests a benign tumor character.

There are no treatment guidelines for cerebellar liponeurocytoma; however, complete tumor resection is considered the gold standard.[9] Additional adjuvant radiotherapy seems to play an important role in preventing tumor recurrence. According to the study by Gembruch et al., tumor resection was mentioned in 72 cases, and total tumor resection was possible in 49 cases (68.05%). Tumor recurrence was not observed in patients treated with total tumor resection and adjuvant radiotherapy.[9] On the other hand, the study by Ghedira et al. suggests reserving postoperative radiotherapy for recurrent lesions or tumors with a high Ki-67 index. Differential diagnosis includes other central nervous system tumors, such as central neurocytomas or medulloblastomas.[10] Horstmann et al. collected 20 samples of cerebellar liponeurocytoma, for which genetic profiles were established. This research revealed that proteins like cadherin 5, cyclic-dependent kinase 6, neurotrophin 3, and erbB3 proto-oncogene were expressed in cerebellar liponeurocytoma cells but not in medulloblastoma or central neurocytoma. Additionally, proteins like adenomatosis polyposis coli-binding protein, CD9 antigen, tissue inhibitor of metalloproteinase 3, retinoblastoma-binding protein 4, growth arrest & DNA damage-inducible protein, insulin growth factor binding protein 5, and vascular endothelial growth factor 1 were present in cerebellar liponeurocytoma cells but not in the cerebellum. Another way to distinguish between healthy brain cells and tumor cells is to measure protein expressions like dual-specificity phosphatase 8, transforming growth factor  $\beta$ , T-cell lymphoma invasion and metastasis I, fatty acid synthase, cytokeratin 8, integrin beta 4, and serine/threonine protein kinase 3, which are expressed by healthy brain tissue of the cerebellum. Moreover, the mentioned study shows another difference between cerebellar liponeurocytoma and medulloblastoma. Isochromosome 17q, which is specific for medulloblastoma, wasn't detected in any cerebellar liponeurocytoma samples.[7] Takami et al. used  $^{18}\text{F}$ -fluorodeoxyglucose (FDG) positron emission tomography to assess the metabolic activity of cerebellar liponeurocytoma. Lower tumor accumulation was seen compared with the normal cerebellar cortex, showing a lesion-to-normal cerebral cortex accumulation ratio of 0.62. On the other hand, the tumor showed higher accumulation on  $^{11}\text{C}$ -methionine PET, with a lesion-to-normal cerebral cortex accumulation ratio of 2.73.[8] The mentioned information may help in the diagnosis of challenging cases of cerebellar liponeurocytoma.

### Conclusions

Cerebellar liponeurocytoma is a rare but distinctive tumor of the central nervous system. The usual symptoms arise from the tumor's typical location in the cerebellum. Its clinical course, with atypical features, may pose significant management challenges. Further research into the tumor's molecular and genetic characteristics is needed to provide insights into optimizing management strategies and improving patient outcomes.

### Disclosure

#### Authors contribution:

Karol Kanon: Conceptualization, Writing - rough preparation, project administration

Oskar Sienkiel: Resources, Data curation

Jan Tomczyk: Investigation, Conceptualization

Mathias Spitaleri: Resources, Project administration

Mateusz Jasiński: Formal analysis, Investigation

Aleksandra Żywicka: Methodology, Formal analysis

Kamila Mozga: Software, Writing- review and editing

Marcin Narloch: Data curation, Writing - rough preparation

Dawid Sewruk: Methodology, Investigation

Michał Szalach: Supervision, Writing- review and editing

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Appendix 1.  
Table 2.

Author	MIB-1	synaptop hysin	Glial fibrillary acidic protein	NSE	Microtubule associated protein 2	NF	Chromogranin A	S100	P53	Epithelial Membrane Antigen	desmin	NeuN	OLIG-2	IDH-1
Karabagli et al.	1.5%	+	nd	+	+	-	-	nd	-	nd	-	nd	nd	nd
Takami et al.	2%	+	-	nd	nd	nd	nd	nd	nd	nd	nd	+	-	nd
Kakkar et al.	3.5%	+	+	+	+	nd	nd	-	-	-	-	+	nd	nd
Ke Wang et al.	5%	+	-	nd	nd	nd	nd	+	nd	-	nd	+	+	nd
Wolf et al.	1%	+	nd	nd	+	nd	nd	nd	-	nd	nd	+	nd	+
Nzegwu et al.	0%	nd	nd	nd	nd	nd	nd	+	nd	nd	+	nd	nd	nd
Pikis et al.	2%	+	+	nd	+	nd	+	nd	-	nd	nd	+	-	-
Hermann et al.	12%	+	+	+	nd	+	+	nd	-	-	-	nd	+	-
Sivaraju et al.	6%	+	+	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
Tucker et al.	4%	+	-	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
Xu et al.	3.5%	+	-	+	+	-	nd	+	nd	nd	nd	-	nd	nd
	1.5%	+	-	+	+	-	nd	+	nd	nd	nd	+	nd	nd
Cai et al.	1.5%	+	+	nd	+	nd	nd	nd	nd	nd	nd	+	-	nd
Chiaromonte et al.	6%	nd	+	nd	nd	-	nd	nd	nd	nd	nd	nd	-	nd
Gembruch et al.	2%	+	nd	nd	nd	-	-	nd	nd	nd	nd	+	nd	nd
Hamzaoglu et al.	2%	+	-	+	nd	nd	nd	nd	nd	nd	n	nd	nd	
Khatri et al.	nd	+	+	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
Deora et al.	4.5%	+	-	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
Linsenmann et al.	5%	+	+	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
Mousavinejad et al.	4.5%	+	-	nd	nd	nd	nd	nd	nd	-	nd	nd	nd	nd
Abuzneid et al.	nd	+	+	+	nd	nd	nd	nd	nd	-	nd	nd	nd	nd
Hirono et al.	3.5%	+	-	nd	nd	+	nd	+	nd	-	nd	nd	-	nd
Wang et al.	7.5%	nd	+	nd	nd	nd	nd	nd	nd	+	nd	+	+	nd
Al-Umran et al.	10%	+	nd	nd	nd	nd	nd	nd	+	nd	nd	nd	nd	nd
Shen et al.	5%	+	+	+	nd	-	nd	nd	+	-	nd	nd	-	nd
Dong et al.	5%	+	+	+	+	-	nd	-	nd	-	nd	nd	-	-
Hadelsberg et al.	12.5%	nd	+	nd	nd	nd	nd	+	nd	nd	nd	nd	nd	nd
Borni et al.	5%	+	+	+	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
Ghedira et al.	1%	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
	1%	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd	nd
Mulone et al.	2%	+	nd	nd	nd	nd	nd	nd	nd	-	nd	+	-	nd
Chaouche et al.	5%	+	+	nd	nd	nd	nd	nd	nd	nd	+	nd	nd	nd
nd - no data														