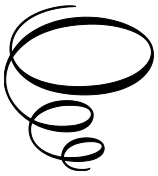
Neuroradiology Spot Diagnosis

Neuroradiology Spot Diagnosis

By

Timo Krings, Ammar Haouimi, Z.H. Choudhuri, Rukhsana Begum and Rabah Bouguelaa

Cambridge Scholars Publishing



Neuroradiology Spot Diagnosis

By Timo Krings, Ammar Haouimi, Z.H. Choudhuri, Rukhsana Begum and Rabah Bouguelaa

This book first published 2025

Cambridge Scholars Publishing

Lady Stephenson Library, Newcastle upon Tyne, NE6 2PA, UK

British Library Cataloguing in Publication Data
A catalogue record for this book is available from the British Library

ISBN: 978-1-0364-4933-9
ISBN (Ebook): 978-1-0364-4934-6

To Our Parents

Heinz
Hiltrud
Timo

Salah
Noua
Ammar

Fazlul
Razia
Choudhuri

Mujibur
Razia
Rukhsana

Ahmed
Rokia
Rabah

Table of Contents

Foreword

Neuroradiology is a dynamic and evolving field that plays a crucial role in the diagnosis and management of neurological disorders. This academic case-based pictorial review offers a unique approach by combining high-quality imaging with real clinical cases. The integration of visuals and case discussions enhances learning and fosters a deeper understanding of complex conditions.

Each case is thoughtfully organized to cover a variety of neurological pathologies, showcasing both common and rare cases. This diversity not only enriches the educational experience but also prepares practitioners for the complexities they may face in their daily practice. The carefully curated images serve as a powerful tool for recognizing key features and improving diagnostic skills.

“Spot Diagnosis: Neuroradiology” authored by Drs. T. Krings, A. Haouimi, Z.H. Choudhuri, R. Begum and R. Bouguelaa is a new edition in a series of case-based textbooks. All authors that received their training in Canada or France, or both, have proven an exemplary record in teaching and education in Neuroimaging including but not limited to the use of online open-source teaching. They are esteemed experts who bring their extensive knowledge and experience to the forefront. Their insights into the clinical relevance of each case provide context that is essential for effective diagnosis and patient management.

As you navigate through the pages, you will find that this book is more than just a pictorial guide; it is a comprehensive resource that encourages critical thinking and lifelong learning. I hope it inspires our colleagues to explore the fascinating world of neuroradiology with curiosity and dedication. This book will be highly appreciated by residents in Radiology, Diagnostic Radiologists, Fellows in Neuroradiology as well as professionals with background in Neurology and Neurosurgery. May this work serve as a valuable companion in our professional journey.

Prof. Karel G. ter Brugge MD, FRCP, FCAR

Professor Emeritus,
Department of Medical Imaging, University of Toronto.

Preface

There are numerous excellent textbooks available in Neuroradiology. This book does not aim to replace these nor is it meant to be complete in the sense of covering the myriad diseases a radiologist may encounter in their daily practice. Instead, the purpose of this book is to provide an easily digestible way to both imaging and clinical features leading to diagnosis including the top differential diagnostic considerations. It is primarily intended for the beginners and can be used as a handy guide for radiology, neurology and neurosurgery residents in preparation for exams or just to browse through both common and uncommon cases. We hope that it may also be helpful for referring physicians and those preparing for board and fellowship examinations.

A vast majority of patients with neurological symptoms end-up with some form of cross-sectional imaging. The interpretation of images can be complicated and necessitates meticulous review. But at times the combination of typical imaging findings and classic clinical symptoms, a "**spot diagnosis**" can be reached with quasi certitude.

In this book we have tried to compile those type of cases but also included the odd, rare adult or pediatric entity with their typical imaging patterns. Only a few relevant images as well as the clinical history are presented for each case and left for the reader to contemplate the diagnosis while the solution to the case including the pertinent imaging findings are presented on the subsequent page thus allowing the reader to self-assess. We added a short discussion on the disease followed by the most common differential diagnosis. At the end of each case there is a briefing on suggestive management, as well as suggestions for further reading.

We hope that this book will serve as a resource for brushing up diagnostic skills and to enhance decision-making in real-world scenarios. With a focus on interactive learning, this case-based approach encourages critical thinking and application of knowledge while enjoying the thrill of the find and the satisfaction of increased confidence in making neuroradiological diagnoses.

T. Krings, MD
A. Haouimi, MD
Z. Haq Choudhuri, MD
R. Begum, MD
R. Bouguelaa, MD

Contributors

Amer ALAREF, MD. Thunder Bay, On. Canada
Fouzi BALA, MD. Tours, France
Zine-Eddine BOUDIAF, MD. Batna, Algeria
Chelia BOUKAABA, MD. Batna, Algeria
Chanon NGAMSOMBAT, MD. Bangkok, Thailand
Mosleh AL-RADDADI, MD. Medinah, Saudi Arabia
Samir ROUABHIA, MD. CHU Batna, Algeria

Special Thanks

AKM Shamsuzzaman, RT. Dhaka, Bangladesh
Atef Bouguelaa, CIM Aurès, Batna, Algeria
Mustapha Benguiba, CIM Aurès, Batna, Algeria
Samia Hocine, CIM Aurès, Batna, Algeria
Asia Kendri, CIM Aurès, Batna, Algeria
Rahima Bougueffa, CIM Aurès, Batna, Algeria

About the Authors

Timo Krings is currently the Chair of the Division of Neurointerventional Radiology at the Lahey Clinic and Director of the Neurointerventional Centre at Beth Israel Lahey Health. He graduated from Aachen, Germany and at Harvard Medical School. After residency with Prof. Armin Thron, he completed his neurointerventional fellowship with Professor Pierre Lasjaunias in Paris, France. Formerly he was a Professor of Radiology and Surgery and held the David Braley and Nancy Gordon Chair in Interventional Neuroradiology at the University of Toronto, Canada. He has trained more than 40 fellows over his career, among them many are serving at leading positions all over the world including the US, Canada, Switzerland, France, Netherlands, Singapore, Thailand, Japan, and China. He published more than 500 peer reviewed articles and authored four major textbooks in Interventional Neuroradiology. Currently he is sitting in the editorial board of multiple peer review journals in Diagnostic and Interventional Neuroradiology in Europe and North America.

Ammar Haouimi is a consultant radiologist at Aurès Imaging center at Batna, Algeria. He got his training from University of Bordeaux II. He was the head of the Department of Radiology and Imaging at Ohud Hospital and Madinah Maternity and Children Hospital (MMCH), Madinah, Saudi Arabia. Currently he is one of the editors of the biggest online open-source teaching radiology website “Radiopaedia.org” where he published more than 2600 cases and contributed to more than 860 articles. He authored seven books on different Radiology subspeciality.

Z.H. Choudhuri is a staff neuroradiologist and former chief of Department of Radiology and Medical Imaging at Edmundston Regional Hospital, NB, Canada. He got his Neuroradiology training under mentorship of Prof. Dominique Doyon and Prof. Pierre Lasjaunias at Hôpital Bicêtre, University of Sorbonne, (Paris V). Previously he served as Senior Consultant at King Fahd Hospital in Medinah, Saudi Arabia. He has extensive teaching experience and has trained many residents both at home and abroad.

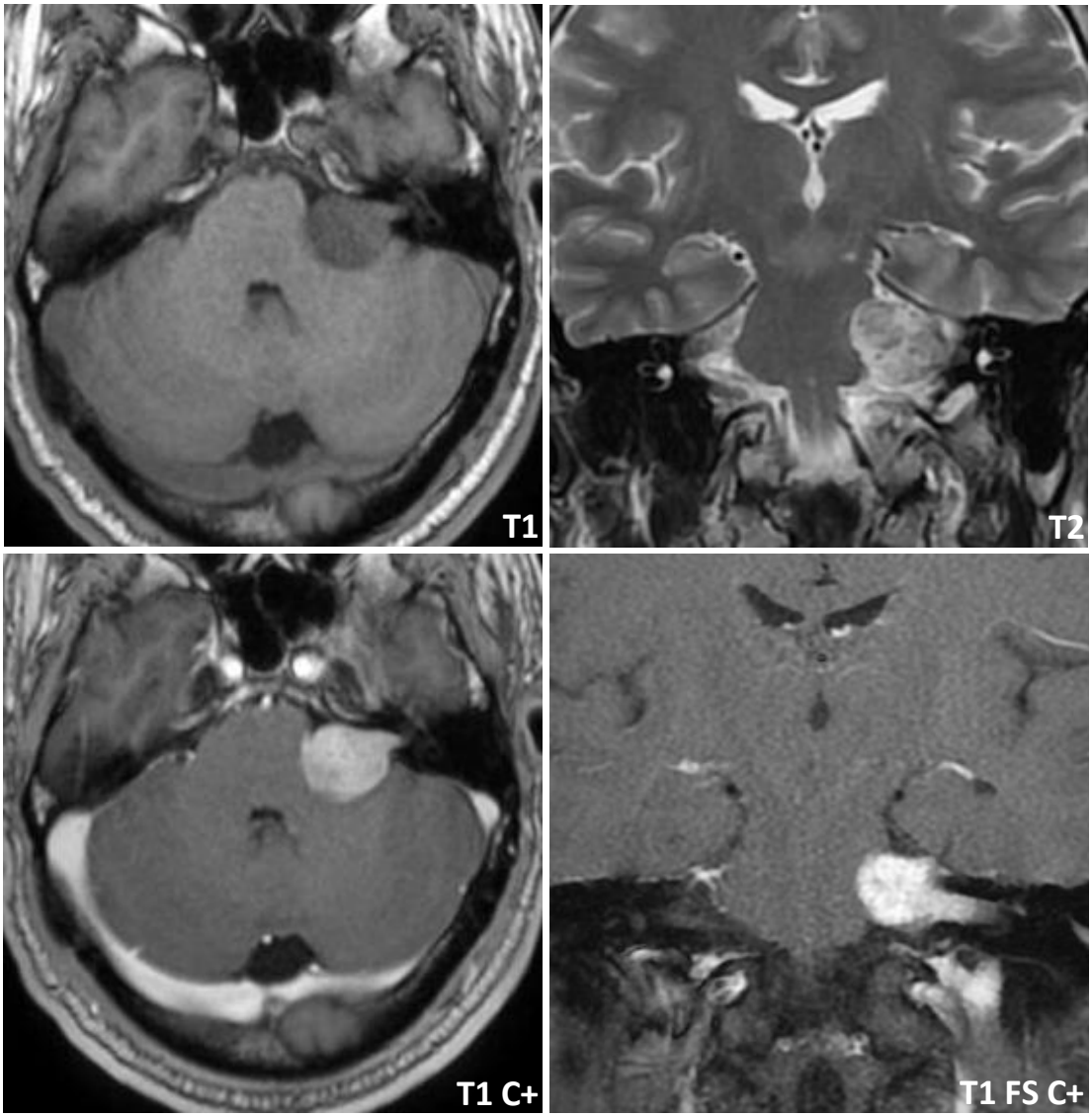
Rukhsana Begum is a staff radiologist and former chief of Radiology and Medical Imaging at Edmundston Regional Hospital, NB, Canada. She specialised from University of Sorbonne (Paris V) and worked at Hôpital Bicêtre, Hôpital Avicenne, Necker–Enfants Malades and Hôpital Robert Debré in Paris. She was head of the department of Radiology and Imaging at Maternity and Children Hospital, Madinah, Saudi Arabia. She has extensive teaching experience, particularly in Pediatric Neuroradiology.

Rabah Bouguelaa is a consultant radiologist and Chief of Aurès Imaging center at Batna, Algeria. He specialized from University of Lyon (Hôpital Edouard Herriot), France. Currently he is one of the contributors of the biggest online open-source teaching radiology website “Radiopaedia.org”. Along with Ammar Haouimi he authored three books on different Radiology subspeciality.

Case 1

Clinical History

A 55-year-old man presented with tinnitus, a slowly progressive left-sided perceptive type of hearing deficit.



Findings

An ice cream cone extra-axial lesion at the left cerebellopontine angle along the course of the acoustico-facial bundle. The lesion has both an intra and extra-canalicular component. It appears slightly hypointense to the brain parenchyma on T1, heterogeneously hyperintense on T2 with intense homogeneous contrast enhancement. No intratumoral necrosis seen. Absent “dural tail sign”. Slight mass effect on adjacent brainstem, middle cerebellar peduncle and fourth ventricle without hydrocephalus.

Diagnosis

Vestibular schwannoma.

Discussion

Vestibular schwannomas represent up to 90% of the lesions involving the cerebellopontine angle. These benign encapsulated slow growing lesions arise from glial-Schwann cells and are considered to be grade 1 lesion of WHO classification. A unilateral well demarcated enhancing lesion along the presumed trajectory of vestibulocochlear bundle can be considered as acoustic neuroma until proven otherwise. The “dural tail sign” is almost always absent. However, when this lesion is bilateral, one should consider the diagnosis of Neurofibromatosis type 2. On rare occasion the lesion can grow rapidly to compress the brainstem and may interfere with vital functions. MRI is the investigation of choice. CT scan is less sensitive however, indirect signs may include widening of internal acoustic meatus with or without erosion on the bone window when it involves an intracanalicular segment.

Common Differential Diagnosis

- Meningioma: often showing “dural tail sign”, may calcify, usually no intracanalicular extension.
- Epidermoid cyst: isointense to CSF on T1 and T2 with restricted diffusion.
- Arachnoid cyst: isointense to the CSF on both T1 and T2, but no restricted diffusion.

Management Options

Vestibular schwanommas grow very slowly (less than 1 mm every year). Treatment is best entertained via a multidisciplinary approach and can include open surgery, radiosurgery or conservative management. A successful surgical resection only prevents further hearing loss but does not restore the hearing already lost.

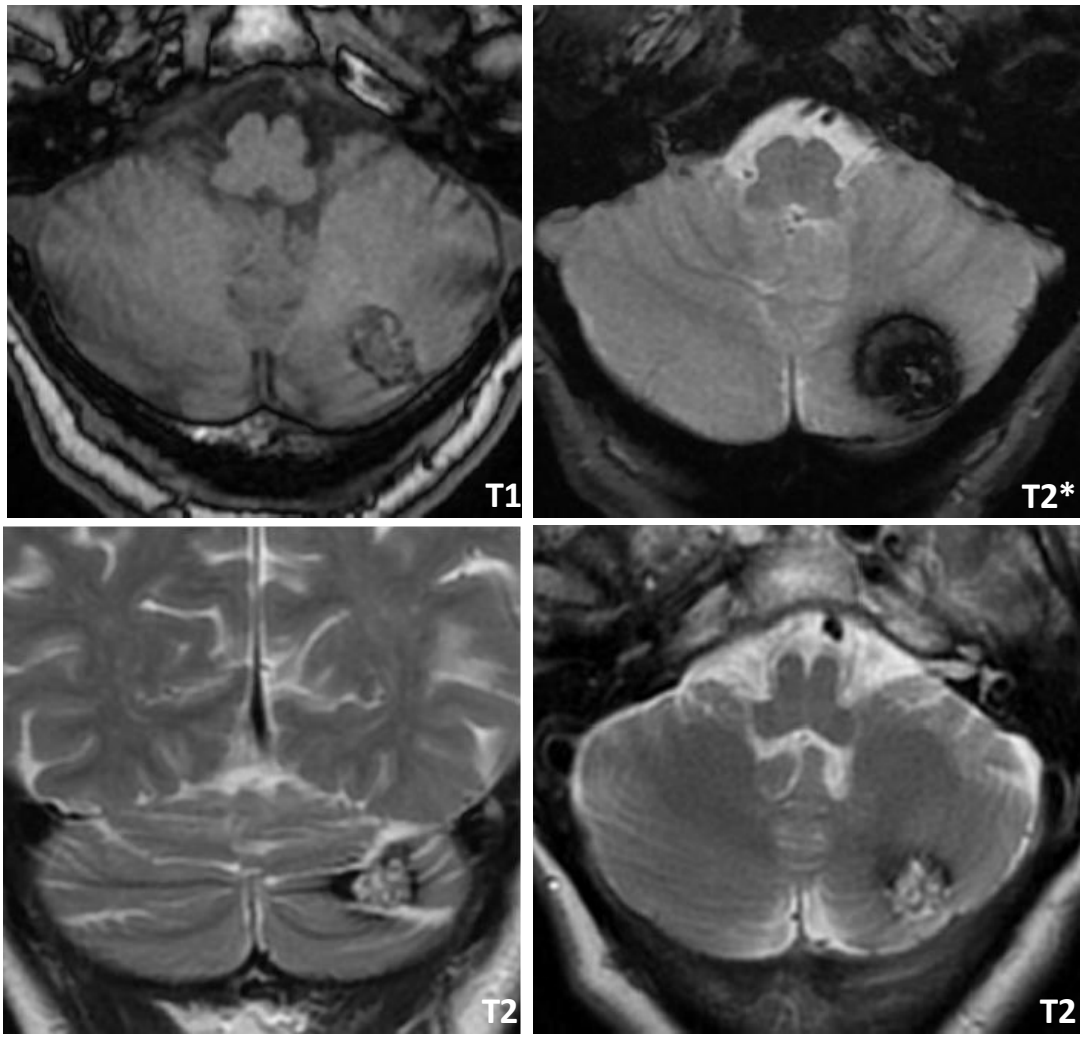
Reference and Further Reading

1. Haouimi, Ammar. 2022. **“**Radiopaedia.org**”**. Accessed on March 07, 2022. https://doi.org/10.53347/rID-74194.
2. Kabashi, Serbeze et al. 2020. **“**Pubmed **”**. Accessed on May 02, 2024. https://pubmed.ncbi.nlm.nih.gov/33627932/.

Case 2

Clinical History

This is an asymptomatic 35-year-old man. Incidental finding.



Findings

An intra-axial left cerebellar “popcorn-like” lesion without any perilesional edema. The MR findings show hemoglobin breakdown products with central high signal on both short and long sequences due to microhemorrhage of varying ages. T2* blooming is due to magnetic susceptibility from the paramagnetic effect of hemosiderin. No contrast enhancement after gadolinium (image not shown). No necrosis or mass effect.

Diagnosis

Cavernoma also known as cavernous hemangioma.

Discussion

Cavernomas are formed by sinusoidal vascular spaces without any normal neural tissue in between. They are angiographically occult. Macroscopically they look like intraparenchymal “raspberries” filling the caverns. Lesions can either be solitary or multiple, inherited or acquired. Cavernomas may also be associated with developmental venous anomalies. An increased pressure secondary to stenosis of collecting venules may lead to intraparenchymal microhemorrhage. Fibroblastic and endovascular proliferation ultimately causes cavernoma formation.

On MRI the mixed signal intensity surrounded by hemosiderin rim is responsible for the typical “popcorn” appearance of the lesion. Gradient echo sequence like T2* and SWI are extremely helpful to characterize the lesion. Occasionally the lesions can show peripheral rim enhancement due to surrounding inflammation and gliosis.

Common Differential Diagnosis

- Hemorrhagic neoplasm: often showing perilesional vasogenic edema and mass effect.
- Amyloid angiopathy specially at supratentorial level: usually multiple and in the elderly.
- Thrombosed AVM: DSA can depict stagnating blood flow.

Management Options

A small cavernoma when discovered incidentally in an asymptomatic patient may be considered as a “do not touch” lesion. There is controversy as to what is the most appropriate management of symptomatic cases. Associated seizures can often be managed conservatively. However, cavernomas may manifest with atypical features like acute hemorrhage. In such cases the imaging follow-up is very useful to confirm diagnosis.

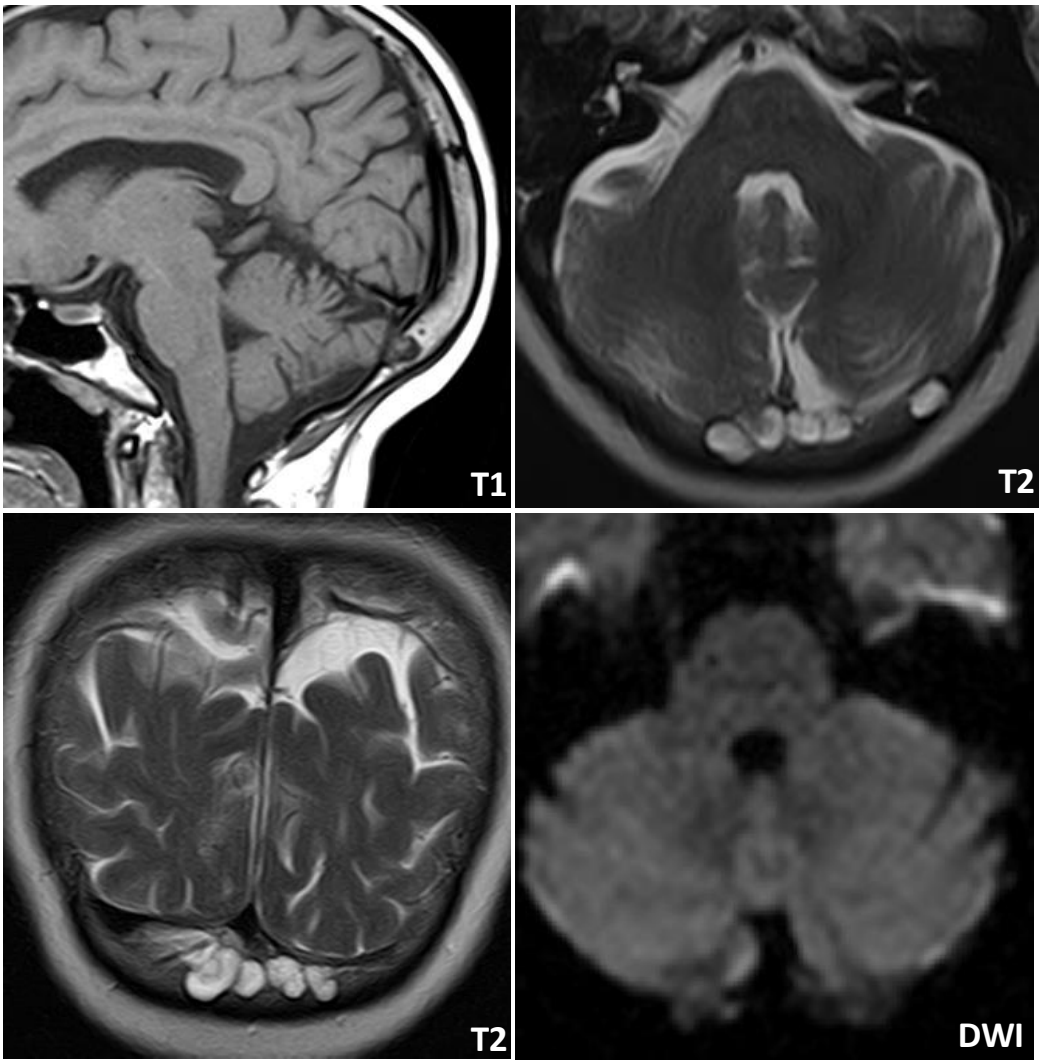
Reference and Further Reading

1. Haouimi, Ammar. 2019. “Radiopaedia.org”. Accessed on march 07, 2022. https://doi.org/10.53347/rID-70060.
2. Maharani, Putri. Hidayati, Hanik Badriyah. Kurniawan, Shahdevi Nandar. 2022. “sciencedirect”. https://doi.org/10.1016/j.radcr.2022.06.088.

Case 3

Clinical History

A 60-year-old woman complaining of chronic headache. Incidental finding on MRI.



Findings

Multiple sharply defined tiny calvarial outpouchings of CSF within the occipital region, adjacent to the lateral and straight sinuses. The lesions are isointense to CSF on all sequences with no restricted diffusion or mass effect. No contrast enhancement (images not shown).

Diagnosis

Pacchionian granulations.

Discussion

Pacchionian granulations are enlarged arachnoid villi projecting into the dural venous sinuses. But at times the granulations penetrate dura without reaching the venous sinus. Though typically the aberrant granulations are seen at the sphenoid bone, they can occur anywhere. CSF pulsations may enlarge aberrant granulation. The size of Pacchionian granulations is usually up to 15 mm. When the size exceeds 15 mm or more they are known as giant arachnoid granulations. Giant granulations often show venous channels and septations. The enlargement may reach the point of CSF rupture which may complicate to meningitis. When Pacchionian granulations are found in the sphenoid or temporal bone, special attention is to be given to sphenoid sinus and mastoid air cells to rule out CSF leakage. MRI and MRV is the investigation of choice. However, CT scan of the skull base on the bone window is recommended to explore the sphenoid sinus.

Common Differential Diagnosis

- Multiple myeloma: often aggressive.
- Dermoid cyst: fatty T1 hyperintensity.
- Epidermoid cyst: restricted diffusion.

Management Options

Aberrant Pacchionian granulations are incidental findings and considered as “do not touch” lesions. No treatment is needed unless it is complicated specially when there is CSF rupture. Complicated meningitis is often treated conservatively. Elective surgical dural repair is recommended to prevent recurrent bouts of meningitis.

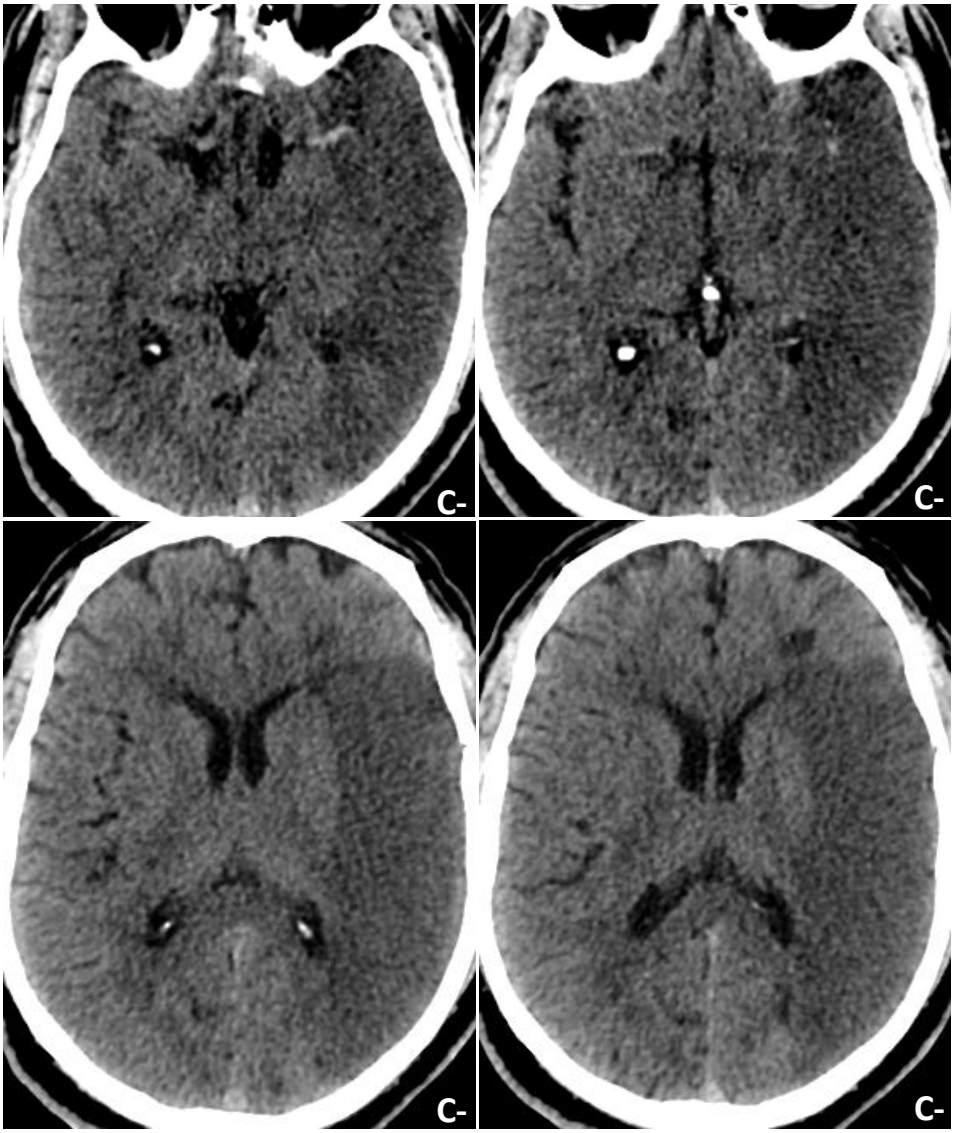
Reference and Further Reading

1. De Keyzer, Bart et al. 2024. “ The neuroradiology journal vol”. Accessed on August 30, 2024. http://doi.org/10.15274/NRJ-2014-10047.
2. Haouimi, Ammar. 2019. “Radiopaedia.org”. Accessed on March 07, 2022. https://doi.org/10.53347/rID-71054.
3. Lu Chao, Xuan et al. 2012. “World J Radiol”. Accessed on May 02, 2024. https://doi.org/10.4329/wjr.v4.i7.341.

Case 4

Clinical History

A 65-year-old man, presented with sudden right-sided neurologic deficit for 12 hours.



Findings

Parenchymal low-density area in the left superficial Sylvian artery territory with effacement of adjacent cortical sulci. It involves both white and gray matter suggesting cytotoxic edema. The central gray matter is of normal aspect. No intraparenchymal hemorrhage, midline shift or hydrocephalus. The left middle cerebral artery appears as linear high density in the Sylvian fissure: “dense MCA sign” and as a dense punctiform area in the Sylvian fissure: “dot sign” due to an intraluminal blood clot.

Diagnosis

Established Middle Cerebral Artery (MCA) infarction.

Discussion

The middle cerebral artery supplies a large portion of the cerebral cortex responsible for vital brain function including language and cognition as well as motor and sensory function. This type of stroke is due to acute blockage or obstruction of the middle cerebral artery. The clinical expression depends on the area involved and can include hemiparesis, hemiplegia, aphasia and hemineglect. The diagnosis of an acute MCA infarction is straightforward in the appropriate clinical context.

Imaging is not only important for diagnosis but also for decision making in the plan of treatment. Though diffusion weighted MR images are more sensitive, CT is the modality of choice considering the widespread 24/7 availability. The most important thing on CT is to rule-out any intraparenchymal hematoma in the acute phase. Imaging also helps to detect early signs of brain herniation. Perfusion imaging can be performed to assess the size of penumbra, necessary for endovascular management.

Common Differential Diagnosis

- Congestive edema due to venous thrombosis.

Management Options

The management of acute MCA infarction depends predominantly on the presence of hemorrhage and the time elapsed since ictus. Intravenous thrombolysis followed by mechanical thrombectomy is the gold standard if diagnosed within 4-5 hours. Perfusion imaging is recommended when considering an endovascular therapy.

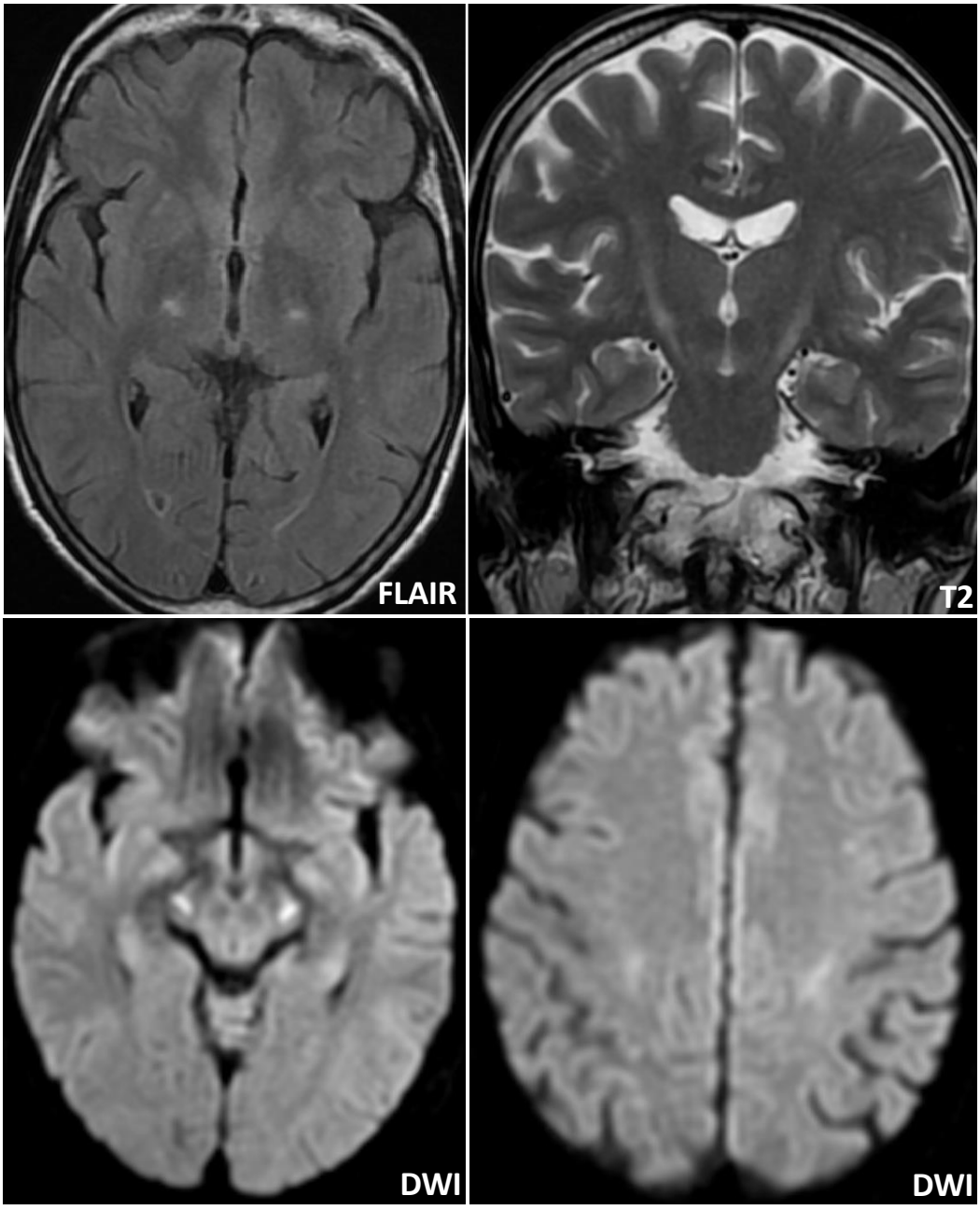
Reference and Further Reading

1. Haouimi, Ammar. 2022. “Radiopaedia.org”. Accessed on March 07, 2022. https://doi.org/10.53347/rID-73621.
2. Moulin T et al. 1996. “Neurology”. Accessed on May 02, 2024. https://doi.org/10.1212/wnl.47.2.366.

Case 5

Clinical History

A 60-year-old hypertensive woman, presented with progressive weakness, gradual loss of hand-eye coordination, muscular cramps and twitching.



Findings

Bilateral T2 high signal at the presumed topography of corticospinal tract. It extends from the posterior limb of the internal capsules to the precentral gyrus upward and downward up to the cerebral peduncles. No mass effect. No enhancement (images not shown).

Diagnosis

Amyotrophic lateral sclerosis (ALS), also known as Lou Gehrig's disease.

Discussion

Amyotrophic lateral sclerosis (ALS) is rare but the most common form of motor neuron disease involving both upper and lower motor neurones. It causes progressive weakness and eventual death mostly due to respiratory insufficiency. The incidence is approximately 1-2 per 100,000 cases. Pathologically there is loss of motor neurones with intraneuronal ubiquitin immunoreactive inclusions. Dopamine deficiency is believed to play a significant role. The progression of the disease is centrifugal with a median survival rate up to 4 years from diagnosis. However, up to 10% of the cases may survive beyond 10 years. In advanced cases radiologic “motor band” sign can be seen particularly on susceptibility weighted images due to high concentration of iron in the microglia of the motor cortex. However, it is prudent to mention that even a normal corticospinal tract can show high signal on 3T/7T MR. Diffusion tensor imaging (DTI) can show abnormality before appearance of pyramidal symptoms.

Common Differential Diagnosis

- Multiple sclerosis: multiple oval and linear foci, usually involves brain and spinal cord.
- Wallerian degeneration: clinical history, usually unilateral.
- Hypertrophic olivary degeneration: T2 high signal of inferior olivary nucleus with hypertrophy.

Management Options

Symptomatic treatment for cramps and spasticity. Glutamate release inhibitor and insulin like growth factors (riluzole) may prolong the survival. Speech and occupational therapy to help communication and day-to-day activities. Physiotherapy for maintenance of strength and mobility. Nutritional counselling must be provided to support the quality of life.

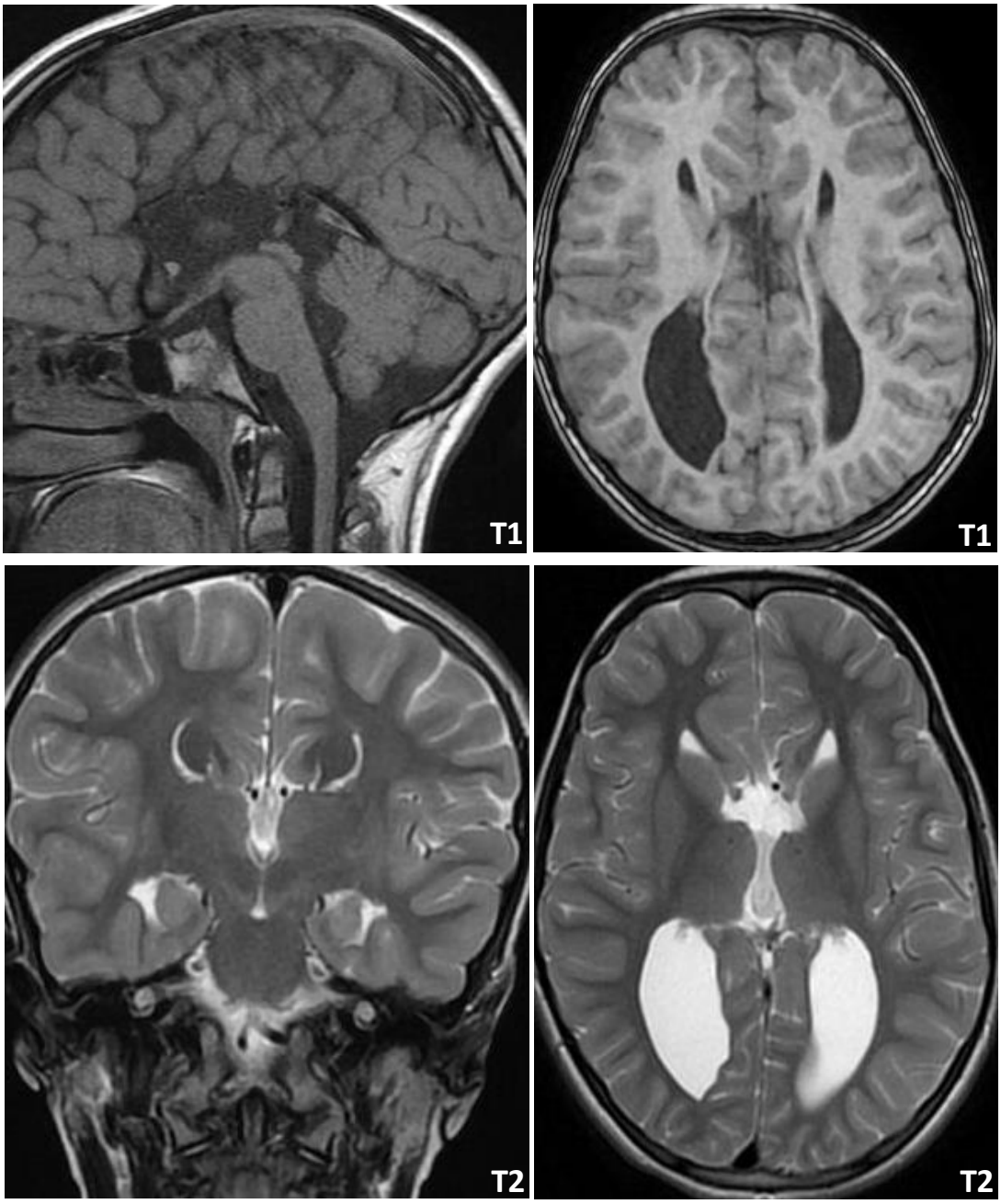
Reference and Further Reading

1. Haouimi, Ammar. 2020. “Radiopaedia.org”. Accessed on March 07, 2022. https://doi.org/10.53347/rID-85071.
2. Khosla, Radhika et al. 2021. “PLoS One”. Accessed on May 02, 2024. https://doi.org/10.1371/journal.pone.0247025.

Case 6

Clinical History

A 4-year-old girl presented with partial epilepsy.



Findings

Absence of corpus callosum with radial distribution of the gyri on the sagittal plane: “sun ray appearance”. Diverging frontal horns giving “Viking helmet or Moose head” appearance on coronal images. Typical “racing car sign” on axial T1 and T2 representing absence of normal “bowtie” appearance of both lateral ventricles.

Diagnosis

Corpus callosum agenesis.

Discussion

Corpus callosum dysgenesis is the most common congenital CNS malformation. Isolated agenesis is rare and often remains asymptomatic. Minor dysgenesis or hypogenesis are mostly discovered incidentally. Though the exact cause of corpus callosum agenesis remain unknown, maternal infection or injury during the 12^{th} week of gestation and exposure to alcohol and toxic medication may predispose to this condition. The clinical picture is often due to associated abnormalities.

In complete agenesis the absent cingulate gyrus renders a “spoke wheel” gyral pattern at the medial surface of each hemisphere. Disruption of the hypothalamopituitary axis may be associated with delayed milestones and seizures.

Diagnosis is typically made with ultrasound antenatally. The diagnosis is challenging before 20-22 weeks. Fetal MR is recommended in such cases and to evaluate associated abnormalities. DTI (Diffusion Tensor Imaging) is very helpful in detecting the corpus callosum agenesis. DTI prominent Probst bundles are seen in the antero-posterior direction running parallel to the lateral ventricles.

Common Differential Diagnosis

- Septo-optic dysplasia: downward pointing frontal horns.
- Lobar holoprosencephaly: usually absent falx, fused fornices, fused thalami.
- Cavum septum pellucidum: pellucidal cyst showing low signal on T1 and high signal on T2.
- Quadrigeminal cistern arachnoid cyst: below the cerebral veins, and not triangular.

Management Options

There is no definitive treatment. Symptomatic management may help. The overall prognosis depends on the presence of associated anomalies.

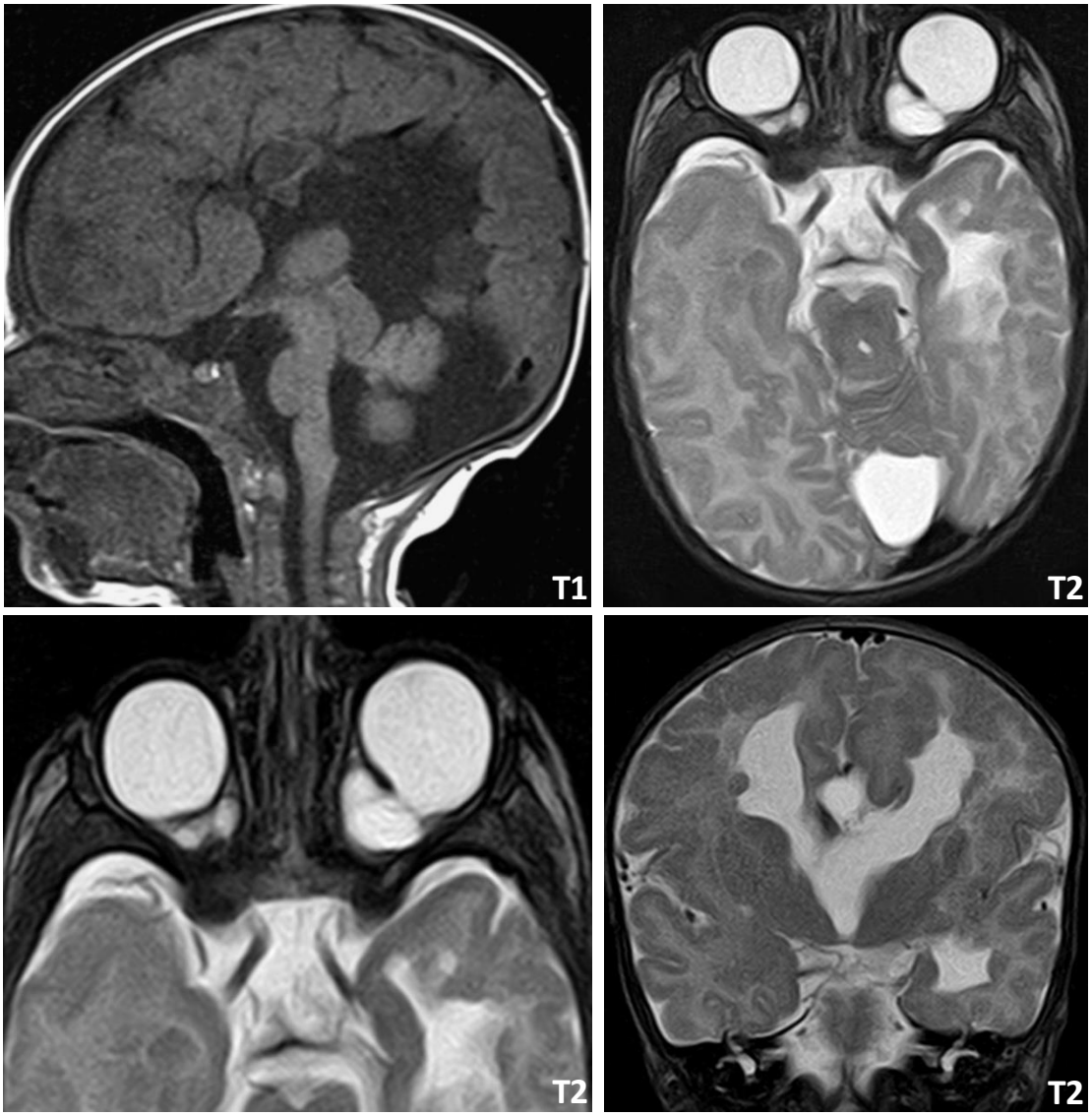
Reference and Further Reading

1. Haouimi Ammar. 2021. “Radiopaedia.org”. Accessed on May 03, 2024. https://doi.org/10.53347/rID-93755.
2. Schell-Apacik, Chayim Can al. 2008. “Am J Med Genet A”. Accessed on May 02, 2024. https://doi.org/10.1002/ajmg.a.32476.

Case 7

Clinical History

A 2-month-old baby girl presented with infantile spasm.



Findings

Bilateral colobomas with cerebellar hypoplasia. Interhemispheric arachnoid cyst, subependymal nodules and gyrational abnormalities. Diverging frontal horns giving “moose head” appearance suggesting corpus callosum agenesis.

Diagnosis

Aicardi Syndrome.

Discussion

Aicardi syndrome is rare, first described by the French neuro-paediatrician Dr. Jean François Aicardi in 1965. It is a congenital encephalopathy of unknown etiology affecting newborn infants, exclusively female. It causes severe mental and physical handicaps including severe psychomotor retardation. The classic clinical triad is corpus callosum dysgenesis, uncontrolled infantile spasm and chorioretinal lacunae.

There are two forms of the syndrome: an early-onset severe form and a late-onset form that has little impact on neurological function. Infantile spasm is often associated with corpus callosum dysgenesis and punched out lesions in the pigmented layer of retina. Retinal findings are often considered as pathognomonic. Special attention is needed to look for associated polymicrogyria and heterotopia once dysgenesis of corpus callosum is identified. Associated abnormalities may include microphthalmia, cleft palate and scoliosis.

Common Differential Diagnosis

- Aicardi-Goutières syndrome: often associated with basal ganglia calcifications and white matter abnormalities. hepatosplenomegaly, vasculopathy and CSF shows lymphocytosis.
- Congenital CMV: often causes periventricular calcification.
- Dandy-Walker malformation with supra-tentorial involvement.

Management Options

There is no definitive treatment. Symptomatic management is often helpful. Physical and occupational therapy may be needed to improve motor skill. The prognosis is poor with a median age of survival between 14.5 years to 22.5 years.

Reference and Further Reading

1. Haouimi, Ammar. 2020. “Radiopaedia.org”. Accessed on March 07, 2022. https://doi.org/10.53347/rID-73945.
2. Hopkins, Boobi et al. 2008. “Am J Med Genet A”. Accessed on May 02; 2024. https://doi.org/10.1002/ajmg.a.32537.