

Cerebellar Liponeurocytoma: Case report of a rare tumor and a brief review of the literature

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Abstract

Cerebellar liponeurocytoma (cLNC) is a rare WHO (World Health Organization) Grade II neuronal tumor characterized by neurocytic differentiation with lipomatous components and typically occurring in the posterior fossa of adults. We present a case of a 51-year-old female with a six-month history of progressive gait instability and incoordination, and subsequently developed morning headaches, nausea, vomiting, and dysarthria. She had signs of increased intracranial pressure. Neurological exams showed truncal ataxia, bilateral dysmetria, dysdiadochokinesia, and papilledema. Her MRI showed a 4.5 x 3.8 x 4.2 cm well-circumscribed, heterogeneous mass located in the right cerebellar hemisphere. Areas of T1 hyperintensity suggestive of fat were observed, accompanied by mild heterogeneous enhancement. The mass compressed the fourth ventricle, resulting in early hydrocephalus. She underwent suboccipital craniotomy with gross total tumor resection. Her surgical pathology revealed a tumor volume of approximately 35% lipomatous component, and low mitotic activity was noted in the uniform round neurocytic cells. Immunohistochemistry (IHC) studies revealed strong positivity for synaptophysin, NeuN, and NSE, with a low Ki-67 of 4%, and thus, the diagnosis of cerebellar liponeurocytoma was confirmed. Three years postoperatively, the patient had notable neurological improvement, complete resolution of intracranial hypertension, and no signs of recurrence. This case illustrates the need to include cLNC in the differential diagnosis of fat-containing posterior fossa masses in adults and emphasizes the improved prognosis with gross total resection.

Keywords: Cerebellar liponeurocytoma; Neurocytic; Lipomatous; Immunohistochemistry; Suboccipital craniotomy; Structured long-term surveillance plan

1. Introduction

Cerebellar liponeurocytoma (cLNC) is a rare adult brain tumor typically found in the posterior fossa. It was first documented in 1978 and was recently included in the WHO classification of central nervous system tumors as grade II due to its low potential for malignancy and slow growth. [1] CNS tumors of this type are rare but have been increasingly documented in the middle-aged population, classically seen in the 4th to 6th decades of life. [2] The most common symptoms associated with posterior fossa mass effect are vomiting, vertigo, instability, and increased intracranial pressure. [3] The rarity and nonspecific presentation of cLNC can lead to misdiagnosis as more common conditions or other tumors that are in the posterior fossa.

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Cerebellar liponeurocytomas typically appear on MRI as well-defined, heterogeneous masses with focal T1 hyperintensity, reflecting fatty differentiation within the tumor. [4] Histologically, there are monomorphic neurocytic cells that have low mitotic and lipomatous elements of varying degrees that are positive in immunohistochemistry (IHC) for neuronal markers, such as synaptophysin and neuron-specific enolase. [2] The differential diagnosis includes medulloblastoma and oligodendroglioma, distinct entities with different prognoses and treatment approaches. Gross total surgical resection remains the most effective treatment and is associated with long-term positive outcomes, although recurrence is possible with incomplete resection. [3] Since cLNC is rare, it is crucial to understand it to make an accurate diagnosis and implement the most effective treatment.

2. Case presentation

2.1. Clinical Presentation

A 51-year-old female was referred to the neurology department, presenting with the development of gait instability and lack of coordination over 6 months. Her symptoms were first explained by stress and an inability to maintain sufficient sleep. The patient initially deferred medical attention, using a walking cane instead, hoping that the symptoms would go away. In the last 2 months, she started getting more severe morning headaches, nausea, and vomiting. Her family noticed progressive dysarthria and irregular fine motor skills, including difficulty writing and buttoning clothes. The patient reported dizziness, particularly with changes in position, and was never perfectly steady on her feet. She denied visual field alterations, hearing impairment, or extremity weakness. She initially consulted her primary care physician, who thought they were due to benign positional vertigo, and prescribed meclizine with little success.

She presented to the emergency department complaining of a severe headache with projectile vomiting and temporary confusion, making the family concerned. Her symptoms indicated that she had a posterior fossa pathology that presented evidence of elevated intracranial pressure and cerebellar dysfunction, which should be followed through urgent neuroimaging studies.

2.2. Evaluation: Examination, History, Labs, and Imaging

The physical examination demonstrated signs of typical cerebellar dysfunction. The patient exhibited truncal ataxia and had a wide-based gait and could not walk tandemly. Finger-to-nose examination revealed bilateral dysmetria, which was more severe on the right. There was bilateral impairment of the Heel-to-shin testing, accompanied by past-pointing. Rapid alternating movements (dysdiadochokinesia) were seriously affected in both hands. There was mild scanning-quality dysarthria of the speech. Funduscopic observation revealed bilateral papilledema, indicating high intracranial pressure. Miscellaneous Cranial nerve examinations were normal, extraocular movements intact, and no nystagmus. Her motor strength was 5/5 with normal reflexes, and sensation was preserved. The vital signs revealed hypertension (150/90), likely secondary to elevated intracranial pressure. The patient was anxious with a confused orientation to the person, place, and time.

Laboratory studies, including complete blood count, comprehensive metabolic panel, and coagulation tests, were all normal. Tumor markers (CEA, CA 19-9, AFP) were negative, suggesting that metastatic disease was unlikely. Brain MRI with contrast and without contrast showed a large, well-circumscribed, heterogeneous mass of the right cerebellar hemisphere (4.5 x 3.8 x 4.2 cm). The lesion showed mixed signal intensity, with T1 hyperintense regions indicating fat and T2 heterogeneity. Post-contrast imaging showed mild heterogeneous enhancement. Significant mass effect caused compression of the fourth ventricle with early hydrocephalus. CT imaging confirmed a mixed-density appearance with areas of fat attenuation and soft-tissue components. No calcifications or hemorrhage were present. The presence of a fat-containing cerebellar mass in a middle-aged adult, along with typical imaging characteristics, was a strong indication of liponeurocytoma; however, it could only be confirmed through histopathological examination. Mixed signal intensity and a mild enhancement pattern were the pathognomonic features supporting this diagnosis.

2.3. Surgical Treatment and Tissue Diagnosis

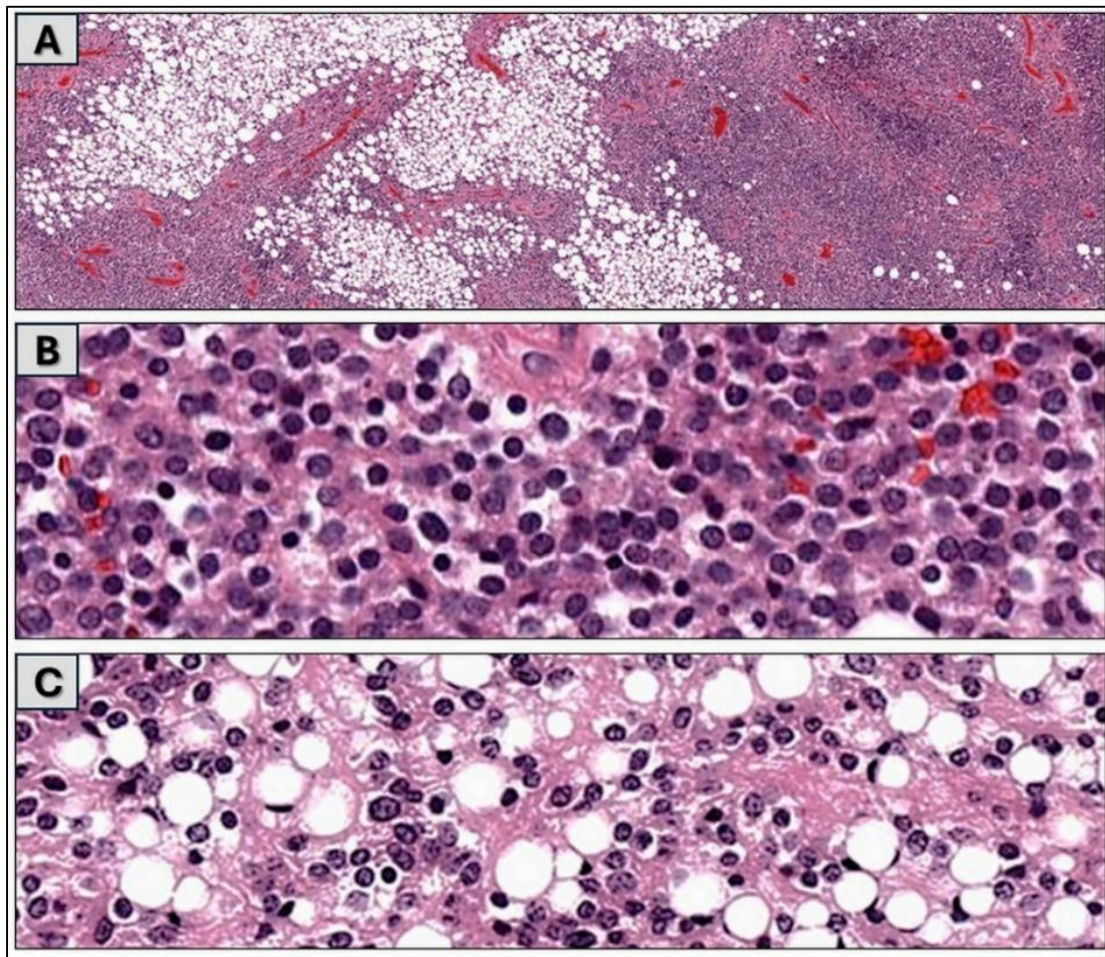
Given the large mass effect and the symptoms of high intracranial pressure, a multidisciplinary tumor board discussion determined that surgery was indicated. The midline approach of the suboccipital craniotomy offered the best access to the cerebellar hemisphere with the least risk to the eloquent parts of the brain. The tumor's well-circumscribed nature enabled gross total resection.

The excised tumor was a well-defined, soft, yellowish mass with scattered traces of solid, grayish tissue. A heterogeneous appearance, with adipose tissue and solid elements, was observed in the cut sections. The tumor was lobulated, with distinct margins in relation to the surrounding cerebellar tissue. Microscopic examination of the tumor

revealed the pathognomonic histological appearance of uniform round neurocytic cells, lacking atypia, accompanied by a component of lipid-laden, fat-like cells. The neurocytes exhibited round to oval nuclei with fine chromatin and low mitotic activity. The lipomatous component accounted for 35% of the tumor volume. There were also occasional neuropil-like areas and occasional ganglion-like cells. Neuropil matrix around vessels simulated perivascular pseudorosettes of ependymoma (Figure 1 A, B, C, D). The neurocytic component was strongly positive for neuronal markers, including synaptophysin, NeuN, and NSE. Staining for GFAP was focally positive. The Ki-67 proliferation index was low (4%), confirming the tumor's benign nature. S-100 protein was focally positive in both components. The tumor cells were negative for Olig2 and EMA. The morphological aspect and IHC profile confirmed the diagnosis of cLNC, WHO Grade II, and molecular studies were not indicated.

With gross total resection, the prognosis for cLNC is excellent. These tumors are WHO Grade II lesions with inherently benign biological behavior and low proliferative potential. Complete excision typically results in a cure with extremely low recurrence rates. No adjuvant therapy was required after gross total resection.

2.4. Outcome and Follow-up



1A: Low power view showing lobules of mixed pattern with sheets of small blue cells (neurocytic component) interspersed with clear, vacuolated areas (fatty/lipidized component). (H&E Stain X20); 1B: High power view showing the neurocytic component with minimal atypia and no mitosis (H&E Stain X40); 1C: High power view showing lipidized component. These cells contain clear, large cytoplasmic bubbles of lipid that indent the nucleus (H&E Stain X40)

Figure 1 Microscopic Examination of the Excised Cerebellar Liponeurocytoma

The primary surgical goal was complete removal of the tumor while preserving normal cerebellar function. Postoperatively, the patient experienced a gradual recovery, with improvement in cerebellar symptoms over the following 6 months. Gait ataxia and coordination difficulties showed significant improvement, with only mild residual deficits, headaches, and signs of increased intracranial pressure resolving completely following tumor removal and normalization of intracranial pressure.

Long-term surveillance was planned, including MRI at 6 months and 1 year, then annually for 5 years, to monitor for rare recurrence. The patient returned to baseline functional status and normal quality of life. During three years of post-surgical treatment, there was no evidence of recurrence or other complications

3. Discussion

3.1. Background (History, epidemiology, risk factors, and WHO classification)

cLNC is a rare, well-differentiated neuronal tumor first described by Bechtel et al. in 1978 as a “lipomatous medulloblastoma” to highlight its better prognosis. [5] Since its initial description, it has been referred to by various names, including neurolipocytoma and lipidized medulloblastoma, reflecting its diagnostic complexity. It was formally recognized as a distinct entity in the 2000 WHO classification as a Grade I tumor. However, owing to increasing reports of recurrence, it was upgraded to a WHO Grade II neoplasm in the 2007 classification, a designation it retains. [6,7]

Epidemiologically, cLNC is exceptionally rare, with fewer than 100 cases reported in the literature. [8] It typically affects adults, with a peak incidence between the fourth and fifth decades of life and shows no significant sex predilection. [9] These tumors usually occur in the cerebellar hemisphere, though they may also involve the vermis. Although considered benign due to their slow growth and low mitotic activity, they have a notable potential for recurrence, which can occur many years after initial treatment, making long-term surveillance essential. [10] No specific genetic, environmental, or syndromic risk factors have been definitively established. However, a familial predisposition with a possible autosomal dominant inheritance pattern has been suggested in some reports. [6]

3.2. Pathogenesis, Pathophysiology

cLNC is an uncommon, WHO Grade 2 glioneuronal tumor primarily affecting the adult cerebellum, characterized by combined neuronal, glial, and adipose differentiation. The cellular origin is suspected to be GABAergic neurons in the cerebellar ventricular zone. [11] These tumors show expression of the transcription factor NEUROG1, unlike the wild-type adult cerebellum, and a significant increase in fatty acid-binding protein 4 (FABP4), which is expressed in adipose tissue, explaining the adipose differentiation [11].

The growth of this tumor is slow and expansive rather than infiltrative, producing a well-circumscribed mass that exerts local mass effect and obstructive hydrocephalus rather than early parenchymal invasion. The increased ICP and progressive cerebellar dysfunction in this patient are consistent with compression of cerebellar circuits and fourth ventricular obstruction by the enlarging lesion. [12] Histologically, the dual population of uniform neurocytic cells and lipidized structures reflects divergent differentiation from a common precursor, as evidenced by strong synaptophysin and GFAP staining. [13]

3.3. Comparative Analysis of Our Case with Existing Literature. (Clinical-radiology-pathology, lab, diagnosis, management, and outcome)

3.3.1. Clinical Presentation

cLNC is an extremely rare tumor of the central nervous system that primarily occurs in adults. Typically described as occurring in adults in the 4th to 6th decades of life. [14,15] Common presentations include symptoms of posterior fossa involvement, such as ataxia, dysarthria, headache, nausea, and signs of raised intracranial pressure due to obstructive hydrocephalus. [16] Our case study describes a 51-year-old female presenting with progressive gait disturbance, truncal ataxia, dysmetria, unstable dysarthria, and signs of increased intracranial pressure, all consistent with the common descriptions of this rare tumor.

Symptoms of cLNC tend to evolve slowly, thereby prompting late or non-diagnostic radiologic evaluation and a prolonged diagnostic period. [15] This was the scenario in the present case. Conservative management of initial symptoms did not reveal any atypical features that might have suggested a more complex tumor, thereby providing a clear representation of the clinical behavior of cLNC, which may be helpful to clinicians managing apparent incidental findings of cLNC.

3.3.2. Diagnostic Workup

Radiologically, cLNC is reported to be a well-defined cerebellar mass with heterogeneous signal and variable fat content. [16] Our patient showed features of a well-defined cerebellar mass with fat within the T1 signal, heterogeneous T2 signal, and moderate contrast enhancement, resulting in mass effect and hydrocephalus. The imaging characteristics

are quite useful in narrowing the differential diagnosis prior to surgery, and a CT scan will show fat attenuation within the mass that supports but is not confirmatory of the diagnosis of cLNC.

The MRI findings, although highly suggestive, were insufficient for radiological diagnosis. A wide differential diagnosis should be considered, including medulloblastoma with lipidized differentiation, ependymoma, central neurocytoma, and cerebellar lipoma. [17] Histopathological examination is mandatory for a definitive diagnosis. The tumor exhibited the characteristic biphasic morphology described in the literature, with homogeneous populations of neurocytes intermingled with lipidized cells. [14] Synaptophysin and NeuN staining were strongly positive, whereas GFAP staining was focally positive. The Ki-67 labelling index was low, and EMA and Olig2 staining were negative. These immunohistochemical characteristics are consistent with those reported previously and help rule out other glial and ependymal tumors. [1, 15, 18]

3.3.3. Management

The surgical technique of a suboccipital craniotomy with total tumor removal, as used in this case, is in accordance with current literature. The necessity of adjuvant radiotherapy is not well established and is therefore generally not recommended after subtotal resection or tumor recurrence. [1] In the current case, the low proliferation index in combination with the fact that the tumor was completely removed also supports the decision not to apply radiotherapy.

3.3.4. Outcome and Follow-up

Most published reports describe excellent outcomes following complete surgical removal of cLNC with excellent survival and low rates of recurrence. [1,15, 18] Our patient showed significant improvement in neurological status postoperatively, with complete resolution of intracranial pressure symptoms and near full recovery from deficits secondary to cerebellar involvement. At three years post-surgery, there has been no sign of tumor recurrence radiologically or clinically. While gross total resection often yields immediate clinical cure, cLNC carries a significant long-term recurrence risk of up to 32% at 10 years, mandating a disciplined, decade-long surveillance protocol regardless of initial surgical success. [15]

4. What Have we learned from this case?

This case provides several practical clinical insights into the diagnosis and management of cerebellar liponeurocytoma. The primary diagnostic lesson is the importance of including rare entities in the differential diagnosis for posterior fossa masses in adults, particularly when imaging reveals fat components. The patient's initial presentation with nonspecific symptoms such as gait instability and headaches, initially attributed to benign causes, is a red flag; progressive cerebellar signs, even if subtle, warrant urgent and thorough neurological investigation, including advanced neuroimaging. This case reinforces the importance of a high index of suspicion to avoid diagnostic delays.

From a management perspective, this report underscores that aggressive gross total resection (GTR) is the cornerstone of treatment and is often curative, leading to significant functional recovery. The patient's favorable outcome post-surgery reaffirms GTR as the primary therapeutic goal. Furthermore, this case highlights the consensus that despite its benign histology and low proliferative index, cLNC requires a structured long-term surveillance plan with serial MRI, as recurrence can occur years after successful resection.

5. Conclusion

Cerebellar liponeurocytoma is a rare WHO Grade II neural tumor that poses a significant diagnostic challenge due to its clinical and radiological overlap with other posterior fossa neoplasms. This case of a 51-year-old female highlights a classic presentation of progressive cerebellar dysfunction and increased intracranial pressure, demonstrating the critical role of timely neuroimaging and definitive histopathological analysis for accurate diagnosis. The presence of lipidized cells on imaging should prompt consideration of this rare entity.

The successful outcome following gross total resection reinforces surgery as the primary treatment modality, capable of providing a favorable prognosis and functional recovery. However, the established risk of recurrence, reported to be as high as 20-32% over 10 years, mandates a disciplined long-term surveillance strategy. This case contributes valuable data to the limited literature, reinforcing the current understanding of cLNC's behavior and affirming the need for vigilance in post-operative management to monitor for potential late recurrence.

Compliance with ethical standards

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All authors make the following declarations:

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Statement of ethical approval

The paper has been sufficiently anonymized to maintain the patient's confidentiality.

Data access statement

All relevant data are included in the paper.

Author contributions

All authors contributed equally to producing this manuscript.

Statement of informed consent

This study was conducted as a retrospective review of archival pathology material collected during routine clinical care. All data were fully de-identified prior to analysis.

References

- [1] Louis DN, Perry A, Wesseling P, Brat DJ, Cree IA, Figarella-Branger D, Hawkins C, Ng HK, Pfister SM, Reifenberger G, Soffietti R. The 2021 WHO classification of tumors of the central nervous system: a summary. *Neuro-oncology*. 2021 Aug 1;23(8):1231-51.
- [2] Jribi A, Dhouib F, Siala W, Fourati N, Daoud J. Cerebellar liponeurocytoma: clinico-epidemiological, pathological, radiological, genetic, therapeutic and evolutionary characteristics—a review of the literature. *Egyptian Journal of Neurosurgery*. 2024 May 28;39(1):39.
- [3] Gembruch O, Junker A, Ahmadipour Y, Sure U, Lemonas E. Cerebellar liponeurocytoma—a rare entity: a case report. *Journal of Medical Case Reports*. 2018 Jun 17;12(1):170.
- [4] Chaouche I, Bouardi NE, Benabderrazik B, Haloua M, Youssef alaoui Lamrani M, Boubbou M, Maaroufi M, Alami B. Cerebellar liponeurocytoma: Rare posterior fossa tumor. *Radiology Case Reports*. 2024 Aug 1;19(8):3382-5.
- [5] Bechtel JT, Patton JM, Takei Y. Mixed mesenchymal and neuroectodermal tumor of the cerebellum. *Acta Neuropathol*. 1978;41(3):255-7.
- [6] Deora H, Prabhuraj AR, Saini J, Yasha TC, Arimappamagan A. Cerebellar Liponeurocytoma: A Rare Fatty Tumor and Its Literature Review. *J Neurosci Rural Pract*. 2019;10(2):360-363.
- [7] de Andrade HR, Fenner F, Vasques VP, et al. Cerebellar liponeurocytoma: illustrative case. *J Neurosurg Case Lessons*. 2025 Mar 17;9(11):CASE24521.
- [8] Sokol Z, Parsells P, Madineni R. Liponeurocytoma: Rare Neoplasm of the Central Nervous System. *Cureus*. 2024 Apr 28;16(4):e59221.

- [9] Ali R, Durrani S, Nathani KR, Jarrah R, Bydon M. Cerebellar Liponeurocytoma: Publication Trends, Scientometrics Analysis, and Critical Review. *World Neurosurg.* 2023;171:e339-e346.
- [10] Shah DS, Sharma H, Patel P, et al. Recurrent liponeurocytoma: A case report and systematic review of the literature. *Surg Neurol Int.* 2022;13:395.
- [11] Anghileri E, Eoli M, Pattera R, Ferroli P, Pollo B, Cuccarini V, Maderna E, Tringali G, Saini M, Salsano E, Finocchiaro G. FABP4 is a candidate marker of cerebellar liponeurocytomas—*Journal of neuro-oncology.* 2012 Jul;108(3):513-9.
- [12] Abuzneid YS, Alzeerelhouseini HIA, Shkokani S, Aqel W, Aldarawish A. Cerebellar liponeurocytoma, a rare tumor: Case report and review of the literature. *Int J Surg Case Rep.* 2021;82:105937.
- [13] Tucker A, Boon-Unge K, McLaughlin N, Ibrahim H, Rao N, Martin N, Everson R, Khanlou N. Cerebellar liponeurocytoma: relevant clinical cytogenetic findings. *Journal of Pathology and Translational Medicine.* 2017 May 1;51(3):335-40.
- [14] Giangaspero F, Cenacchi G, Roncaroli F, et al. Cerebellar liponeurocytoma: a distinct clinicopathological entity. *American Journal of Surgical Pathology.* 1996;20:772–780.
- [15] Radke J, Gehlhaar C, Lenze D, et al. Cerebellar liponeurocytoma: clinical, histological, and molecular characterization. *Acta Neuropathologica.* 2012;124:675–683.
- [16] Koeller KK, Rushing EJ. From the archives of the AFIP: cerebellar neoplasms in adults. *Radiographics.* 2005;25:1609–1633.
- [17] Burger PC, Scheithauer BW. *Tumors of the Central Nervous System.* AFIP Atlas of Tumor Pathology. Washington, DC: Armed Forces Institute of Pathology; 2007.
- [18] Louis DN, Perry A, Wesseling P, et al. The 2021 WHO classification of tumors of the central nervous system. *Neuro-Oncology.* 2021;23:1231–1251.