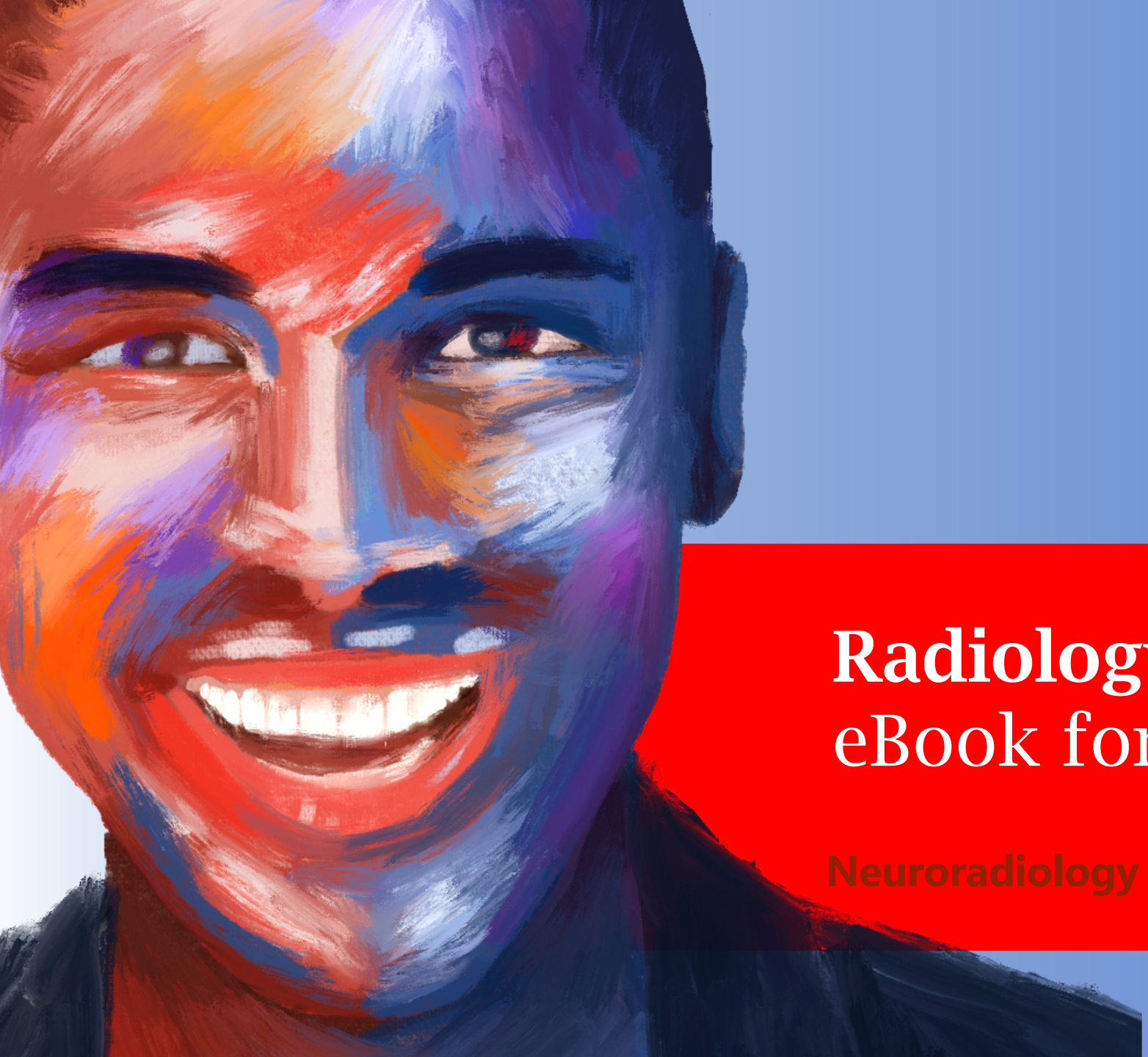

Radiológiai Klinika
SZTE

Radiology case reports
eBook for radiology practicals

Neuroradiology

Radiology case reports

Preface

The European Society of Radiologists published the electronic textbook for undergraduate education in 2023. This textbook has become the foundation of radiology education in most European countries and many other parts of the world. Europe's top radiologists have contributed their knowledge and instructive cases. This continually expanding textbook covers the theory of radiology fundamentals according to the requirements of undergraduate and residency education, with new chapters continuously added.

In addition to theoretical education, there is only a few textbooks, notes, or electronic materials available for practical training. Therefore, we have embarked on compiling an e-Book to aid practical education. Our goal is to provide students/residents with material compiled along a similar concept to complement the ESR textbook. Alongside cases, the material includes explanations and important information to deepen theoretical knowledge and its practical application. As medical practice is evidence-based, the e-Book contains many references, primarily guidelines, alongside excellent summary articles.

We hope this e-Book will serve as a useful tool in university radiology education!

Kincses Zsigmond Tamás

Radiology case reports

Neuroradiology

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Neuroradiology

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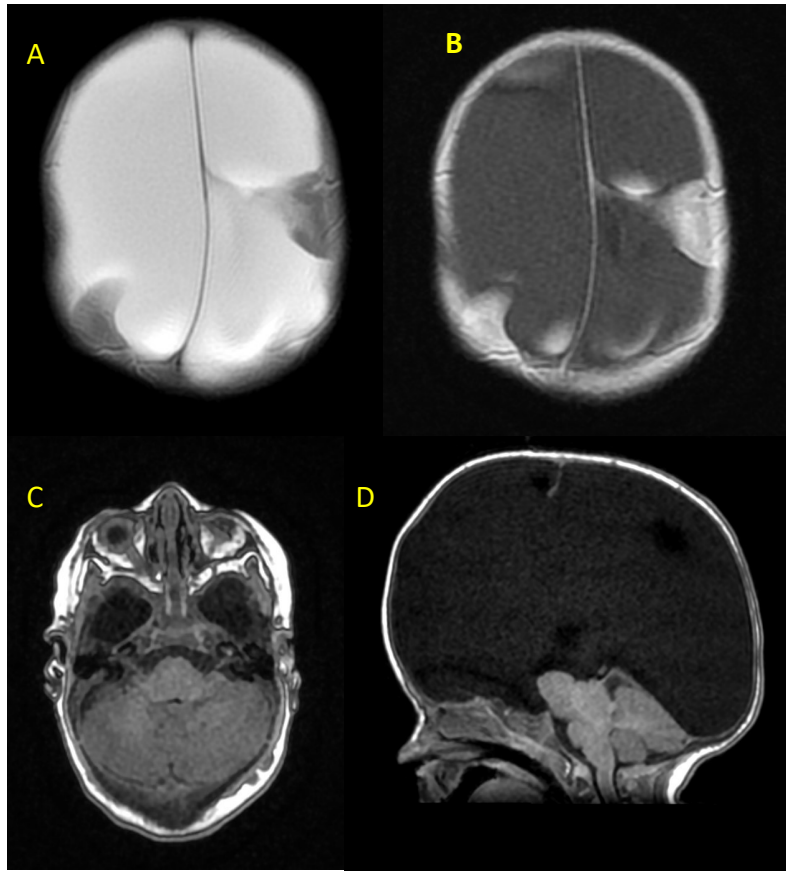
History: 5-month-old girl. Fifth pregnancy, fourth delivery. Urgent cesarean section due to neglected pregnancy, uncertain term, and malpresentation disorder. Born weighing 3250 grams with Apgar scores of 4-9. Meconium-stained amniotic fluid. At birth, dysmorphic appearance, did not cry, cyanotic. Heart rate persistently >100/min. Initiation of respiration after 1.5 minutes of Neopuff respiratory support. Pregnancy history includes smoking, flu, and multiple episodes of vaginal fluor.
Occasional seizures observed in the right upper limb. Hydranencephaly suspected based on ultrasound examination.

Status: Deeply set ears, two preauricular pendulums on the right side. Infantile palm-sized hyperpigmentation in the left gluteal region. Forced posture, head tilted to the right, pupils symmetric, unresponsive to light. Hypotonic musculature. Reflexes elicitable. Weight: 3kg

Diagnosis: **Anencephalia**



A: T2-weighted, B: FLAIR image. The intracranial space is filled with "free fluid," cerebrospinal fluid, which appears as high signal intensity on the T2-weighted image and is suppressed with the FLAIR inversion pulse. C: Axial T1-weighted image. The infratentorial structures are relatively spared. The image was taken at the level of the vestibulocochlear nerves. D: In the sagittal T1-weighted image, it is clearly visible that the rhombencephalon, mesencephalon, and diencephalon are relatively spared while there is almost complete absence of the telencephalon.

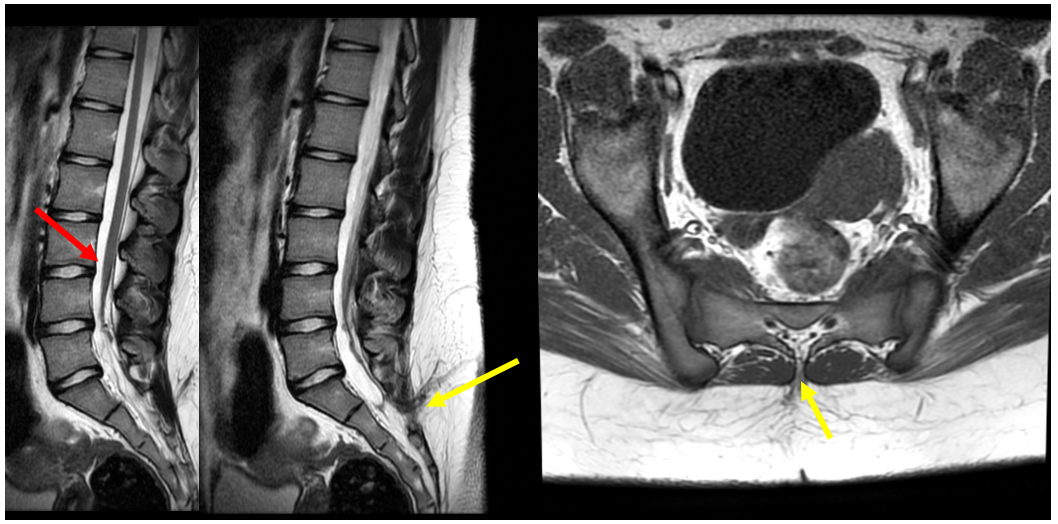
International consensus recommendations on the diagnostic work-up for malformations of cortical development

Radiology case reports

Neuroradiology

Medical history: 14 years old girl. Spina bifida and thetered cord is known since birth. No particular complains.
Status: No remarkable in her neurological status. Small hairy region around the sacal region.

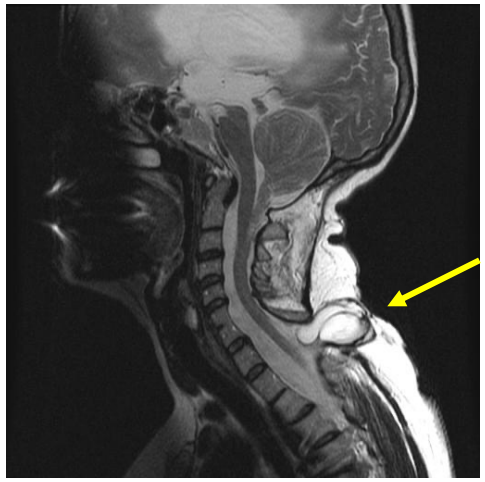
Diagnosis: **Spina bifida**



Sagittal and axial T2 weighted images about the lumbo-sacral spine. The yellow arrows indicate the spina bifida. The red arrow shows the thetered cord, the conus is at the level of LIV vertebra.

Medical history: 51 years old women. The hydrocephalus and the Arnold-Chiari malformation was known previously. Her complain was about unsteady gait, urinary incontinence and memory impairment.

Diagnosis: **Meningocele**



Sagittal T2 weighted images showing the cervical meningocele (yellow arrow). The menicses and the CSF is protruding through the bone defect of the vertebral column. Arnold-Chiary malformation and hydrocephalus also visible.

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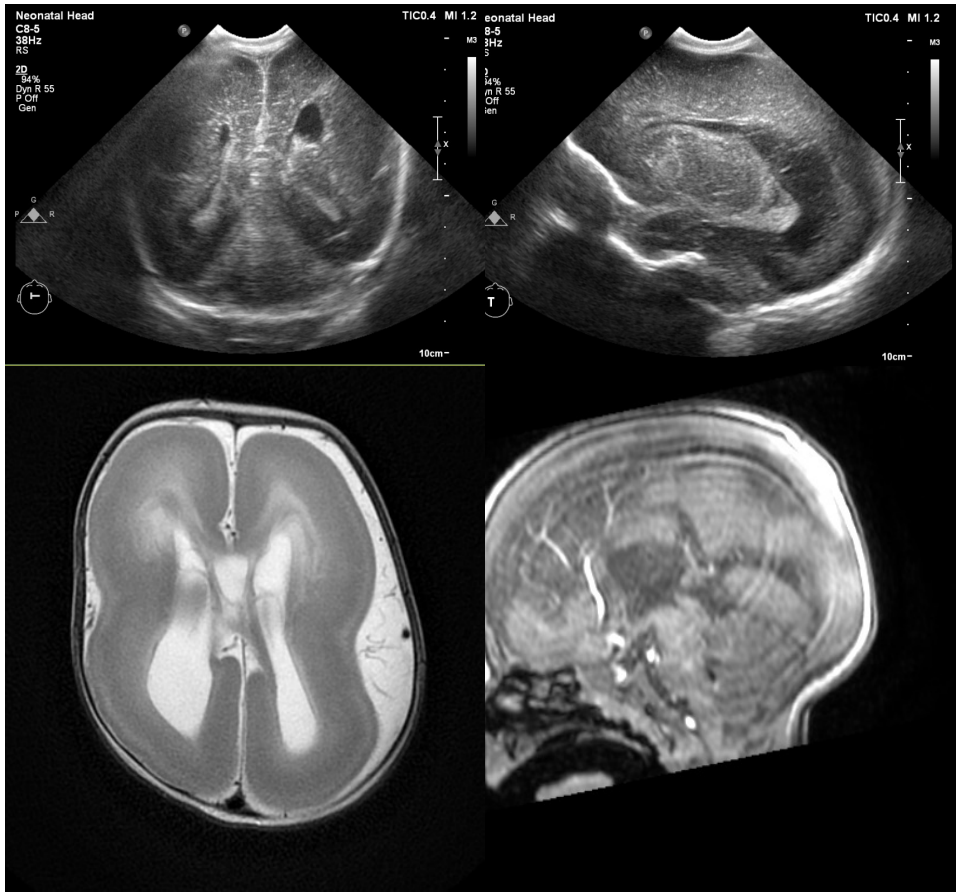
Radiology case reports

Neuroradiology

Medical history: From third (III/III) pregnancy, at 39 weeks gestation was born weighing 2580 grams, with an Apgar score of 10-10. Facial dysmorphia and microcephalia was detected.
At around 4 months of age West syndrome appeared (infantile spasms, hypsarrhythmia, developmental arrest).
Genetics: 17p13.3 deletion (Miller-Dieker syndrome)

Lissencephaly
Widespread, abnormal transmantel migration
Radiological features:
-Agyria (generalised or focal) or oligogyria.
-Figure-of-eight shaped brain
-Thickened cortex with 4 layers (3 visible on MRI)
-often combined with other migration deficits

Diagnosis: **Lissencephaly**



The transfontanelle ultrasound on the 2nd day after birth shows lissencephaly and hypoplasia of the corpus callosum. The T2 (left) and the T1 (right) weighted MR images found the lack of gyri and sulci, thickened cortex consistent with lissencephaly. Also a partial corpus callosum agenesis was found.

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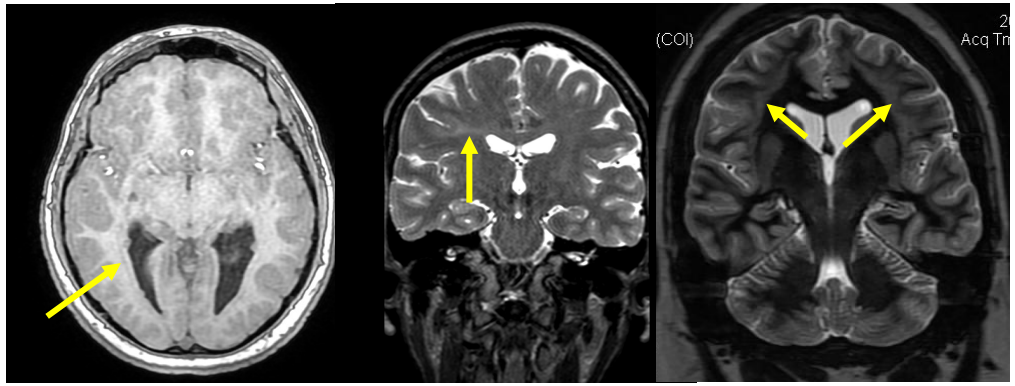
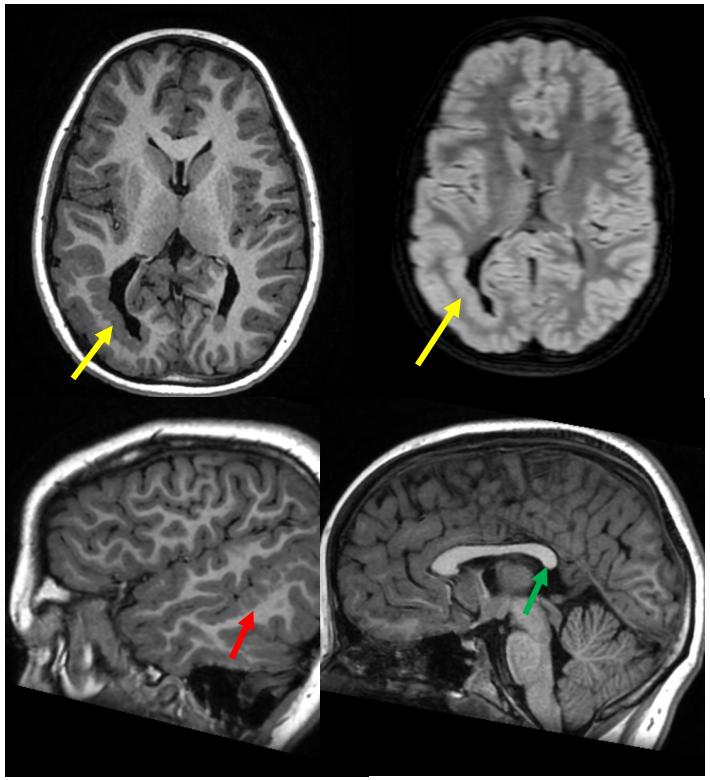
Radiology case reports

Medial history: Since the age of 11, he has been experiencing monthly to bi-monthly episodes visual sensations and vomiting. These episodes typically last for a couple of minutes followed by a brief period of confusion. On several occasions, he has also experienced tonic-clonic seizures.
Neurological status: Noting remarkable.

Gray matter heterotopia
Disorder of neuronal migration
Normal neurons in abnormal locations
Isointense with the normal gray matter
Types:
- Periventricular nodular heterotopia
- Subcortical heterotopia
- Subcortical band heterotopia



Migration disorders in three other patients with epilepsy. On the left there is a thin band of abnormal cortex between the lateral ventricle and the cortex (band heterotopia). In the middle, there is a T2 hyperintense band connecting the ventricle and the focal cortical dysplasia. On the right, bilateral subcortical band heterotopia is shown.

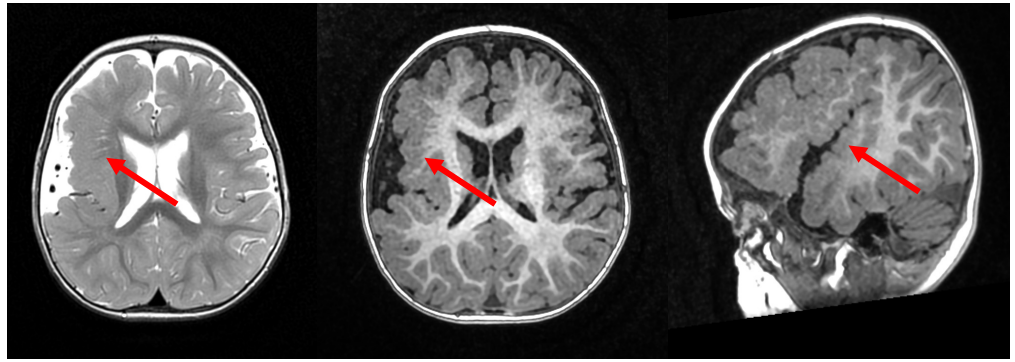


On the axial T1 (left) and FLAIR (right) images a periventricular nodular gray matter heterotopia is seen on the right side at the lateral edge of the posterior horn of the lateral ventricle (yellow arrows). On the lower left sagittal T1 weighted image polymicrogyria is visible in the temporal lobe (red arrow). Note the hypoplasia of the splenium of the corpus callosum.

Radiology case reports

Anamnesis: 14 months old boy. His thumb is in his palm on the left. He uses the left hand less frequently. His motor development is delayed: he turns to both sides, can sit and sit up by himself, has started crawling but does not yet climb. He babbles a few syllables. He never had convulsion. *Medical history:* He was operated for interrupted aortic arch + ASD + VSD.

Diagnosis: **Polymicrogyria**

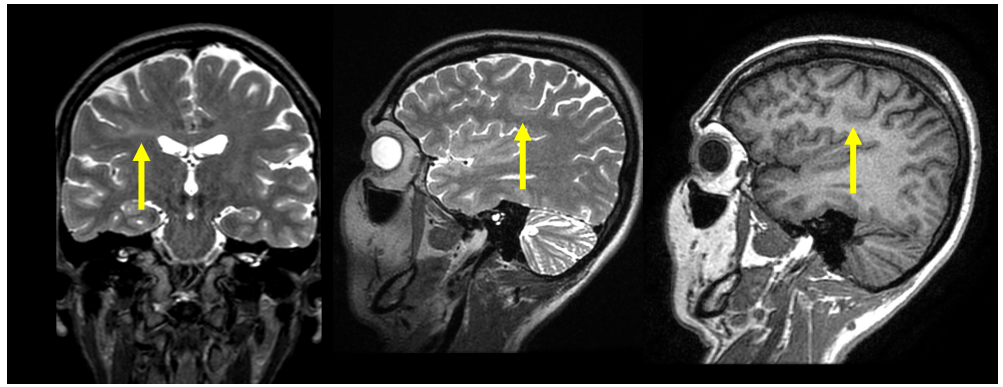


On the T2 (left), and T1 (middle and right) weighted images polymicrogyria is visible in the right perisilvian region. The intensity of the cortex is normal, but the gyri are small and compact.

Anamnesis: First seizure: at the age of 28, in the 28th week of her first pregnancy. For years, she has been experiencing epileptic seizures characterized by muscle tension and twiching on the left side of her face and neck, occurring 1-2 times a month, before or on the 1st day of menstruation, without loss of consciousness, lasting for a few minutes. She had generalised tonic-clonic seizure twice, related to treatment reduction. No complete seizure control even with combination of three antiepileptic drugs in therapeutic dose >> *therapy resistant epilepsy*

EEG: spike-and-wave activity in the right frontal leads during simplex partial seizures.

Diagnosis: **Focal cortical dysplasia**



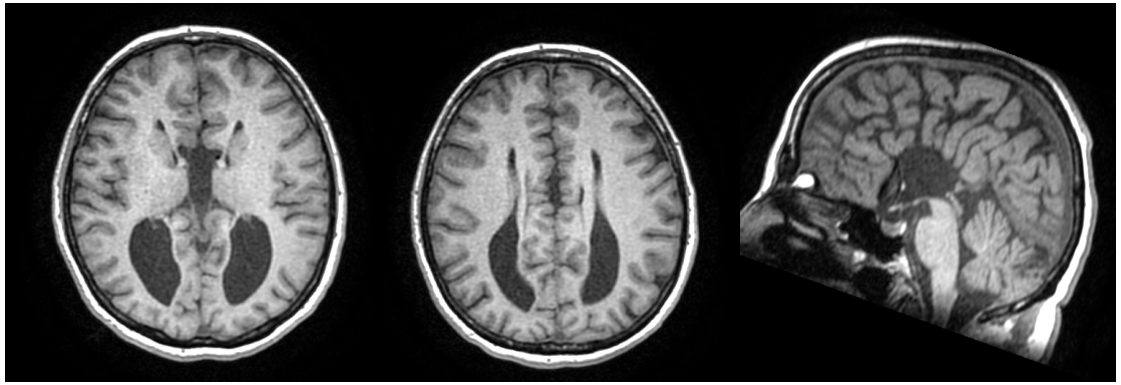
From left to right: coronal and sagittal T2 weighted and sagittal T1 weighted images. At the bottom of the right central sulcus the cortex is thicker, the intensity is increased, the gray-white matter border is not sharp. There is a band of hyperintense white matter connecting the lesion to the ventricle (transmantal sign).

Radiology case reports

Neuroradiology

Anamnesis: 48 years old woman. She had an MRI scan because of dizziness. Formerly, she was treated for depression.
Status: Nothing remarkable. No major cognitive deficit.

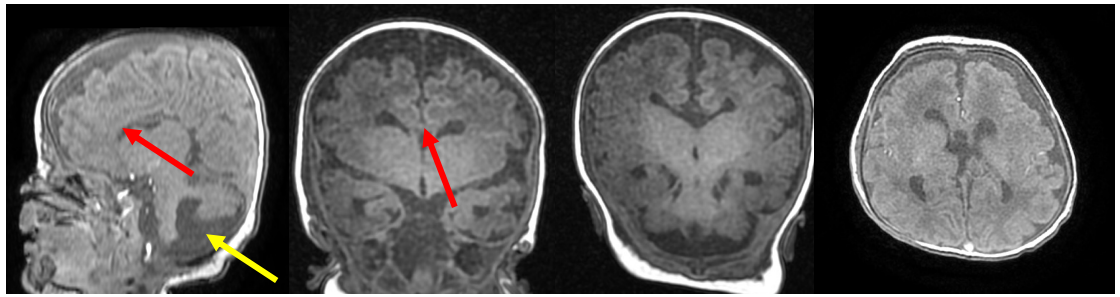
Diagnosis: **Corpus callosum agenesia**



Axial and sagittal T1 weighted images. Complete absence of the corpus callosum. The ventricles on the leftward image reminds a racing car.

Medical history: 1.5-month-old premature infant. He was born from an unmonitored pregnancy, at 34 weeks of gestation, weighing 2400 grams, delivered per vias naturales, with Apgar scores of 6-8. Two months later, he passed away due to multiple developmental disorders and respiratory failure.

Diagnosis: ***Partial* corpus callosum agenesia and Dandy-Walker sy.**



T1 weighted MRI images of an achondroplastic newborn with partial corpus callosum agnenesia. The red arrows are pointing to the genu of the corpus callosum. The body and the splenium is missing. Note the Dandy-Walker malformation (yellow arrow): enlarged posterior fossa, vermian hypoplasia, cephald rotation of the vermian remnant, cystic dilatation of the 4th ventricle posterriorly

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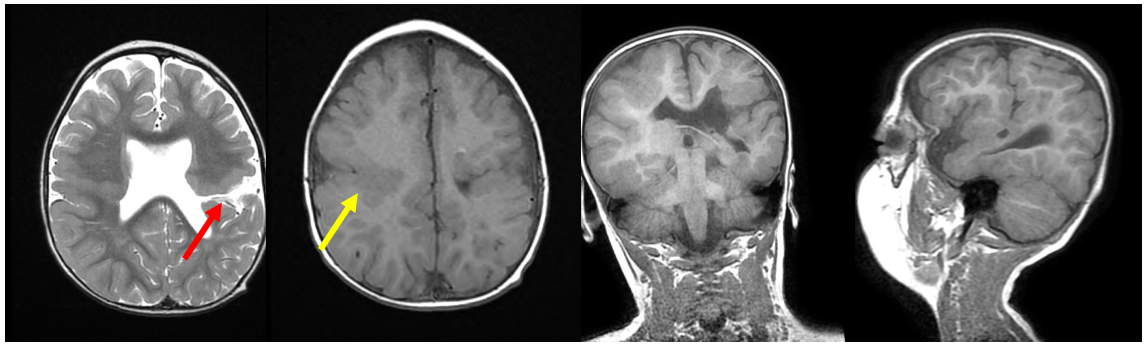
Radiology case reports

Medical history: 1.5-year-old toddler. According to the mother, the child does not speak words, only makes sounds. Sits supported. Reaches for objects. Based on the referral, the child developed normally until 6 months of age, but the mother did not take him for the recommended examinations.
Perinatal history: born at 40 weeks, weighing 3060 grams, Apgar score 10/10.



Axial T2 and T1 weighted and coronal and sagittal T1 weighted MRI scans. Bilateral schizencephaly, open lips on the left (red arrow), closed lips on the right (yellow arrow). There is a narrow right transhemispheric cleft bordered by polymicrogyric cortex on the left. The similar cleft on the right does not reach the ventricle. Not the lack of septum pellucidum.

Diagnosis: **Schizencephaly**

Schizencephaly
Cleft lined by polymicrogyric grey matter extending across the full thickness of the cerebral hemispheres from the ventricular surface to the pial surface.
Two forms:
- Open lip: The cleft is fully patent and filled with CSF
- Closed lip: The cleft is sealed by abuting cortical margins and does not reach the ventricles.

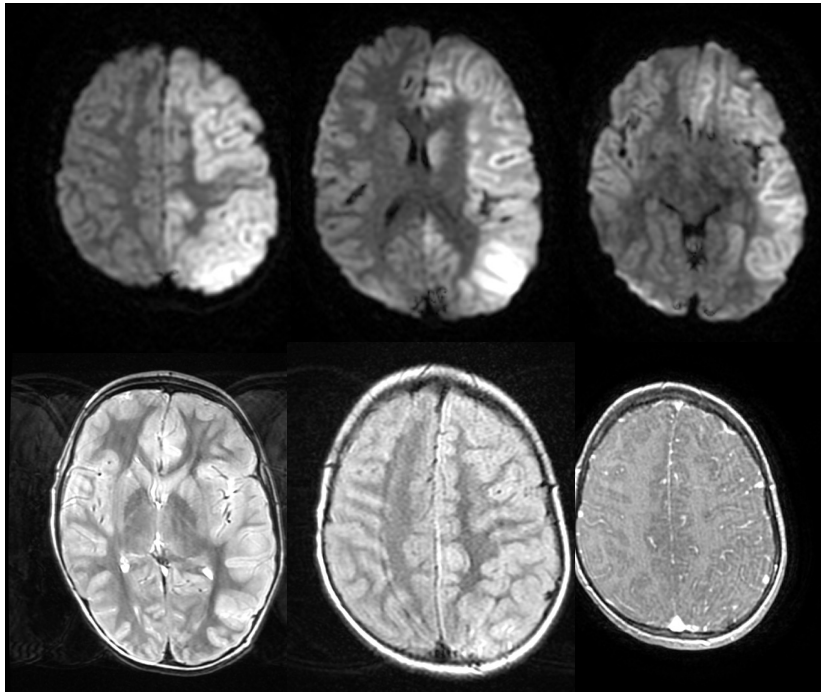
Definitions and classification of malformations of cortical development practical guidelines

Radiology case reports

Medical history: The 8-year-old child was transferred from the Department of Maxillofacial Surgery to the Pediatric Department, due to a focal seizure. The patient was known to have restrictive cardiomyopathy and mitral and tricuspid valve insufficiency. A heart transplant was planned, and dental sanitation was performed a day before the seizure. In the morning, the parents noticed that the child was tired and lethargic. Later he had a tonic-clonic seizure localised to the left side. It was difficult to control the status epilepticus. His level of consciousness did not improve after the seizure.

CSF: WBC: 55 M/L RBC: 9 M/L total protein: 0.51 g/L cytology: predominance of polymorphonuclears.

Diagnosis: **Cortical diffusion restriction due to lingering epileptic activity. Cytotoxic oedema.**



Upper row: Diffusion weighted images. Left hemispherical cortical diffusion restriction that does not respect vascular territories. Lower row: T2 weighted, FLAIR images show slight cortical hyperintensity according to the diffusion restriction. On the postcontrast T1 weighted image (lower right) arachnoidal enhancement is visible in the sulci a consequence of purulent meningitis.

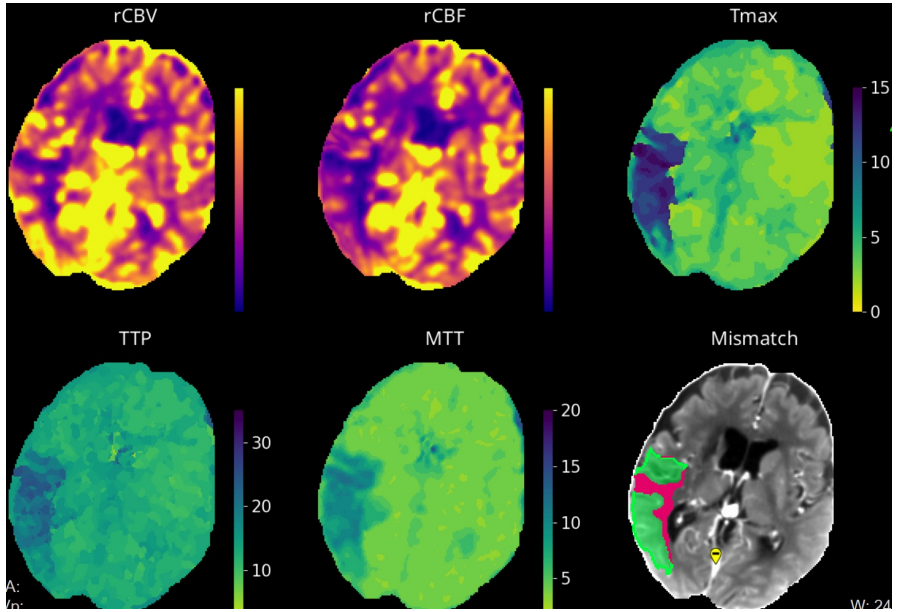
Radiology case reports

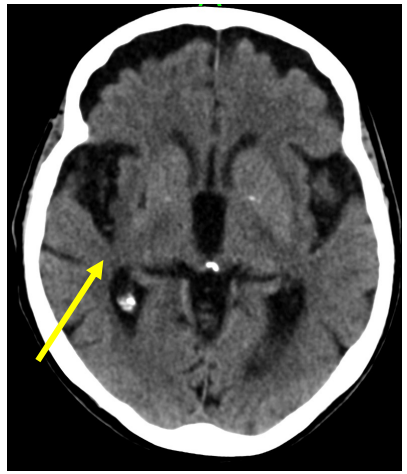
Neuroradiology

Medical History: 87-year-old woman. Examination initiated due to collapse. Started at 19:00 with right-sided fixation, left corner of the mouth dragged, left upper limb weakened. Arrived for CT scan at 21:42. Medical History: Known left internal carotid artery (ICA) stenosis (75-90%).

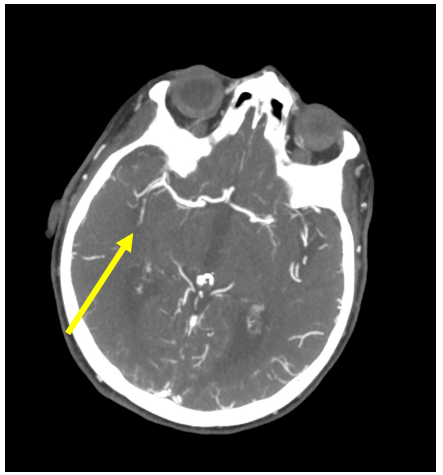
Status: Eyes deviating to the right, left-sided hemiparesis (upper limb: proximal strength: 4/5, distal strength: 2-3/5, lower limb: 3/5). NIHSS: 5.

AHA/ASA Guideline for the early management of patients with acute ischemic stroke: 2019 update





Native CT: Blurred gray-white matter border in the posterior part of the right insula. ASPECT score: 9/10.



The wall of the right middle cerebral artery (M2 segment) is irregular, followed by occlusion.

Therapy >> systemic i.v. thrombolysis

In the perfusion CT image, prolonged Tmax (>6s) is clearly visible. Cerebral blood flow (CBF) less than 30% of the contralateral side in only in a small area, representing the core.

- Malformations/epilepsy
- Cerebrovascular disorders
  - Ischaemic stroke
- Head trauma
- Brain tumours
- CNS infections
- Inflammatory CNS disorders
- Neurodegenerative disorders

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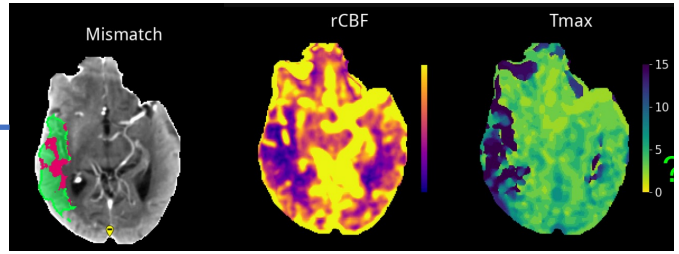
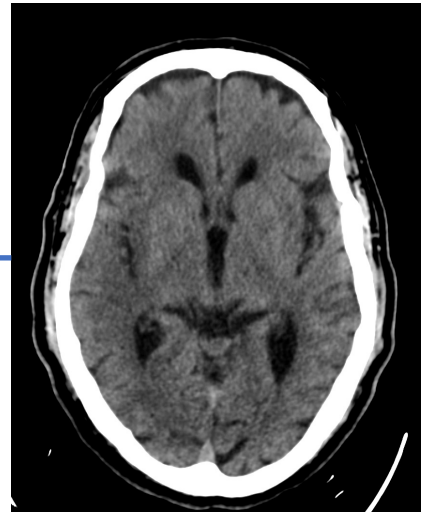
Radiology case reports

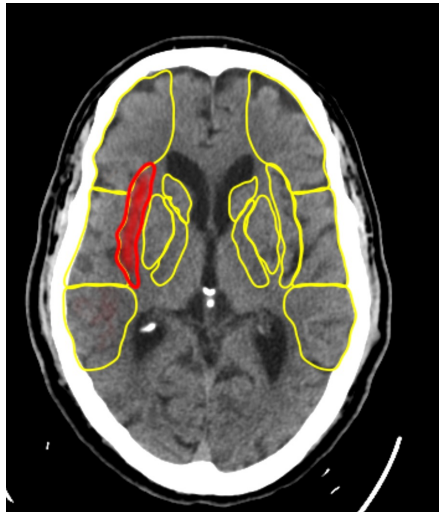
Neuroradiology

Medical History: Went to bed without complaints at 22:00, woke up at 03:00 feeling dizzy but managed to go to the bathroom. By 05:00 upon awakening, asymmetry of the face, left-sided limb weakness, and speech disorder were noted. Arrived for CT scan at 07:26.
Status: Left-sided hemiparesis 2-3/5, central VII paresis, dysarthria. NIH Stroke Scale: 9.



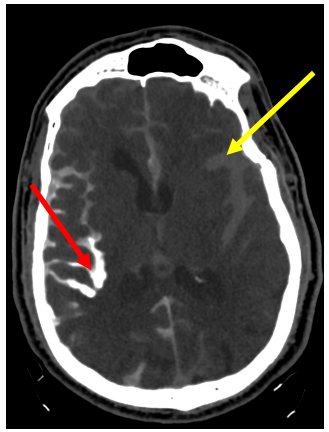
The CT perfusion measurements revealed a large penumbra (green) and a small core (red). (Left-side image).



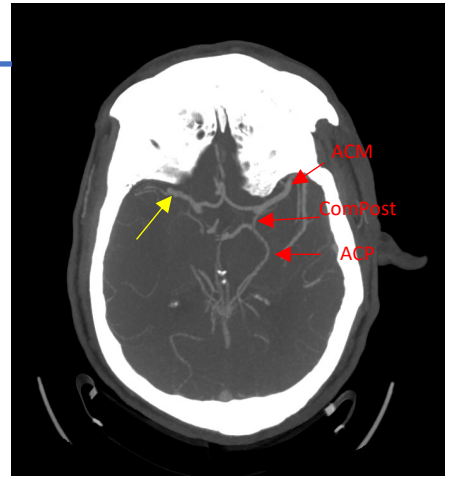


The symptoms are already evident in the native CT image with early signs of infarction visible in the right insular region (blurred gray-white matter border). On the right side image, the application using artificial intelligence highlights the pathological area in red. ASPECT score: 9.

Mechanical thrombectomy



During the thrombectomy, the opened vessel ruptured. Severe subarachnoid hemorrhage is evident (hyperdense blood in the sulci, indicated by the yellow arrow) along with contrast extravasation (denser hyperdensity, indicated by the red arrow).



The yellow arrow indicates the occluded right middle cerebral artery (MCA) on the CT angiography maximum intensity projection (MIP) reconstructions. On the left side, the normal middle cerebral artery (MCA), posterior communicating artery (PCom), and posterior cerebral artery (PCA) are visible. On the right side, the PCA arises from the MCA via the PCom (a common anatomical variant) and is also occluded

- Malformations/epilepsy
- Cerebrovascular disorders
  - Ischaemic stroke
- Head trauma
- Brain tumours
- CNS infections
- Inflammatory CNS disorders
- Neurodegenerative disorders

Radiológiai Klinika SZTE

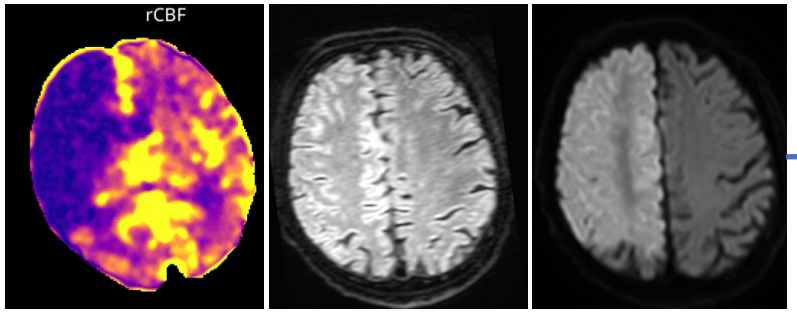
Radiology case reports

Neuroradiology

Medical History: 67-year-old male. Went to bed at 9:00 PM. At 11:30 PM, spouse noticed severe unilateral limb weakness and speech disorder. Initial CT performed at the primary care facility (3:01 AM), then transferred to a stroke center. Arrived for CT scan at 5:19 AM.

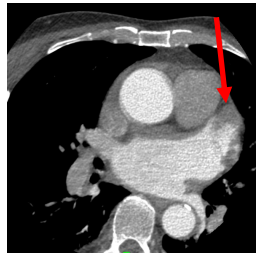
Medical History: Known atrial fibrillation and dilatative cardiomyopathy. Prescribed apixaban but hasn't taken it for several days.

Status: Eyes deviating to the right, central facial paresis on the left side, left-sided hemiplegia (0/5), hyperreflexia on the left side, left-sided Babinski sign, anosognosia. NIHSS: 23.

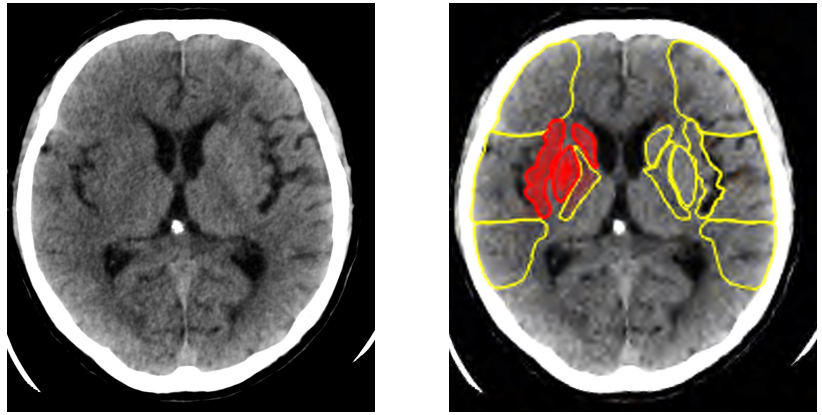


The perfusion CT scan shows a significant decrease in rCBF (left image) corresponding to the territory supplied by the right MCA (core). In the same area, diffusion restriction is visible (right image), consistent with hyperintensity in the cortex on FLAIR images (middle image). This FLAIR-DWI match (that the infarction is visible not only on the diffusion-weighted image but also on the FLAIR sequence), typically appearing 3-4 hours after vessel occlusion.

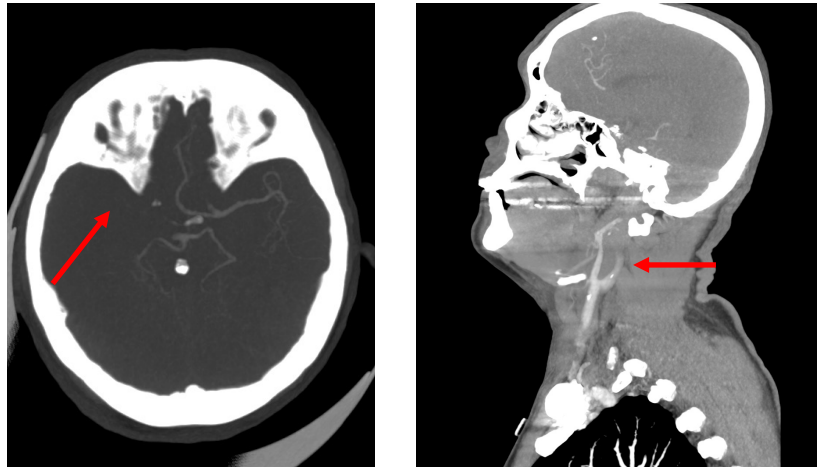
Large core, small penumbra >> No thrombolysis/thrombectomy



On the CT angiography examination, the thrombus is clearly visible in the left atrial appendage, which supposedly caused the stroke (arrow).



The symptoms are already evident in the native CT image with early signs of infarction visible in the right insular and basal ganglia regions (blurred gray-white matter border). On the right side image, AI highlights the pathological area in red. ASPECT score: 7.



On the CT angiography maximum intensity projection (MIP) reconstructions, the occlusion of the right internal carotid artery and the absence of filling in the branches of the middle cerebral artery system are clearly visible.

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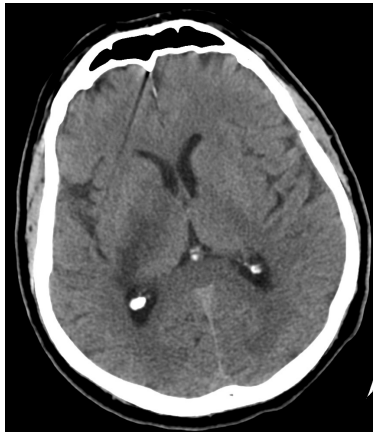
Radiology case reports

Neuroradiology

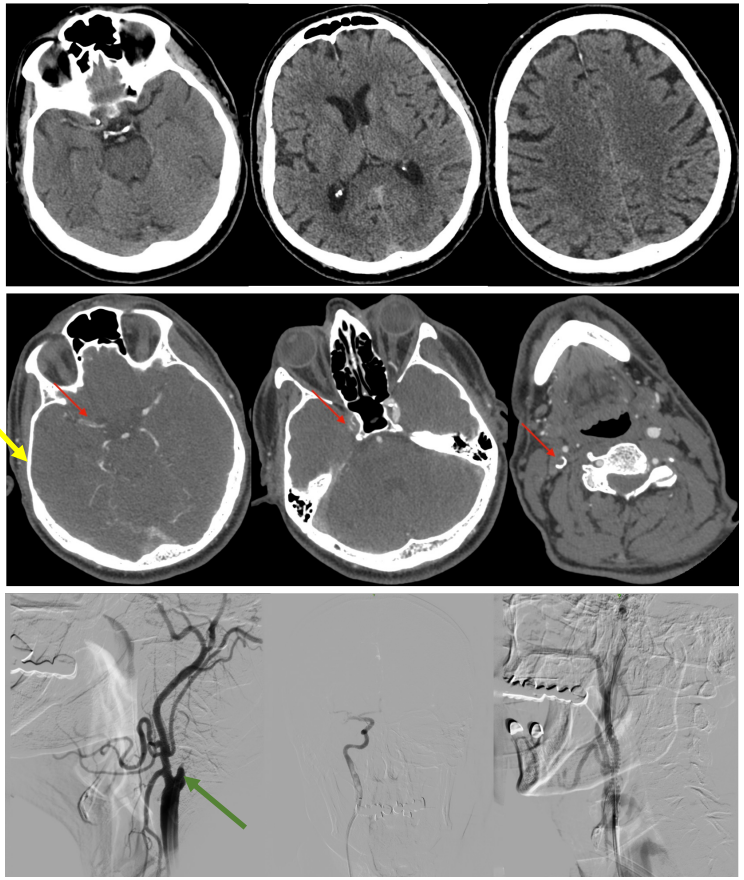
Medical History: 69-year-old male. Onset of symptoms: 9:30 AM. Arrived for CT scan at 14:14. Time elapsed since symptom onset: 4 hours and 44 minutes (beyond the 4.5-hour thrombolysis time window, within the 6-hour thrombectomy time window).
Status: Left-sided hemiparesis 3/5, left-sided central facial paresis, positive Babinski reflex, dysarthria. NIHSS: 6.

Time windows for stroke treatment

- *Large vessle occlusion >> mechanical thrombectomy: 6 hours*
- *No large vessle occlusion >> intravenous thrombolysis: 4.5 hours*



On the follow-up native CT scan performed 24 hours after the intervention, hypodensity can be observed corresponding to the thalamus and the posterior horn of the internal capsule on the right side, consistent with residual infarction.



On the non-enhanced CT images, there was no early signs of ischaemia, (ASPECT score: 10).
On the CT angiography images, the internal carotid artery is occluded after the bifurcation (red arrows). The middle cerebral artery shows partial filling via the ophthalmic artery and the anterior communicating artery (yellow arrow).
On the digital subtraction angiography (DSA) examination, the stump of the internal carotid artery is visible (green arrow) corresponding to the occlusion (bottom left image). After thrombectomy, circulation has been restored (bottom right image).

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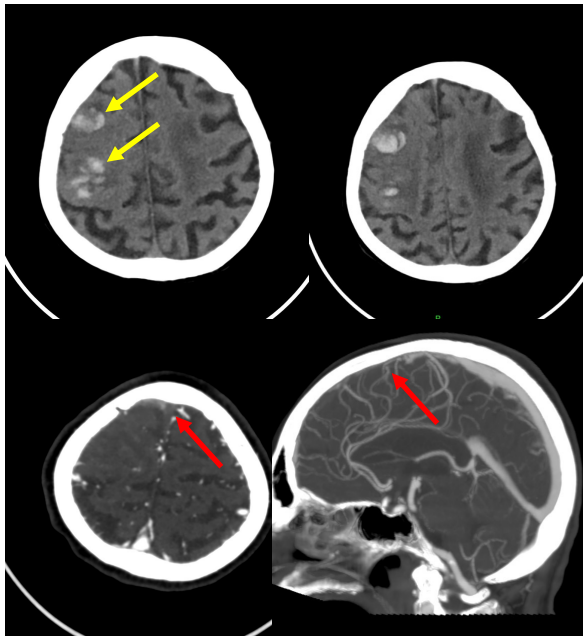
Radiology case reports

Medical History: 74-year-old female. Complained of headaches in the days leading up to the examination, which intensified. On the day of the examination, she experienced a convulsive seizure with loss of consciousness, starting with twitching of the left upper limb.
Medical History: Under investigation for rectal tumor.
Status: Glasgow Coma Scale (GCS): 8-1-2. Deep stupor. Grimaces in response to pain stimuli, pulls limbs, less so on the left side. Anisocoria, corneal reflex preserved, oculocephalic reflex absent. Babinski reflex present on both sides.

Diagnosis: **Thrombosis of the superior sagittal sinus**

Sinus thrombosis
Risk factors: Inherited prothrombotic conditions, infections (meningitis, otitis, mastoiditis, sinusitis), head trauma, vasculitis, hematologic disorders (e.g., polycythemia, thrombocytopenia, hematologic malignancies), dehydration, cancer, pregnancy, lumbar puncture.
Symptoms: Progressive headache, focal neurological deficits, altered consciousness, convulsive seizures.
Diagnosis: D-dimer (sensitive but not specific!!!), CT/MR venography, digital subtraction angiography (DSA).
Therapy: Anticoagulation (even in the presence of bleeding!), thrombolysis (?), antiepileptic drugs, analgesics, intracranial pressure reduction, decompressive craniectomy (in case of impending herniation).
Secondary prevention: Anticoagulation (with vitamin K antagonists, for 3-12 months).



Top row: Non-enhanced CT. On the right side, in a lobar location, there is a space-occupying, multifocal, hyperdense hemorrhage (yellow arrows).
Bottom row: CT venography. On the axial image on the left side, the absence of contrast enhancement is visible in the anterior part of the superior sagittal sinus (empty triangle sign). The sagittal maximum intensity projection (MIP) reconstruction also shows the lack of enhancement.

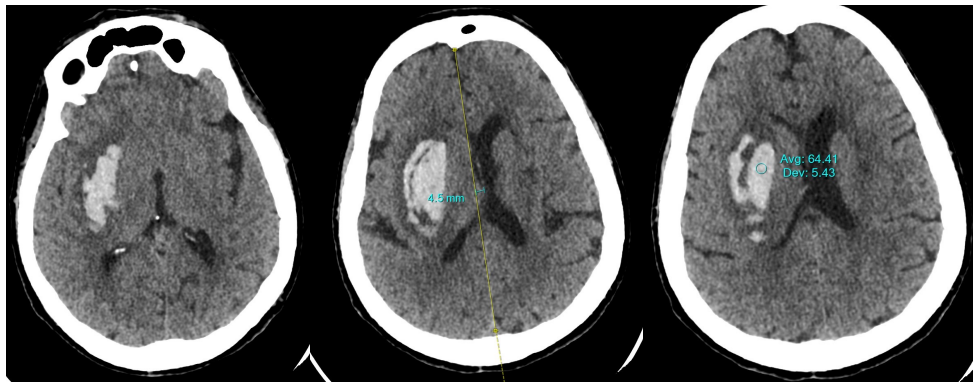
ESO guideline for the diagnosis and treament of cerebral venous thrombosis.

Radiology case reports

Medical History: 72-year-old female. Seen by relatives during breakfast at 8:00 AM. Found lying on the floor at 8:30 AM. Did not move her left upper and lower limbs. Speech was garbled.
Status: Somnolent, left-sided hemiparesis 0/5 (upper limb) 1/5 (lower limb), left-sided central facial paresis, positive Babinski reflex, dysarthria, partial gaze weakness. NIH Stroke Scale: 16.
Previous medical conditions: Hypertension, hyperlipidemia.

In case of intracranial haemorrhage, additional CT angiography/MR/DSA is required for:

- Lobar haemorrhage in patients younger than 70 years old
- Brainstem or posterior fossa haemorrhage in patients younger than 45 years old
- Brainstem or posterior fossa haemorrhage in patients aged 45-75 years old without hypertension or microvascular damage on imaging
- Intraventricular haemorrhage
- Suspicion of sinus thrombosis (clinical or imaging)



On the non-enhanced CT images, in the region of the right basal ganglia ('in loco typico') hyperdense hemorrhage is visible with mass effect and surrounded by a narrow edematous band. The midline shift is 4.5 mm. The density of the fresh hemorrhage is 60-70 Hounsfield Units (HU).

Diagnosis: **Intracerebral haemorrhage**

AHA/ASA: 2022 Guideline for the Management of Patients With Spontaneuous Intacerebral Hemorrhage

Radiology case reports

Neuroradiology

Medical History: 84-year-old female patient. On the day before admission, she felt as if something was "grabbing" her left arm. Perhaps her left arm was weaker. She bumped into things and knocked things over. On the day of admission, she was found lying on the floor, not moving her left side.
Status: Left-sided gaze weakness, left central facial paresis, left homonymous hemianopia, left-sided hemiparesis (upper limb: 0/5, lower limb: 2/5). Sensory and visual neglect. Left-sided pain and tactile hemihypoesthesia.

Modified Boston Criteria v2.0

Age: >50 years

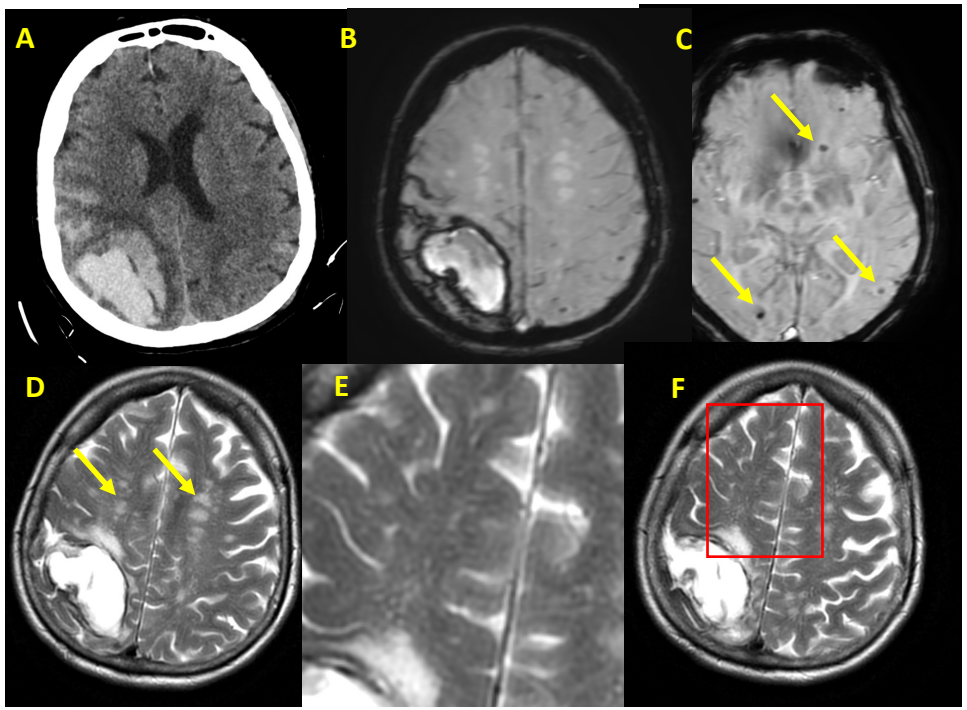
- Spontaneous intracranial haemorrhage / transient neurological symptoms / cognitive impairment-dementia ≥2 lobar haemorrhages (ICH, CMB, cSS/cSAH*)

Or

- 1 lobar haemorrhage (ICH, CMB, cSS/cSAH) + 1 white matter abnormality (CSO-PVS or WMH-MS**)

Absence of brainstem haemorrhage (ICH, CMB)
Cerebellum is not considered either lobar or brainstem.
Other causes behind the haemorrhage are excluded.

*ICH: intracranial haemorrhage, CMB: cerebral microbleeds, cSS: cortical superficial siderosis, cSAH: convexity subarachnoid haemorrhage
** CSO-PVS: centrum semiovale perivascular space dilation, WMH-MS: multispot patterned white matter lesions



A: On the non-enhanced CT image, a lobar hemorrhage in the right parietal lobe is visible, along with hyperdensity in the sulci indicating subarachnoid spread. B: On the susceptibility-weighted MR image, lobar hemorrhage and subarachnoid hemorrhage corresponding to the CT are visible. C: On the susceptibility-weighted imaging (SWI) image taken at the level of the midbrain, multiple microhemorrhages are visible in a lobar localization (arrows). D: On the T2-weighted image, dot-like T2 hyperintense lesions are visible in the corona radiata. E-F: On the magnified image, perivascular space dilation in the form of dots or lines is appearing in the corona radiata.

Diagnosis: **Cerebral amyloid angiopathy**

Boston Criteria v.2.0 for cerebral amyloid angiopathy

Radiológiai Klinika SZTE

Radiology case reports

Neuroradiology

Medical History: 54-year-old female patient. The evening before admission, she experienced sudden, severe occipital ”thunderclap” headache. The pain did not subside until the morning. In the morning, she experienced a blackout. She was held, and her head was not struck. No convulsions were observed. Confusion did not accompany the discomfort. Since then, she has been slightly slowed.

Status: Slightly slowed. Stiff neck, positive Kernig and Brudzinski signs. Right-sided Babinski reflex.

Aneurysmal subarachnoid bleeding

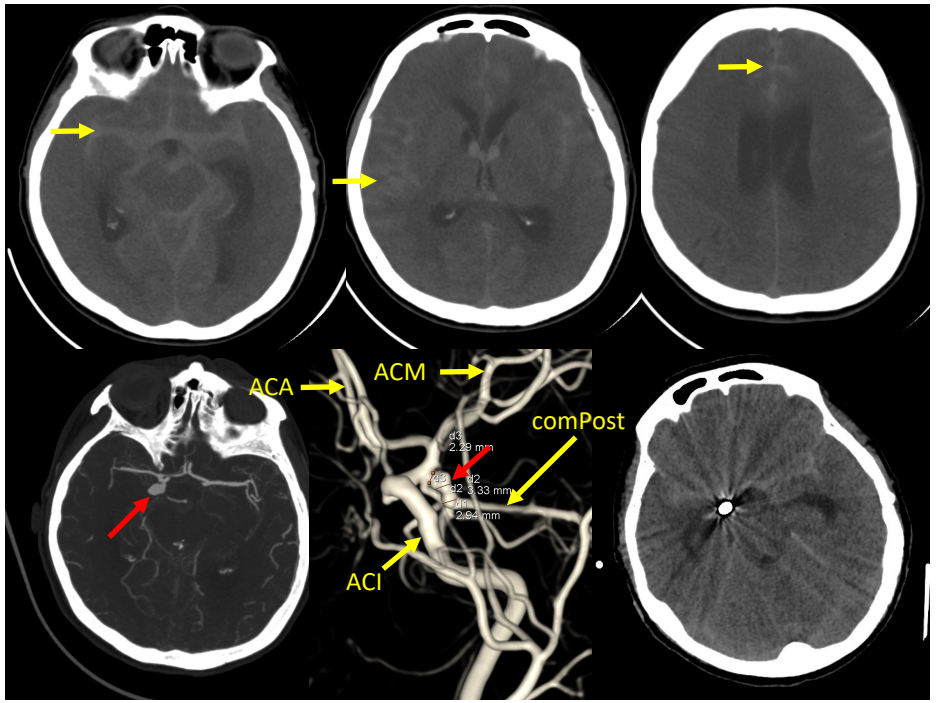
Symptoms: Sudden onset severe thunderclap headache (peaking at the beginning), neck stiffness, meningeal irritation signs, with or w/o neurological symptoms

Diagnosis:

- <6 hours: non-enhanced CT
- >6 hours: non-enhanced CT, if negative >> LP
- Proven SAH >> CT angio or DSA to identify the source of bleeding

Treatment:

- Normotension, normovolemia
- Surgical or endovascular treatment if possible within 24 hours.
- Thrombosis profilaxis (after securing the aneurysm)
- Vasospasm: CTA or TCD monitoring. Enteral nimodipine prevention.



Top row: Successive slices show hyperdensity in the basal cisterns and sulci (arrows), indicating subarachnoid haemorrhage.
Bottom row: On the CT angiography images on the left side, there is an aneurysm on the right internal carotid artery, at the origin of the posterior communicating artery. In the DSA examination, the red arrow points to the aneurysm. On the bottom right image, the coil-treated aneurysm is visible on the follow-up CT scan.

Diagnosis: **Aneurysmal subarachnoid haemorrhage**

2023 AHA/ASA Guideline for the Management of Patients with Aneurysmal Subarachnoidal Hemorrhage

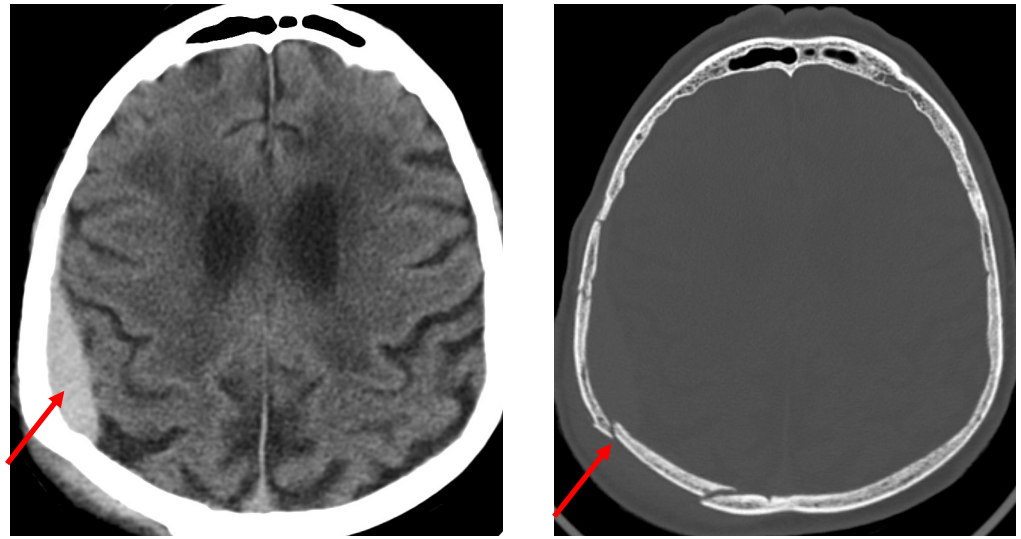
Radiológiai Klinika SZTE

Radiology case reports

Medical history: 77-year-old female. Fell at home, hit her head. Has had severe nausea since then. No loss of consciousness, remembers the event.
Previous medical history: Takes warfarin for atrial fibrillation. INR: 1.88

Status: Palm-sized swelling on the right parietal area. No focal neurological signs.

Diagnosis: **Epidural bleeding**



On the non-enhanced CT examination, a well-defined, lens-shaped, hyperdense, extra-axial, space-occupying lesion is clearly visible in the right parietal region, consistent with epidural hemorrhage. On the bone window images (right), a fracture of the parietal bone with cortical impression is visible.

American College of Radiology ACR Appropriateness Criteria: Head Trauma

Indication non-enhanced head CT in cases of acute mild head trauma if there was loss of consciousness or post-traumatic amnesia:

- Focal neurological deficit
- Vomiting
- Severe headache
- Age over 65 years
- Drug or alcohol intoxication
- Short-term memory deficit
- Bruising above the clavicle
- Post-traumatic convulsive episode
- GCS<15
- Anticoagulation

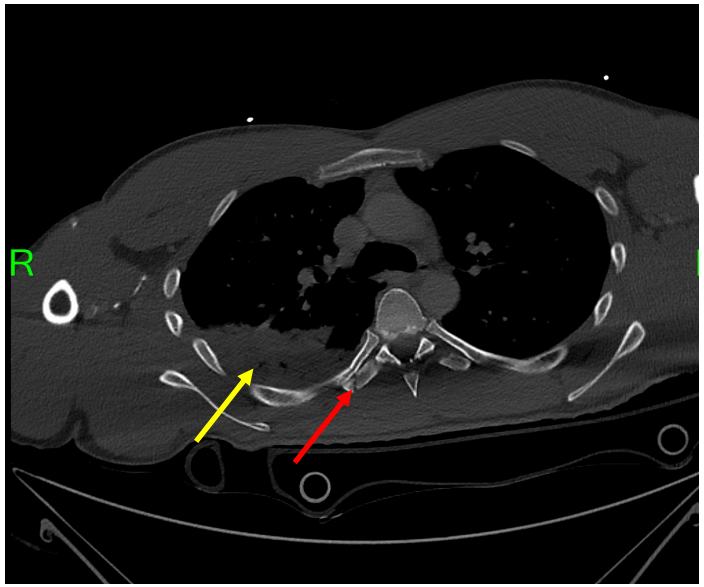
Indication for native skull CT in cases of acute mild head trauma if there was NO loss of consciousness or post-traumatic amnesia:

- Focal neurological deficit
- Vomiting
- Severe headache
- Age over 65 years
- Signs of skull base fracture
- GCS<15
- Anticoagulation
- High-risk mechanism of vehichle
  - Ejection from a car
  - Pedestrian struck by a car
  - Fall from a height greater than 1 meter or from 5 steps

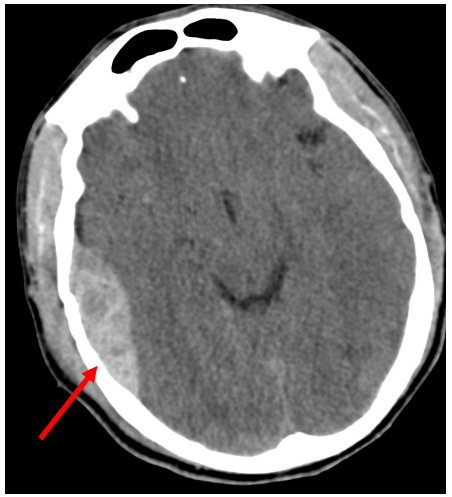
Radiology case reports

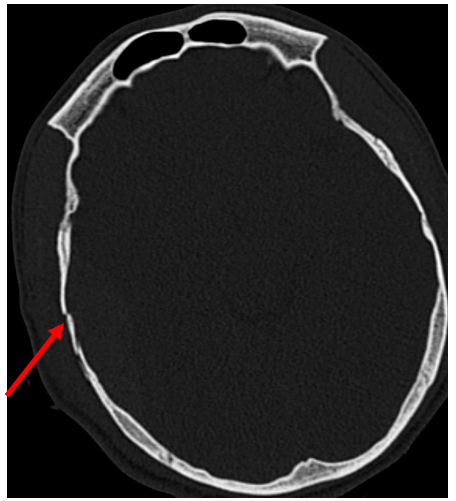
Medical history: 26-year-old male. Fell into a 4-5 meter deep well while in intoxicated, hitting his head. Vomited multiple times.
Status: GCS 13, somnolent. Blood draining from the right ear canal.

Diagnosis: **Epidural bleeding**

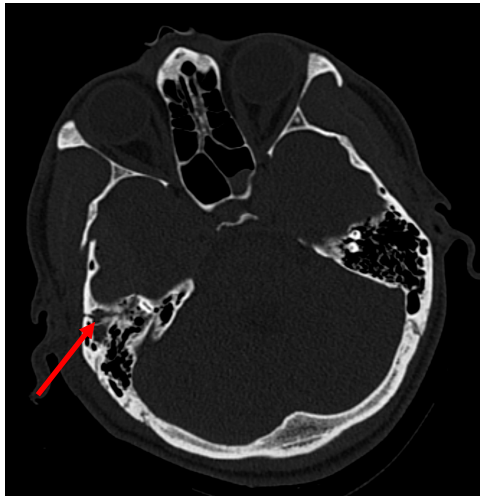


On the bone window images of the non-enhanced chest CT, a non-displaced fracture of the right transverse process of the Th.V. vertebra and the posterior arch of the rib at the same level is visible (red arrow). The decreased air content in the lung is due to pulmonary contusion (yellow arrow).





On the non-enhanced CT, a well-defined, lens-shaped, hyperdense, extra-axial, space-occupying lesion is visible in the right temporo-parietal region, consistent with epidural hemorrhage. On the bone window images (right), a fracture of the temporal bone was identified. The fracture extends to the mastoid air cells as well (lower image, arrow).

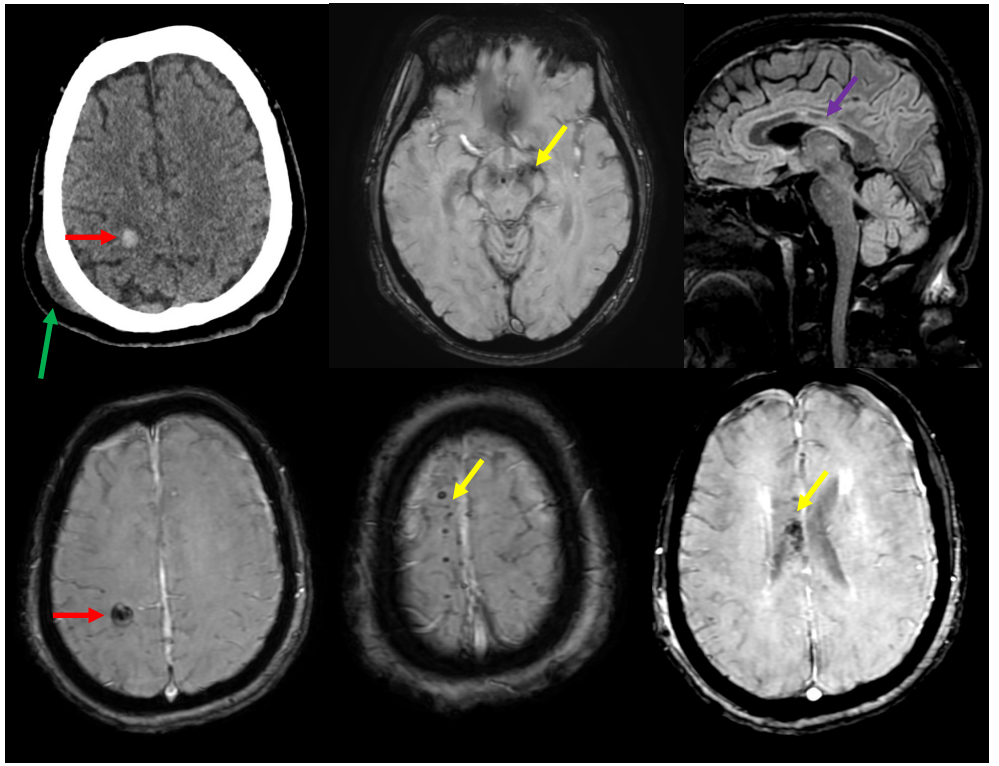


Radiology case reports

Medical history: 53-year-old female patient. Was involved in a car accident as a passenger on the highway. Lost consciousness briefly. Does not remember the events.

Status: No abnormal neurological findings.

Diagnosis: **Diffuse axonal injury**



On the non-enhanced head CT examination (upper left), a small hyperdense haemorrhage is visible in the white matter below the central sulcus (red arrow). On the susceptibility-weighted MR image, in addition to the haemorrhage seen on CT, punctate hypointensities are visible in the subcortical white matter, corpus callosum, and crus cerebri (yellow arrows), corresponding to microbleeds. A swelling of the subcutaneous tissues can be observed in the right parietal region on CT (green arrow). On the sagittal FLAIR image, the hyperintensity in the corpus callosum (purple arrow) is also indicative of axonal damage.

Diffuse axonal injury on MRI:
The diffuse axonal injury that occurs during traumatic brain injury is most sensitively detected by susceptibility-weighted MR images. Hypointense microbleeds appear in:

- the corpus callosum
- at the gray-white matter junction
- in the brainstem

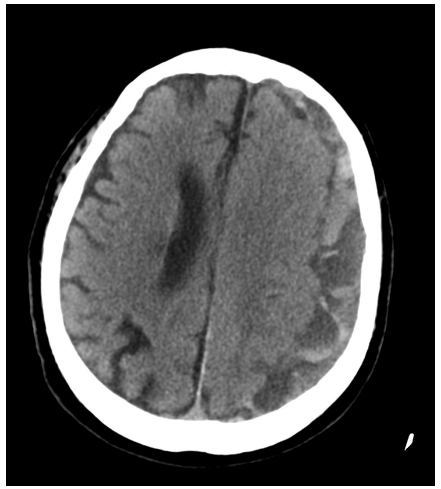
T2/FLAIR images can also demonstrate white matter damage.

Radiology case reports

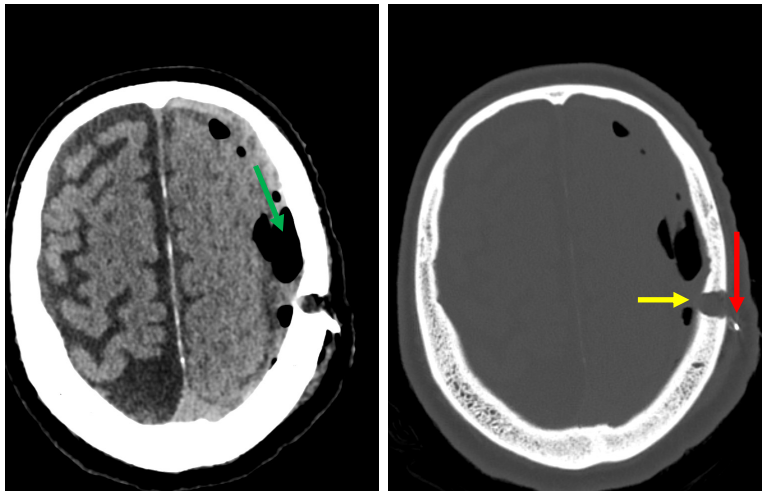
Medical history: 78-year-old male. Felt unwell while working in the garden, his legs weakened. One month prior, he had fallen and hit his head.
Status: GCS 15, mild ataxia, slightly unstable in Romberg position.
Medications: Not receiving anticoagulants or antiplatelet agents

Diagnosis: **Subdural haematoma**

Acute subdural hematoma occurs due to rupture of the bridging veins. It most commonly affects the elderly population, and anticoagulation and antiplatelet therapies significantly increase the risk. As a result of acute bleeding, inflammatory mediators are released, neoangiogenesis begins, and this often leads to repeated bleeding.



On the non-enhaced CT, the left-sided fronto-parietal subdural hemorrhage is visible. The hypodense areas indicate older bleeding, while the hyperdense areas correspond to a fresh component.



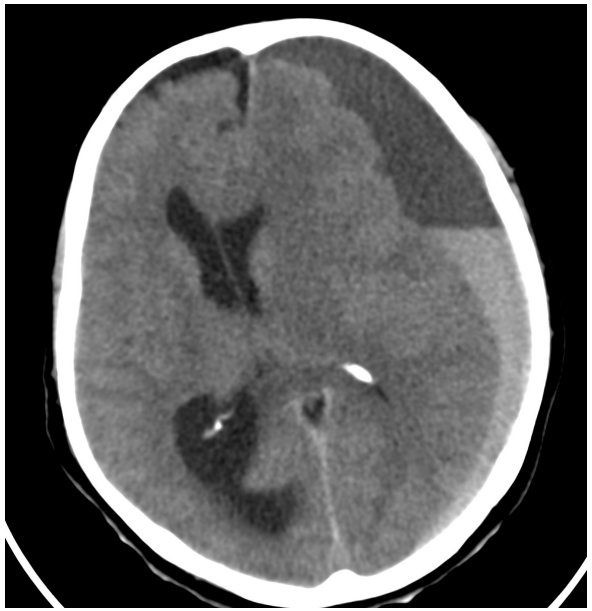
Initially, catheter embolization of the middle meningeal artery and the hematoma capsule was performed, followed by evacuation of the hematoma through a burr hole. On the lower non-enhanced soft tissue and bone window images, a decrease in the size of the subdural hematoma is visible. The green arrow indicates the postoperative air collection, the yellow arrow points to the burr hole, and the red arrow marks the subperiosteal drain.

Radiology case reports

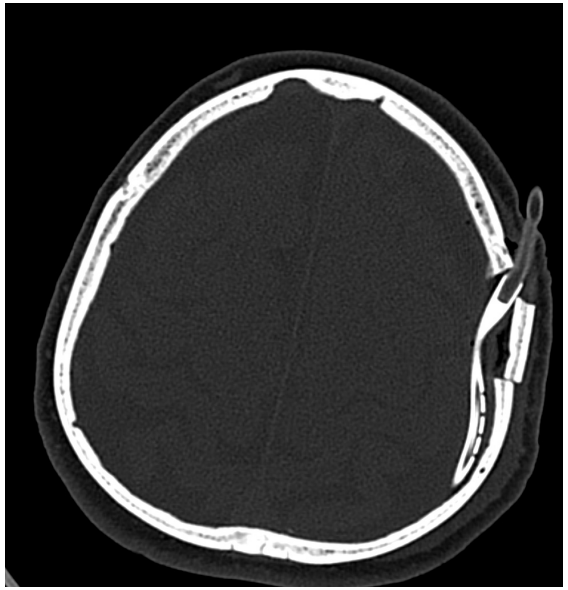
Anamnesis: 64-year-old woman. Found at home lying on the floor in her own vomit. No information regarding trauma. No signs of external injury.
Status: Somnolent, right lower limb paresis graded 3/5, positive Babinski sign.

Medications: Anticoagulated with warfarin due to multiple deep vein thromboses (INR=3.14).

Diagnosis: **Subdural haematoma**



The non-enhanced cranial CT shows a left fronto-parietal subdural hemorrhage. The hypodense areas suggest older bleeding, while the hyperdense areas correspond to fresh components. The bleeding has a significant mass effect, causing a midline shift of 14 mm.



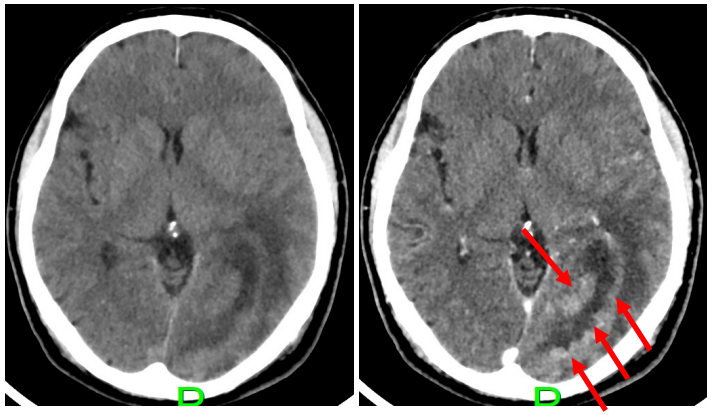
They performed a mini-craniotomy to evacuate the bleeding and inserted a subdural drain.

Radiology case reports

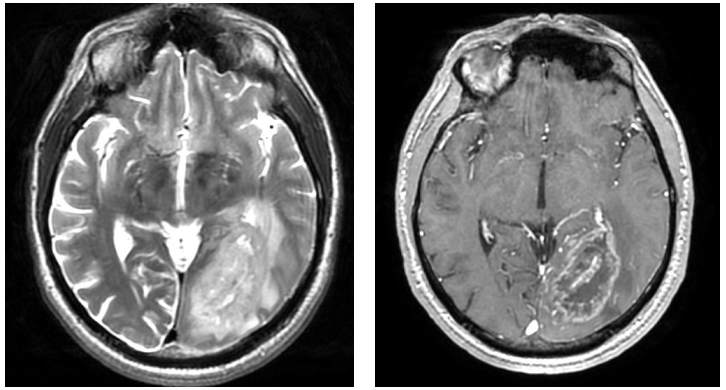
Anamnesis: 66-year-old male. He has been experiencing progressively worsening headaches for the past month, prompting a CT examination.
Status: Right-sided homonymous hemianopia.

Glioblastoma belongs to the group of adult diffuse astrocytomas. It is classified as WHO grade 4, an extremely malignant tumor. It most commonly occurs in elderly (average age at diagnosis is 65 years). It is characterized by rapid progression and a very poor prognosis (median survival of 16-18 months).
It is characterized by cellular and nuclear atypia, frequent mitotic formations, necrosis, and vascular proliferation. Genetically, it is typically IDH wild-type (diagnostic criterion).

The histopathological examination results indicate the diagnosis according to the 2021 WHO classification as follows:
Glioblastoma, IDH wild-type, WHO grade 4



In the non-enhanced head CT (left side), there is a hypodense edema extending in a finger-like manner in the left occipital lobe. On the contrast-enhanced images (right), the ring-enhancing mass representing the tumor can be seen.



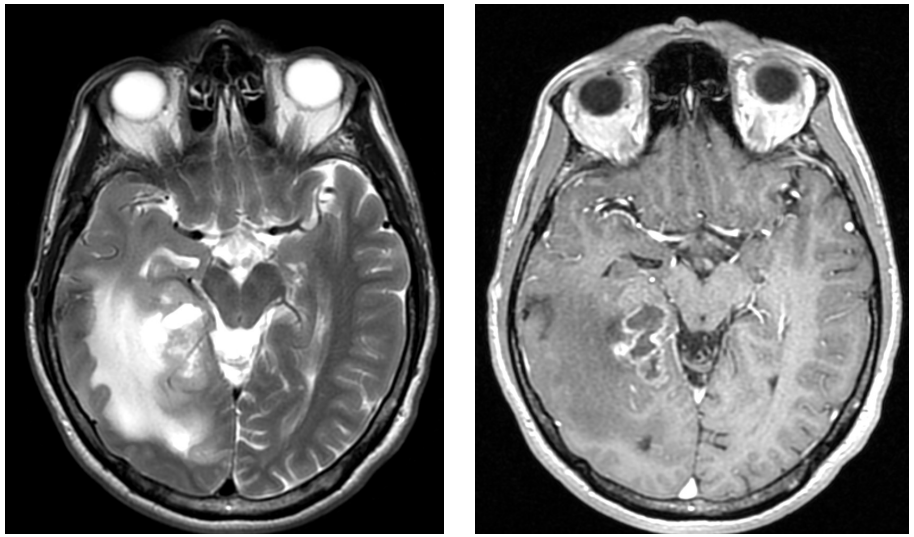
In the T2-weighted (left) MR images, around the left occipital region and the vasogenic edema can be observed around a large mass. In the post-contrast T1-weighted images, the tumor exhibits ring-enhancement.

A Radiologist's Guide to the 2021 WHO Central Nervous System Tumor Classification

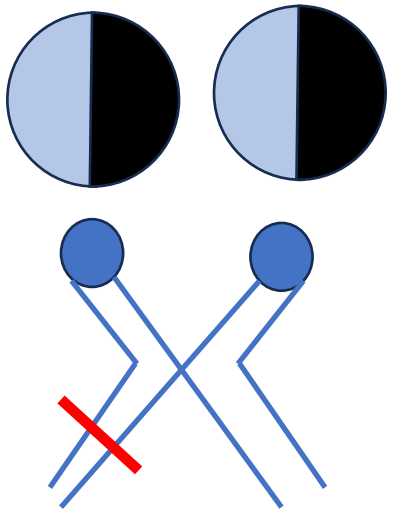
Radiology case reports

Anamnesis: 57-year-old male. He noticed left visual field loss a year ago but did not seek medical attention. Later, he experienced problems with spatial orientation and could not recognize familiar places. He also noticed memory problems. His examination began due to numbness in the upper extremities and facial drooping.

Az inferotemporalis kéreg funkciói:
-arcok felismerése (fusiform face area), zavara: prosopagnosia
-térbeli tájékozódás, zavara: topographagnosia
-tárgyak felismerése, zavara: apperceptiv agnosia
A hippocampus funkciója:
-rövidtávú memória, zavara: amnesia (patient H.M.)
A radiatio optica sérülése:
-homonim hemianopia



On the T2-weighted (left) MR images, the mass in the right temporal region and the vasogenic edema are visible. On the post-contrast T1-weighted images, the tumor shows ring-like contrast enhancement and contains cystic components.



According to the 2021 WHO classification, the diagnosis based on histopathological examination is:
Glioblastoma, IDH wild type, WHO grade 4.

Radiology case reports

Neuroradiology

Anamnesis: The 62-year-old man was examined due to dizziness and unsteadiness while walking. He experiences difficulty with fine movements in his right upper limb. He may feel less pain in his right lower limb.
Status: There is tactile hypoesthesia in the right lower limb.

Histology: The tumor cells exhibit oligodendroglial features, and the tumor border is diffuse. Perinuclear halo formation, dystrophic microcalcifications, and a capillary network reminiscent of "chicken-wire" are observed.
Immunophenotype: IDH mutant
Genetics: 1p/19q codeletion

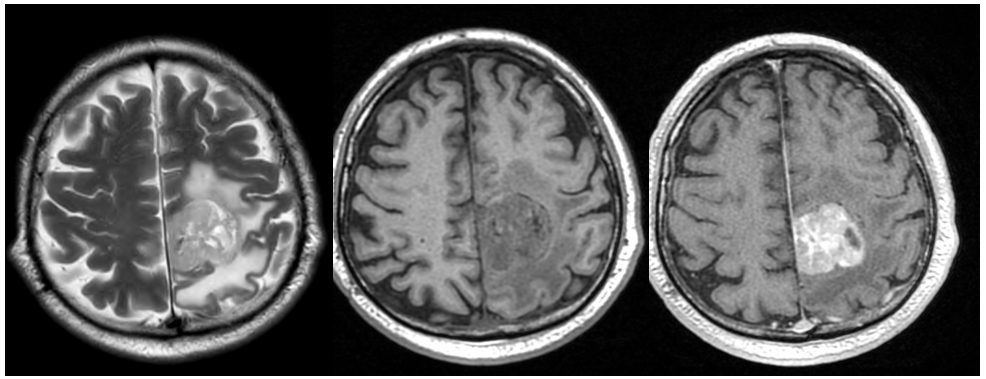
Diagnosis:
Oligodendroglioma 1p/19q codeletion WHO Gr. 2

Histopathology features of oligodendroglioma:
- Perinuclear halo (fried-egg appearance of cells)
- "Chicken-wire" pattern of capillary network

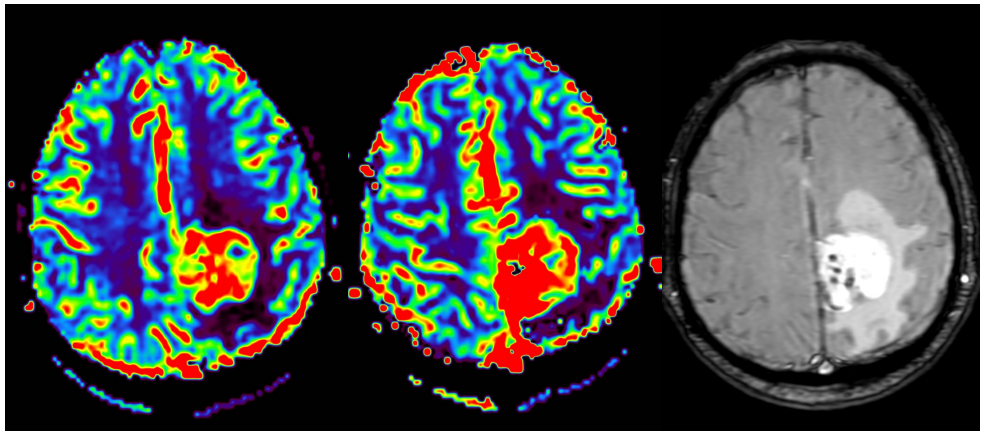
Histological features for WHO grade 2 or 3:
- Microvascular proliferation
- Necrosis
- Mitotic activity

Genetic characteristics:
- IDH mutation
- 1p/19q codeletion

Radiological features of oligodendroglioma:
- Heterogeneous appearance on T1 and T2-weighted images
- Presence of cysts
- Calcifications (88% sensitivity)
- Enhancement (partial) seen in about half of the cases.



On the T2-weighted image (left), there is a heterogeneous, hyperintense lesion in the precentral gyrus, surrounded by glove finger-shaped edema. On the non-enhanced T1-weighted images (middle), the lesion appears iso-intense to the cortex. After contrast administration, the lesion demonstrates vivid enhancement (right).



The perfusion MRI (left CBV, middle CBF) shows hyperperfusion corresponding to the tumor. On the susceptibility-weighted image, a band-like hypointensity indicating calcification is observed in the center of the tumor, which is characteristic of oligodendrogliomas.

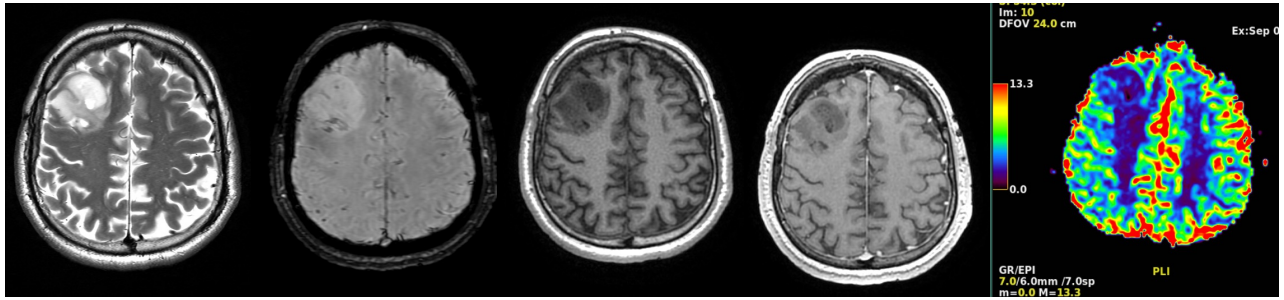
Radiológiai Klinika SZTE

Radiology case reports

Neuroradiology

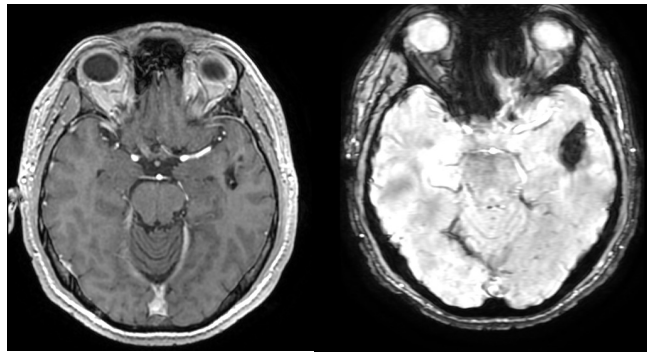
Anamnesis: 42-year-old male. His left upper limb became cramped and felt weak. On another occasion, he experienced a secondary generalized epileptic seizure starting with stiffening and then jerking of the left upper limb.
Status: Negative.

Diagnosis:
Oligodendroglioma 1p/19q codeletion WHO Gr. 2



On the T2-weighted images (left), there is a relatively sharp-edged, heterogeneous lesion visible in the right frontal lobe, at the gray-white matter interface. On the susceptibility-weighted images (second from the left), hypointense focal areas correspond to calcifications. On the non-enhanced T1-weighted image, the lesion appears slightly hypointense compared to the gray matter, with heterogeneity. On the post-contrast T1-weighted images (fourth from the left), the lesion shows faint, partial enhancement. On the perfusion CBV maps, hypoperfusion can be observed.

On the left side, in the temporal pole, a lesion independent of the above-mentioned tumor is visible. On the susceptibility-weighted images (right), a hypointense lesion causing a booming artifact is observed. No abnormal contrast enhancement is seen on the post-contrast T1-weighted images.
Diagnosis: Cavernoma.

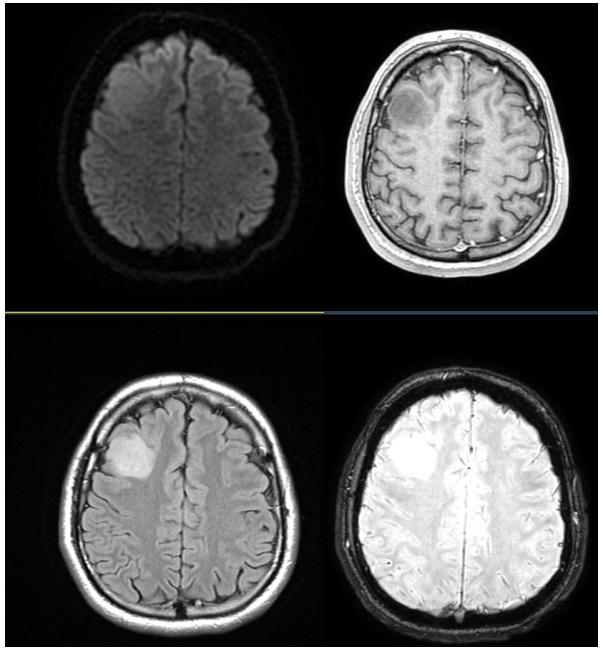


Radiológiai Klinika SZTE

Radiology case reports

Anamnesis: 35-year-old male. The patient, who stutters, perceived his stuttering to be worsening. He felt coordination disturbance and weakness in his left upper extremity, which later resolved, leaving mild sensory impairment in the left upper limb.
Status: Hypoesthesia in the left upper limb.

Diagnosis:
Oligodendroglioma 1p/19q codeletion WHO Gr. 2

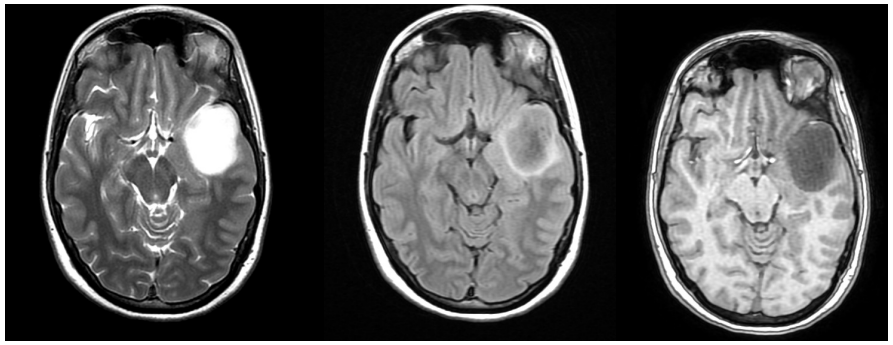


In the right frontal lobe, directly beneath the cortex, there is a lesion visible as hypointense on T1-weighted (upper right) and hyperintense on T2/FLAIR (lower left) images. No contrast enhancement is observed (upper right). Diffusion-weighted imaging (upper left) shows no diffusion restriction. Susceptibility-weighted imaging (lower right) reveals no evidence of hemorrhage or calcification.

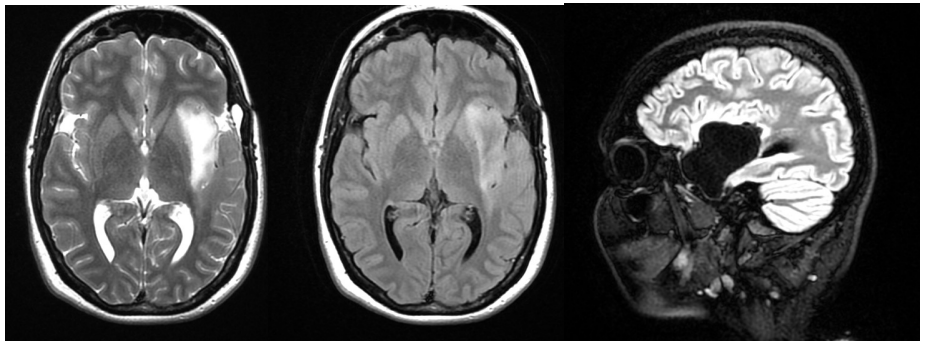
Radiology case reports

Anamnesis: 34-year-old pregnant woman (30 weeks gestation). During the night, her husband woke up to find her stiffened, followed by convulsions. After the seizure, she was unresponsive for about 15 minutes.
Status: Unremarkable.

Diagnosis:
Astrocytoma, IDH mutant, WHO Gr. 2



From left to right: T2-weighted, FLAIR and post-contrast T1-weighted images. In the left temporal pole, a mass with high signal intensity is visible on the T2-weighted image. On the FLAIR image, there is reduced signal intensity in the center of the tumor due to the inversion pulse effect, while high signal intensity remains at the periphery (T2-FLAIR mismatch). The tumor does not show significant contrast enhancement.



The tumor was observed for three years, after which growth was detected. On the left T2-weighted and middle FLAIR images, the expansion of the tumor towards the left insula is visible. On the right sagittal FLAIR image, the post-operative condition is visible.

The T2-FLAIR mismatch
A phenomenon occurs in well-demarcated tumors, which show homogeneous hyperintensity on T2-weighted images, central signal decrease, and a thin remaining hyperintense rim on FLAIR sequences. The appearance of this phenomenon is attributed to the extremely long MRI T2 relaxation time of IDH-mutant gliomas.
This feature is highly specific for IDH-mutant astrocytomas .

Radiology case reports

Anamnesis: 65 years old male. His symptoms began two weeks prior to admission in the form of general weakness. A week before his admission, he experienced headache, slurred speech, drooping of the left corner of the mouth, accompanied by nausea and vomiting. He also felt weakness in his left limbs, with his right leg lagging behind when walking.
Status: Left central facial paresis, dysarthria, left hemihypaesthesia, left hemiparesis (4/5), Babinski reflex on both sides, ataxia on the right.

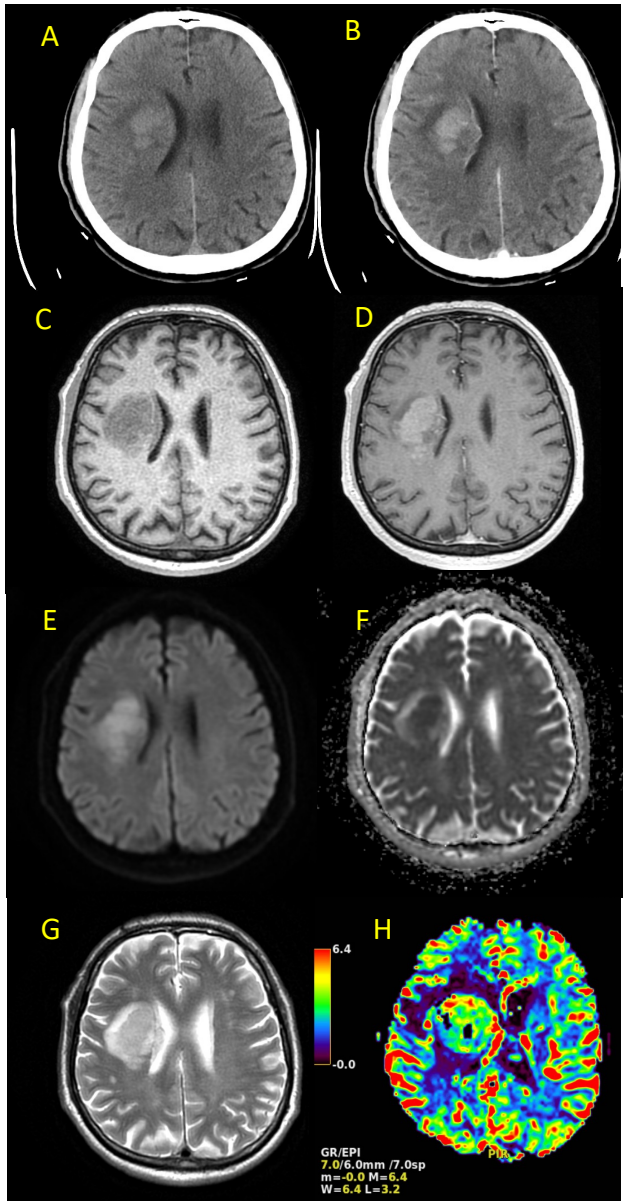
Diagnosis: Histopathology confirmed **primary central nervous system, diffuse large B-cell lymphoma**

Imaging characteristics of primary CNS lymphomas:

- Hyperattenuating enhancing mass on the CT
- T1 hypointense
- Variable on T2
- Homogenous vivid enhancement
- Restricted diffusion
- Foci of haemorrhage is common on SWI

IPCG consensus recommendations for MRI and PET imaging of primary CNS lymphoma

EANO guidelines for treatment of primary CNS lymphoma



Non-enhanced (A) and contrast enhanced (B) CT scans, showing a mildly hyperdense, enhancing mass in the right corona radiata. Same lesion is shown on the non-enhanced and postcontrast T1 weighted images. The hypointense lesions shows considerable enhancement. Diffusion weighted image (E) and the ADC map (F) indicate restricted diffusion in the tumor. There is only a modest surrounding edema around the hyperintense mass on the T2 weighted image. CBV map (H) from the MR perfusion study a few weeks later shows a modestly increased perfusion in the tumor.

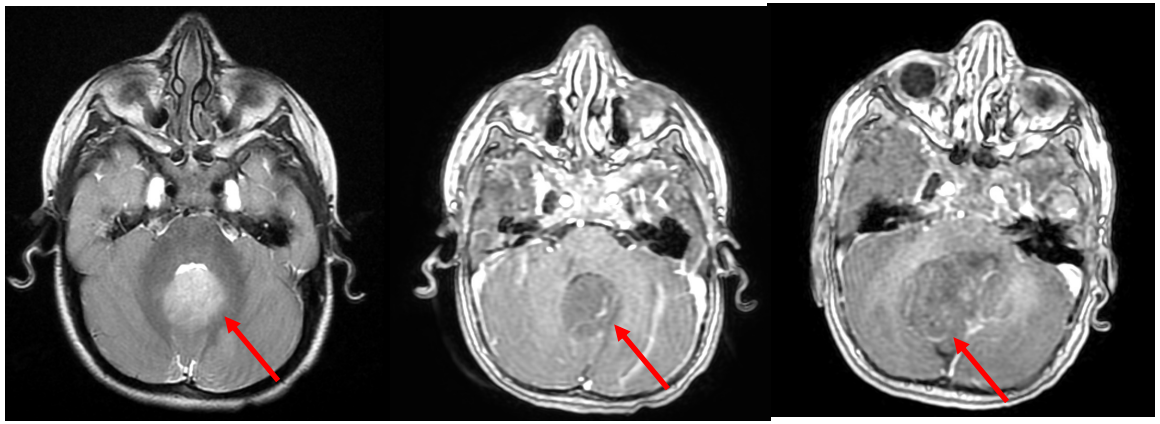
Radiológiai Klinika
SZTE

Radiology case reports

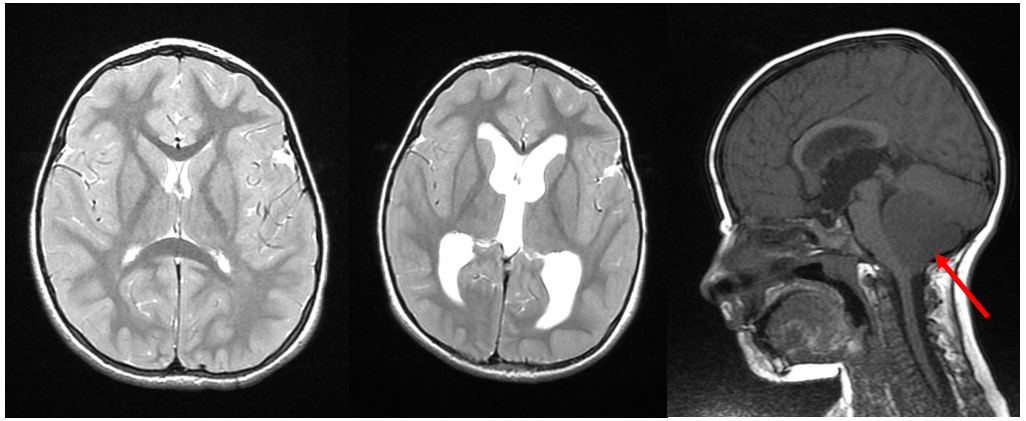
Anamnesis: 6-year-old boy with Rubinstein-Taybi syndrome, identified with a tumor on MRI examination related to the syndrome. There were no new symptoms associated with the occipital tumor. Surgery was not performed. Six months later, his gait became unsteady. A follow-up MRI was then performed.
Status: Somato-mental retardation. Ataxic gait..

Diagnosis: **Medulloblastoma**

Medulloblastoma
An embryonal tumor of the cerebellum. It has four subgroups with different genetic alterations, age of onset, and prognosis.
Epidemiology: Represents 63% of childhood intracranial tumors. Typically manifests around 6 years of age.
Symptoms: Headache, nausea, coordination problems, signs of increased intracranial pressure.
Imaging: Cerebellar tumor, usually midline, compressing the fourth ventricle, sometimes invading the brainstem. Typically shows heterogeneous contrast enhancement and diffusion restriction. Often causes hydrocephalus. Often metastasizes, hence imaging of the entire neuraxis is necessary.
Treatment: Standard therapy (maximal surgical resection + chemotherapy + radiation therapy for ages 3 and above) provides 70-85% 5-year survival.



On the left side the two images (T2-weighted and post-contrast T1-weighted), a mass can be seen in the midline of the cerebellum, narrowing the fourth ventricle, appearing hyperintense on T2 and hypointense on T1, with no contrast enhancement. On the post-contrast T1-weighted image taken six months later, the tumor has approximately doubled in size.



On the axial T2-weighted images taken six months apart (left: earlier, right: later), hydrocephalus is developed, caused by the tumour filling the fourth ventricle (arrow in the sagittal T1-weighted image).

Radiológiai Klinika SZTE

Radiology case reports

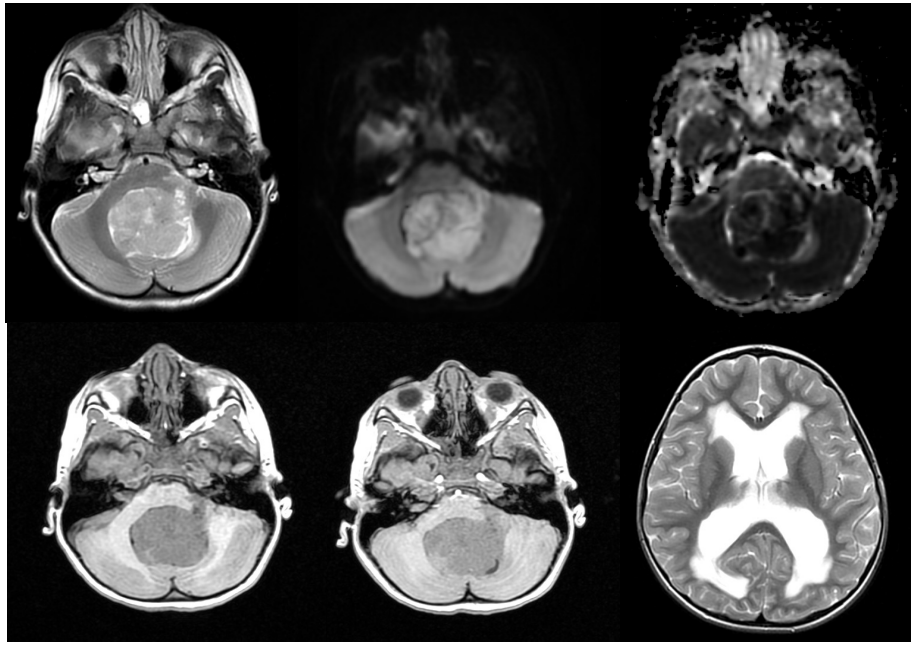
Medical history: 4-year-old girl. Recurring vomiting, walking disorder, balance disorder, and eye movement deficit over one week.

Diagnosis: **Medulloblastoma**
WHO Gr. II, Non-WNT/non-SHH Group 3.
”small round-cell tumor”

Medulloblastomas

Medulloblastomas can be classified into four groups:

- *WNT group:* Accounts for 10% of all cases. Occurs after 4 years of age (can also occur in adults). Equal gender distribution (1:1 ratio). Rarely metastasizes. Excellent prognosis.
- *SHH group:* Occurs in children under 3 years old and in individuals over 16 years old. Slightly more common in males.
- *Group 3:* Represents 25% of all medulloblastomas. Common in infants and early childhood. Often metastasizes. Five-year survival rate is less than 60%.
- *Group 4:* Represents 35-40% of all medulloblastomas. Most common in adolescents. Male-to-female ratio is 3:1. By the time of diagnosis, about one-third already have metastases. Survival rate is moderate.



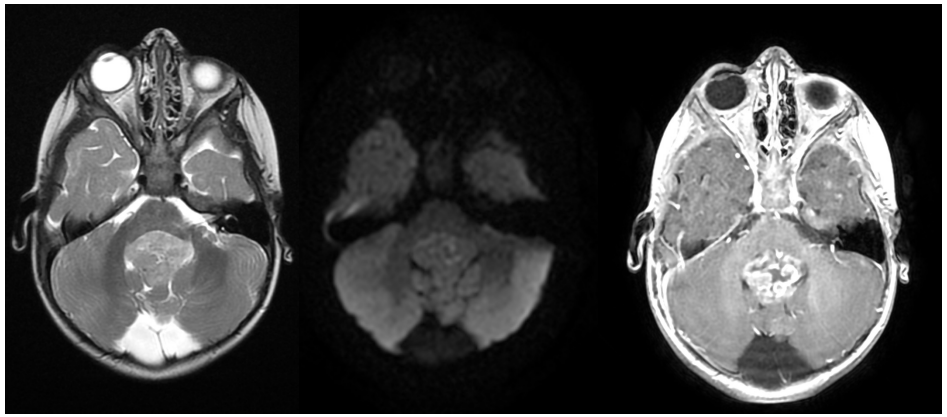
In the cerebellum, a midline lesion is visible protruding into the fourth ventricle, showing T2 hyperintensity, T1 hypointensity, and diffusion restriction. On the axial T2-weighted image at the level of the lateral ventricles (bottom right), consequential hydrocephalus and periventricular T2 hyperintensity is visible.

Radiology case reports

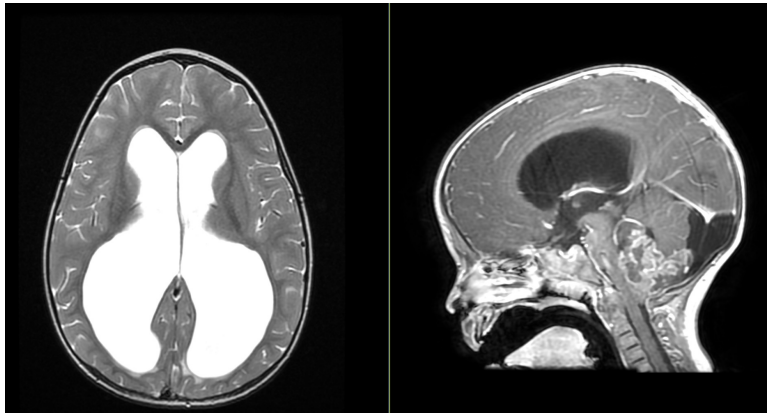
Neuroradiology

Anamnesis: 1.5 old boy. In the past weeks, he has been vomiting several times in the mornings. He refuses to eat or drink. Appears drowsy. No fever reported.

Diagnosis:
Anaplastic ependymoma, WHO Gr.III.



From left to right: T2-weighted, diffusion-weighted, post-contrast T1-weighted images. In the midline, infratentorially, a well-defined, heterogeneous, T2 hyperintense tumor with heterogeneous contrast enhancement is observed, extending into the fourth ventricle. There is no diffusion restriction (in contrast to medulloblastoma).



On the axial T2-weighted image (right), dilation of the supratentorial ventricular system is visible. On the sagittal post-contrast T1-weighted image, it can be seen that the tumor compressing the fourth ventricle causes the obstruction of cerebrospinal fluid flow.

Ependymoma

Ependymomas are neuroepithelial tumors that arise from ependymal cells lining the ventricles, the central canal of the spinal cord, or cortical remnants.

Localization: Supratentorial, infratentorial, or spinal.

Epidemiology: They account for 1.6-1.8% of central nervous system tumors. They are more common in children, with intracranial predominance in childhood and spinal predominance in adulthood.

Imaging: They typically present as well-defined lesions with heterogeneous signal intensity on T1 and T2-weighted images, along with variable and heterogeneous enhancement on contrast-enhanced images.

Treatment: The treatment typically involves maximal surgical resection, followed by chemotherapy and radiation therapy when feasible.

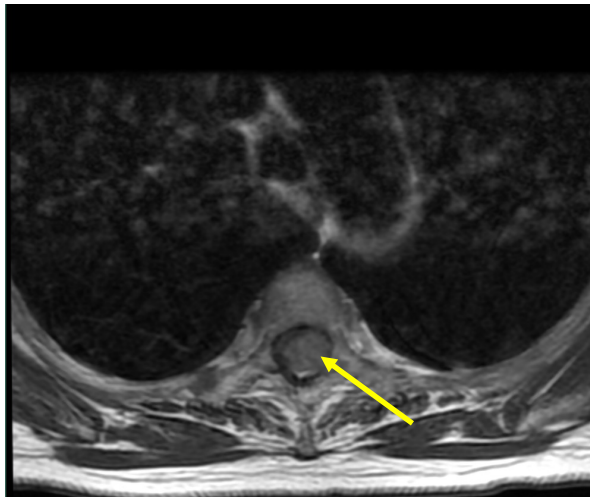
Radiológiai Klinika SZTE

Radiology case reports

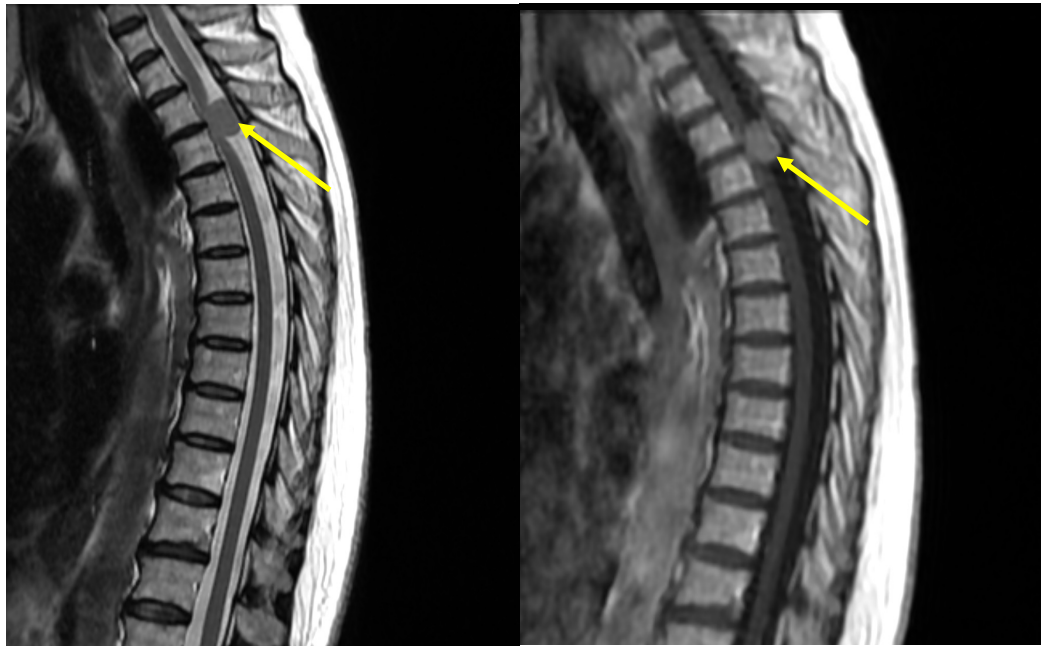
Anamnesis: 63-year-old female patient. Initially felt a belt-like numbness, which later progressed to involve both lower extremities. A week before the examination, she experienced weakness in her legs and urinary incontinence.

Status: Impaired joint position sensation in the lower extremity on the left side. Impaired sensation of heat and pain in the lower extremity on the right side. Left lower limb paresis (3/5). Left-sided Babinski reflex. Hyperreflexia in the left lower limb. Urinary incontinence. Uses a walker for mobility.

Diagnosis: **Memingeoma WHO Gr. I.**



Axial post-contrast T1-weighted images. The tumor displaces and compresses the medulla to the right.



Sagittal T2-weighted and post-contrast T1-weighted images. At the level of the ThII-ThIII intervertebral disc, an extra-axial, sharp-bordered, enhancing mass is visible in the spinal canal.

Brown-Sequard syndrome:

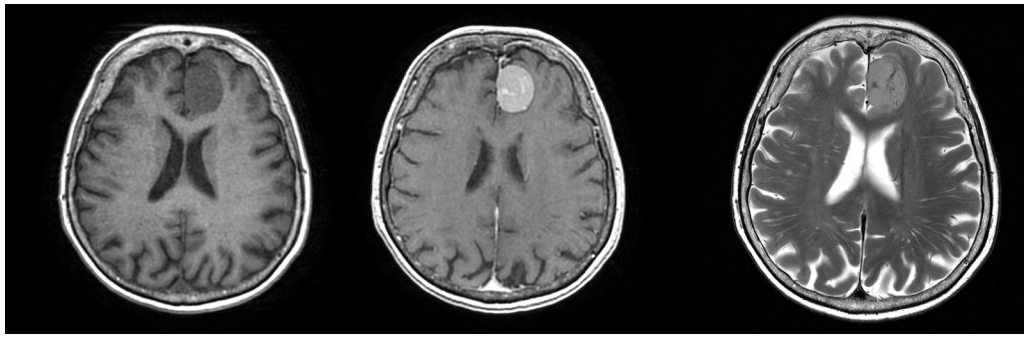
Spinal cord hemisection:

- Contralaterally, distal to the lesion, loss of temperature and pain sensation.
- Ipsilaterally, distal to the lesion, loss of proprioception and vibration sensation.
- At the level of the lesion, ipsilaterally, anesthesia affecting all sensory modalities.
- Ipsilaterally, distal to the lesion, paresis and long tract signs.

Radiology case reports

Anamnesis: 45-year-old female patient. Investigation started due to headache.
Status: Nothing remarkable.

Diagnosis: **Meningeoma**

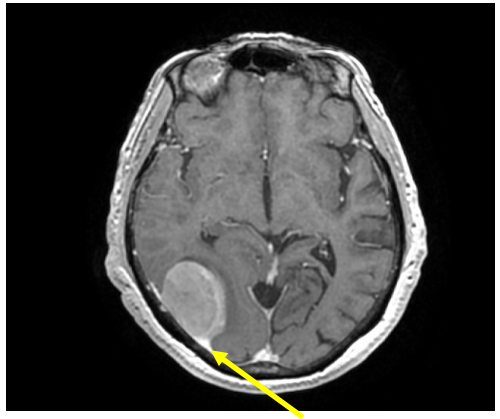


Axial non-enhanced and post-contrast T1 as well as T2 weighted images. On the left side, in a parafalcine location, there is an extra-axial lesion with a sharp border, attached to the dura, showing homogeneous contrast enhancement.

Anamnesis: 70-year-old female patient. MRI examination was performed due to balance disorder. The patient reports a recent deterioration in her vision.
Status: On the right side, there is a small visual field narrowing nasally.

Diagnosis: **Meningeoma**

On the right side, there is an extra-axial, dura-based lesion with homogeneous contrast enhancement visible in the occipito-temporal region. The arrow indicates the dural tail sign.

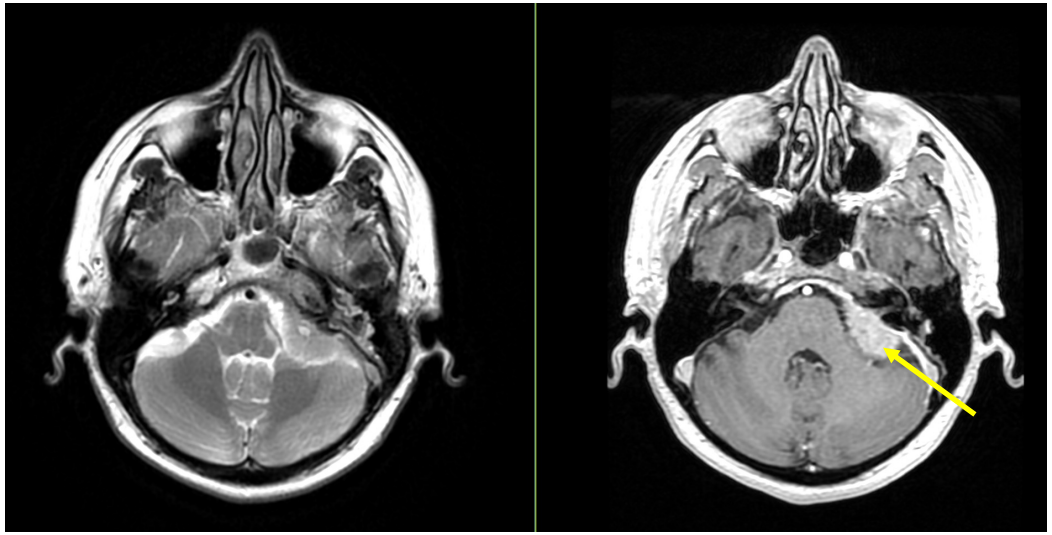


Dural-tail sign
The dural tail sign refers to thickening and enhancement of the dura. It is most commonly seen in association with meningiomas. It is thought to be caused by venous stasis, but in many cases, the tumor also infiltrates the dura in this area.

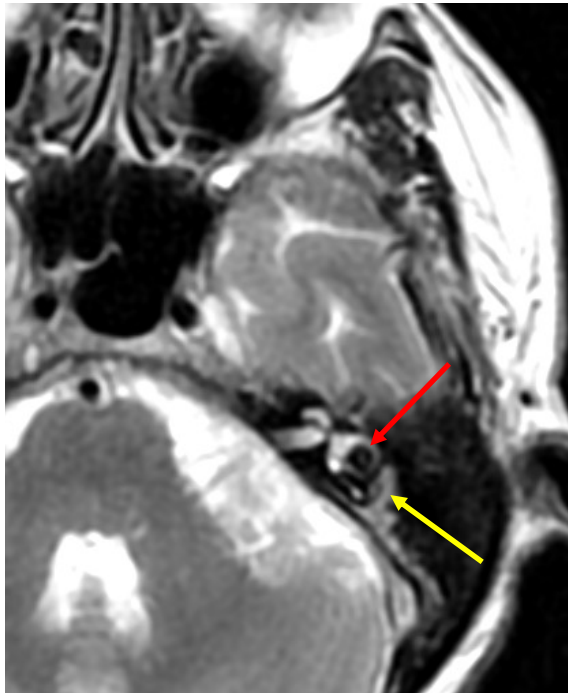
Radiology case reports

Anamnesis: The examination of the 63-year-old female patient began due to sensorineural hearing loss on the left side. It showed a type B tympanogram. Tissue samples were taken from the middle ear during paracentesis, confirming a meningeothelial meningioma.

Diagnosis: **Memingeoma WHO Gr. I.**



Axial T2 and post-contrast T1 weighted images. On the left side, an extra-axial lesion, associated with the dura, extending into the internal acoustic meatus, is visible in the cerebellopontine angle. The tumor does not widen the internal auditory canal.

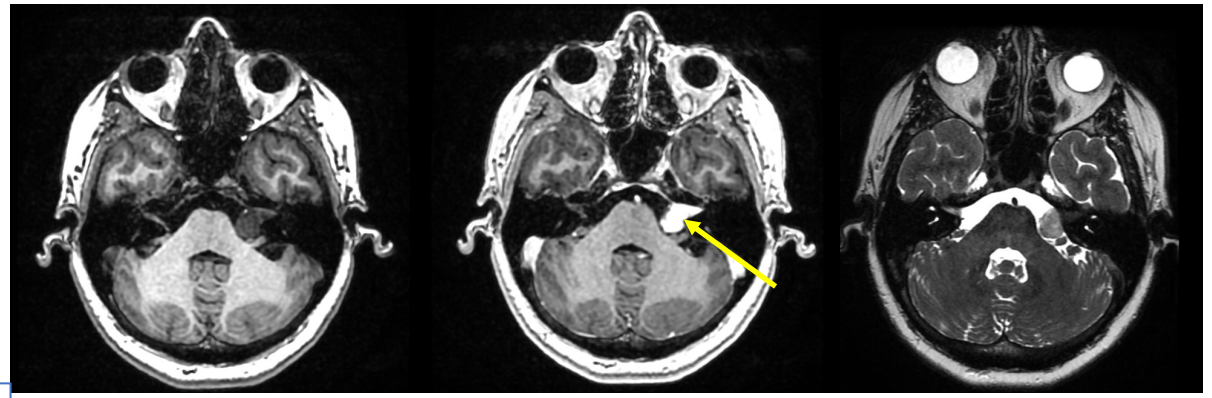


On the axial T2-weighted images, a part of the tumor extending into the middle ear cavity is visible (yellow arrow). The red arrow indicates the semicircular canal.

Radiology case reports

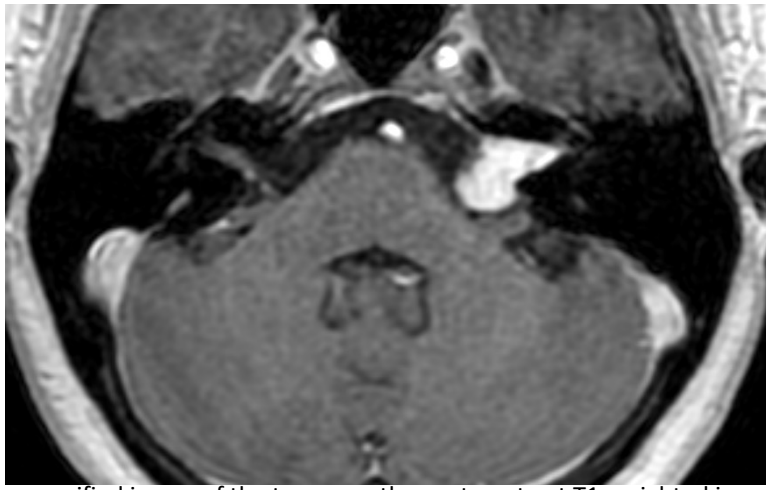
Anamnesis: A 55-year-old female patient underwent an MRI examination due to progressive hearing loss in the left ear.
Status: Sensorineural hearing loss on the left side.

Diagnosis: **Vestibularis Schwannoma**



Axial non-enhanced and post-contrast T1-weighted, as well as T2-weighted images. On the left side, an extra-axial, contrast-enhancing mass lesion is visible in the cerebellopontine angle, growing outward from the internal acoustic meatus. The tumor widens the internal auditory canal. Compare with cerebellopontine angle meningioma!

Vestibular Schwannoma / Acoustic Neuroma
Symptoms: Unilateral sensorineural hearing loss
Epidemiology: The third most common benign intracranial tumor after meningioma and pituitary adenoma. It most commonly affects the 60-70 age group.
Imaging: Tumor showing intense contrast enhancement in the internal auditory canal or protruding from it. Cystic degeneration may occur. The internal auditory meatus may widen. In the cerebellopontine angle, it may compress the brainstem and other cranial nerves.
Treatment: If small, observation. If large, surgical treatment. There are a few studies on radiosurgery.



The magnified image of the tumor on the post-contrast T1-weighted image.

EANO guideline on the diagnosis and treatment of vestibular schwannoma

Radiology case reports

Neuroradiology

Anamnesis: 32-year-old female patient. Being treated for a superficial and nodular melanoma (pT3a, Clark level: III, Breslow depth: 3.040mm, mitotic activity: 6-9/mm2, no ulceration, BRAF positive) developed on the basis of a nevus in the occipital area. She underwent surgery previously for neck lymph node and lung metastases. Metastases were identified on regular cranial MRI. She did not present with any neurological symptoms.
Neurological status: Negative

Diagnosis: **Melanoma malignum metastases**

Diagnosis of metastases of malignant melanoma:

- Ultrasonography of locoregional lymph nodes: Stage IB or higher.
- Contrast-enhanced chest-abdomen CT or PET: Stage III or higher.
- Contrast-enhanced brain MRI: Stage III or higher.

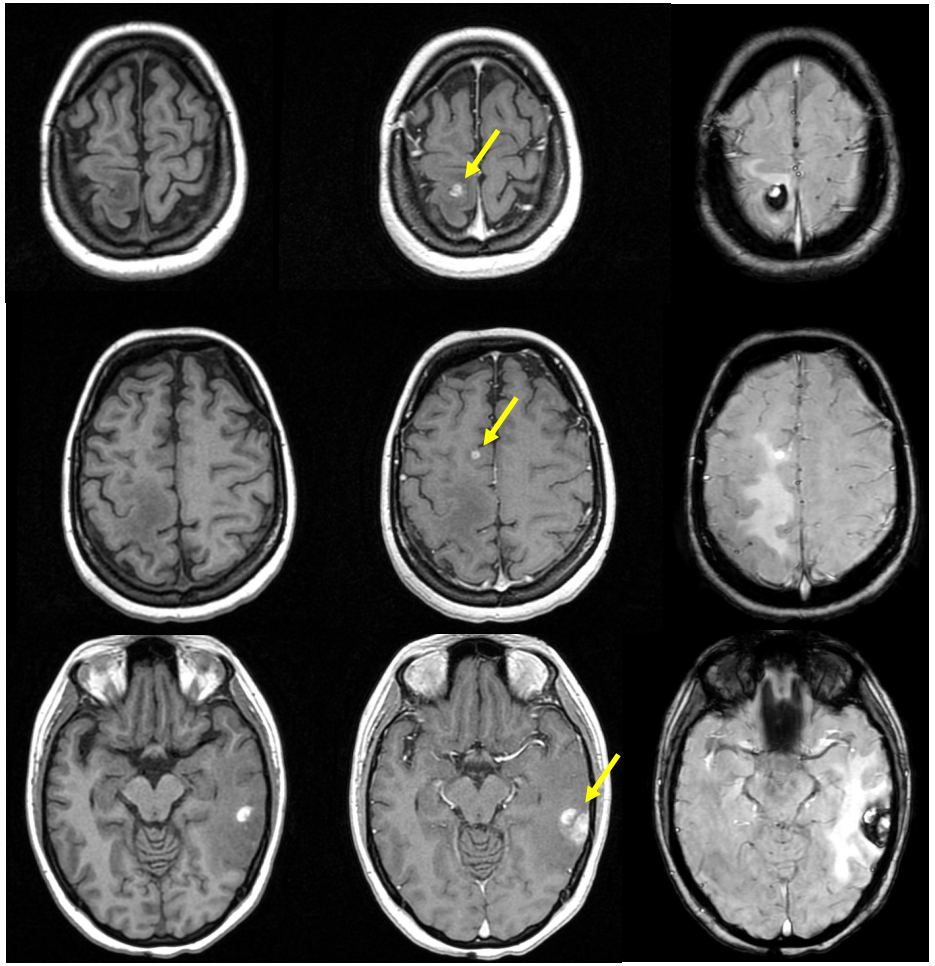
During follow-up, metastases of malignant melanoma should be monitored as follows:

- Ultrasonography of lymph nodes: Every 3-6 months above stage I.
- Neck, chest, abdomen, pelvis CT/PET, and brain MRI: every 3-6 months.

Stages of malignant melanoma (simplified):

I: Tumor thickness is less than 2 mm.
II: Thickness greater than 2 mm and no lymph node involvement.
III: Lymph node involvement.
IV: Distant metastasis.

[European consensus-based interdisciplinary guideline for melanoma. Part 1: Diagnostics: Update 2022]



From left to right: non-enhanced, post-contrast T1-weighted, and susceptibility-weighted images. Arrows indicate the metastases on the post-contrast images. The often seen hypointensity on the SWI images corresponding to the metastases is due to bleeding. The hyperintensity seen on the lowest non-enhanced T1 image indicates bleeding younger than one month. Melanoma metastases frequently appear at the gray-white matter interface.

Radiológiai Klinika SZTE

Radiology case reports

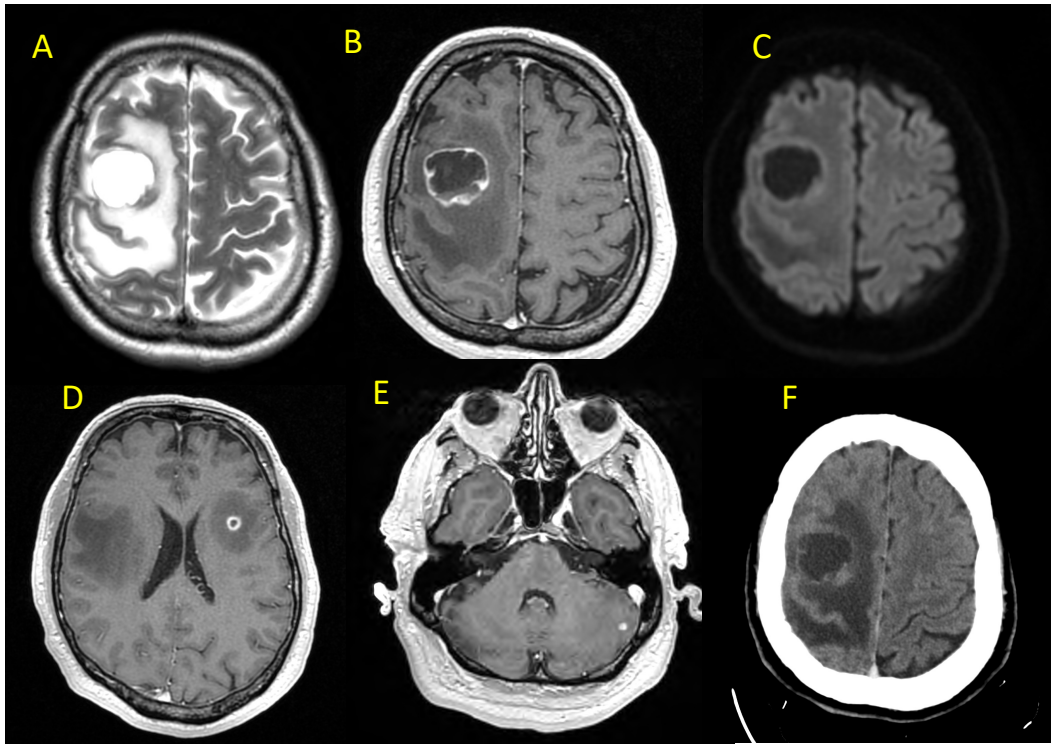
Anamnesis: 67-year-old male. Two years ago, lung adenocarcinoma was diagnosed. Following left upper lobectomy and adjuvant chemotherapy, left-sided limb weakness developed one and a half years later.
Neurological status: Mild left-sided hemiparesis (4/5).

Diagnosis: **Lung adenocarcinoma metastases**

Brain metastases:
Brain metastases can be found in nearly 30% of all cancer patients.
Radiological characteristics: Multiplex (often) ring-enhancing lesions surrounded by vasogenic edema
Usually appear at the gray-white matter junction.
In hemispheres 80%, in cerebellum 5%
Hemorrhagic transformation (melanoma, thyroid, renal)
Common: lung, melanoma
Rare: prostate, head and neck, esophagus

Vasogenic edema:
A form of brain edema where the blood-brain barrier is compromised. The edema is extracellular.
Causes: tumors, infections
Radiological features:
- Preserved gray-white matter border
- No diffusion restriction
- Glove-finger-like spread
- Sulci efface, midline shift

Compare with: cytotoxic edema.



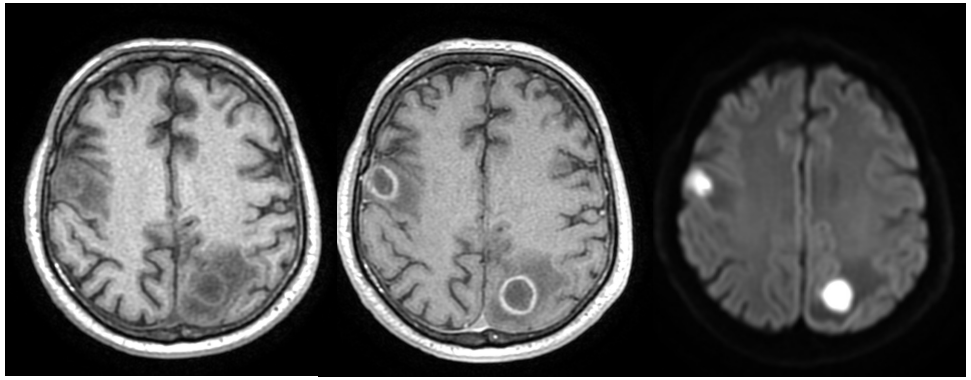
A: T2-weighted, B, D, E: post-contrast T1-weighted, C: diffusion-weighted MR images. F: post-contrast CT image. On the right side, in the frontal white matter, a cystic, ring-enhancing lesion, not restricting diffusion (see case report of brain abscess), is visible. Similar metastases are present on the left side, both frontally and cerebellarly. The metastases are surrounded by vasogenic edema.

Radiology case reports

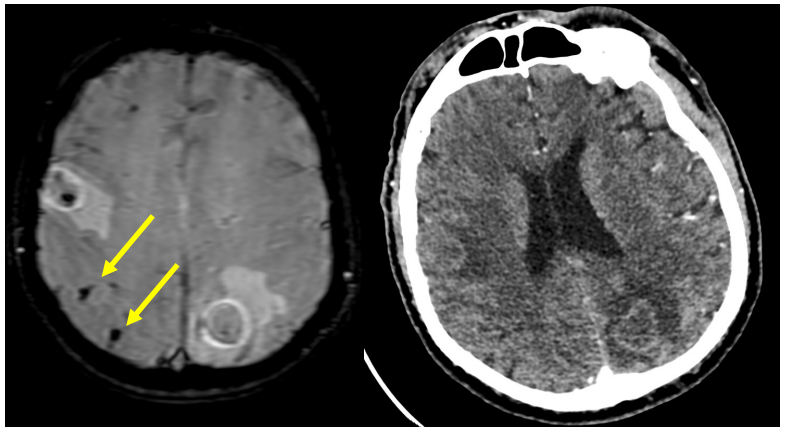
Anamnesis: 70-year-old male patient with polyarthritis, antiphospholipid syndrome, mitral valve prosthesis, and atrial fibrillation, who is anticoagulated. Admitted due to high INR levels. One month before admission, he experienced a gradual deterioration in speech. A day before the MRI examination, he developed numbness and weakness in the left upper extremity. In the morning, he experienced twitching on the left side of his face, which ceased after parenteral administration of benzodiazepine. *Neurological status:* Central facial paresis on the left side, left-sided hemiparesis rated 4/5.

Diagnosis: **Brain abscess**

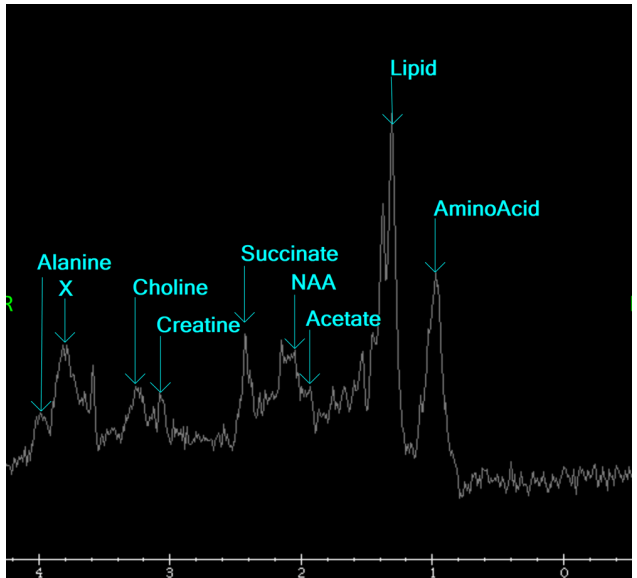
The transthoracic echocardiogram found vegetation on the mitral prosthetic valve.
>> **Endocarditis**



From left to right: native and post-contrast T1-weighted, and diffusion-weighted images. On the right side, in the frontal region, and on the left side in the precentral gyrus, and on the right side in the superior parietal lobule, there are ring-enhancing lesions surrounded by edema. These cystic lesions, can be distinguished from neoplasia by the diffusion restriction typical for abscesses.



On susceptibility-weighted images, the lesion in the left parietal region exhibits a double-rim sign (inner hypointense, outer hyperintense ring). The hypointense structures marked with arrows are most likely septic emboli. Abscesses can also be identified on contrast-enhanced CT scans performed before the MRI examination, although without MRI, they cannot be distinguished from neoplasms.

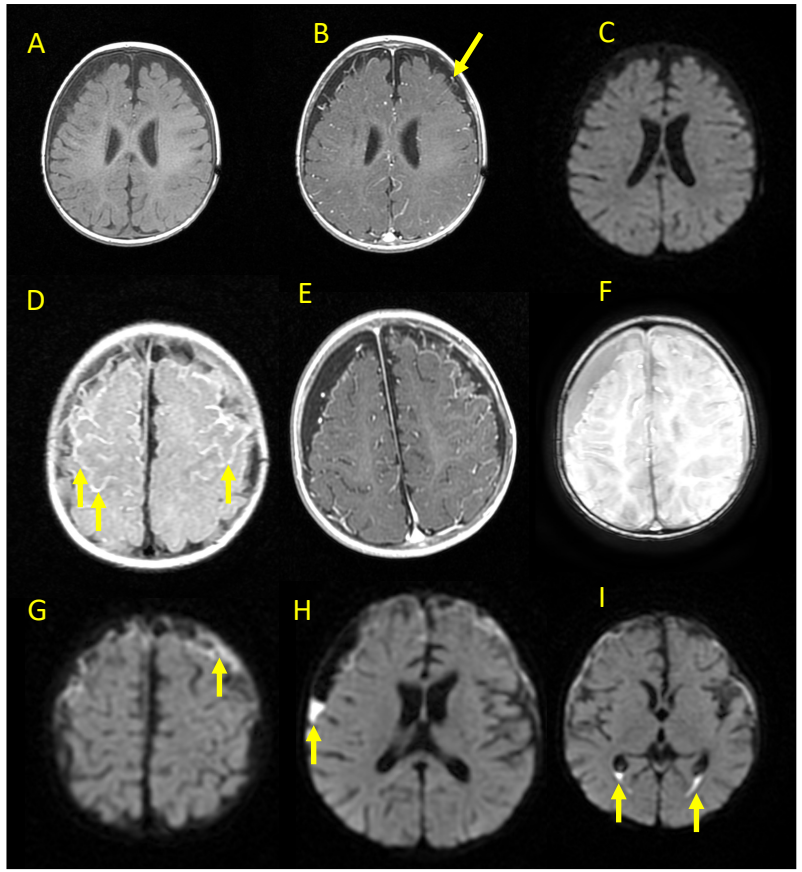


The single-voxel spectroscopy performed from the parietal lesion (TE: 35ms) identified multiple amino acid peaks and a high lipid peak, consistent with brain abscess.

Radiology case reports

Medical history: 6.5 months old boy. He has been febrile (38.7-38.9°C axillary) the week before his admission. During this time, his appetite has been good, and he has been drinking plenty of fluids. On the morning of hospital admission, he vomited twice. Over a few hours he become somnolent and anisocoria developed. MRI (1st row) and lumbar puncture was carried out. Purulent meningits was diagnosed and the pathogen was Neiseria meningitidis. With antibiotic therapy there was a transient improvement, but after 3 days he become somnolent again. MRI scan was repeated (2nd and 3rd row).

CSF: WBC: 6827 M/L RBC: 491 M/L Total protein: 2.63 g/L Cytology: predominant polymorphonuclear leukocytes, CSF glucose: 0.183 mmol/L, serum glucose: 4.39 mmol/L.
Microbiology: Neiseria meningitidis PCR: positive.



Non-enhanced (A), enhaced (B) and DWI (C) images at admission. Enlarged bilateral frontal subdural spaces, with prominent dilated bridging veins (arrows). No diffusion restriction was seen.
Contrast enhanced T1 weighted images (D, E) show intense arachnoidal enhancement (yellow arrows). Susceptibility weighted images (F) indicate no haemorrhage in the subdural space (no hypointensity). The diffusion weighted images show diffusion restriction in the subdural space and in the ventricles (arrows) due to the accumulated pus.

ESCMID guideline: Diagnosis and treatment of acute bacterial meningitis

Radiology case reports

Anamnesis: 23-year-old female patient. Three weeks prior to the imaging, she had upper respiratory tract infection and fever. She took antibiotics. A week later, she gradually developed severe headache (VAS: 10/10) that did not respond to painkillers. The pain was localized in the forehead and temple area. She vomited several times. She was sensitive to light. The following day, she had transient numbness on the left side of her body and face.

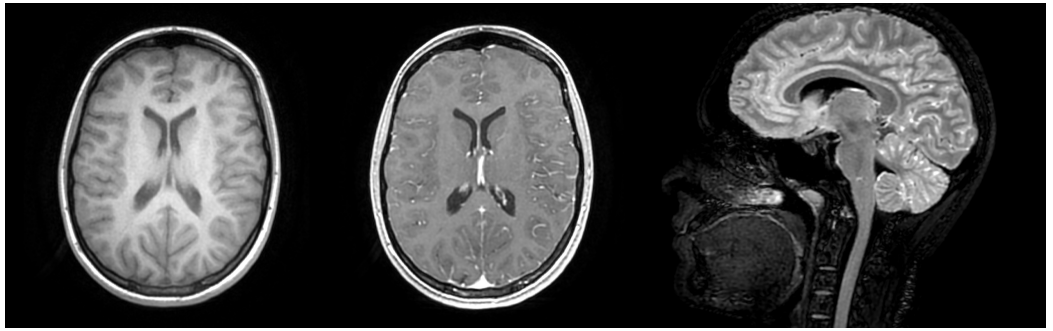
Status: Neck stiffness, positive meningeal signs.

EEG: Predominant slow wave series in the bifronto-temporal region on the left side, without clear epileptiform characteristics.

CSF: WBC: 492/µl (mononuclear), RBC: 4/µl, total protein: 2.4 mg/dl, Glucose: 3.06 mmol/L

Microbiological examination: Parvovirus ELISA IgM and IgG positive in serum. Parvovirus PCR positive in CSF.

Diagnosis: **Viral meningitis**



From left to right: Axial native, post-contrast T1-weighted, and sagittal post-contrast FLAIR images. The arachnoidal enchancement is visible, and accordingly, hyperintensity is observed in the sulci on the FLAIR images.

Imaging signs of meningitis:

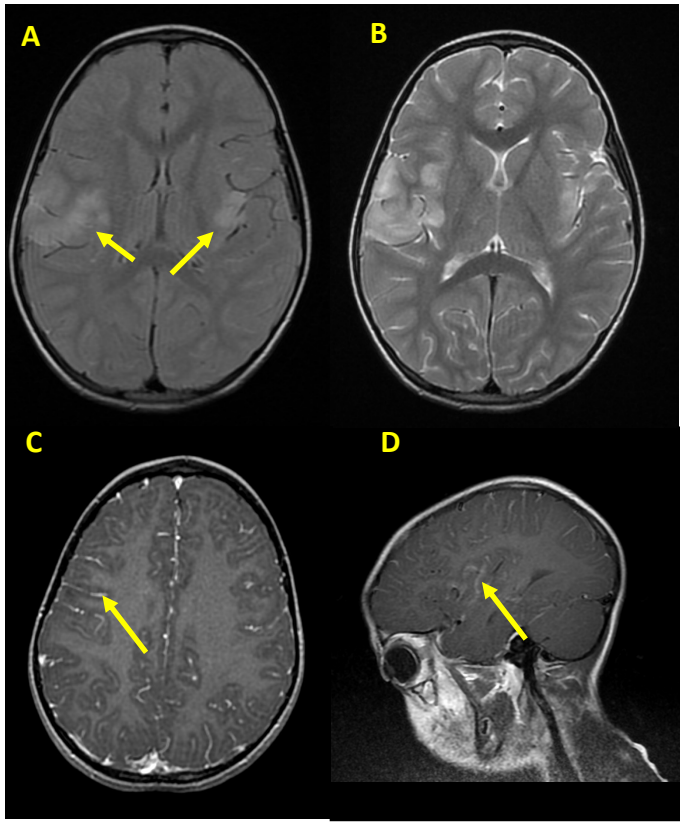
- CT is generally not informative.
- MR: leptomeningeal enhancement, contrast enhancement in sulci on FLAIR. It may be negative!

Check the surrounding sinuses, mastoid cells in purulent meningitis cases!
If there is also a lesion in the brain parenchyma, it is encephalitis!

Radiology case reports

Anamnesis: A 4.5-year-old girl is brought to the hospital by ambulance due to headache, vomiting, lethargy, fever, and suspicion of dehydration. She has been feverish for three days. She has also been vomiting for a day. She has no appetite.
Status: Neck stiffness. Otherwise non-remarkable.
Laboratory findings: White blood cell count (WBC): 6 G/l, C-reactive protein (CRP): 2.2 mg/l, procalcitonin (PCT): 0.07 ng/ml.
Cerebrospinal fluid (CSF): WBC: 25 M/l (75% lymphocytes), red blood cells (RBC): 15 M/l, total protein: 0.28 g/L. Glucose (Glu): 4.73 mmol/L (Serum Glu: 6.14 mmol/l).
CSF PCR: HSV-1.

Diagnózis: **Herpes simplex encephalitis/meningitis**



A: FLAIR, B: T2-weighted, C-D: post-contrast T1-weighted MRI images. Bilateral asymmetric hyperintensity is observed in the insular regions on FLAIR and T2-weighted images. Arachnoid enhancement is seen in the sulci.

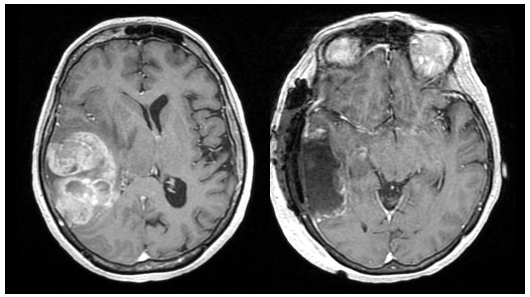
CSF analysis in meningitis

	Normal	Septic (bacterial)	Serosus (viral)
pressure	normal	increased	normal/increased
White blood cells	0-5/mm^3	100-60.000/mm^3	5-1000/mm^3
Polymorphs	0	>80%	<50%
Glucose	75% of the serum	<40% of the serum	normal

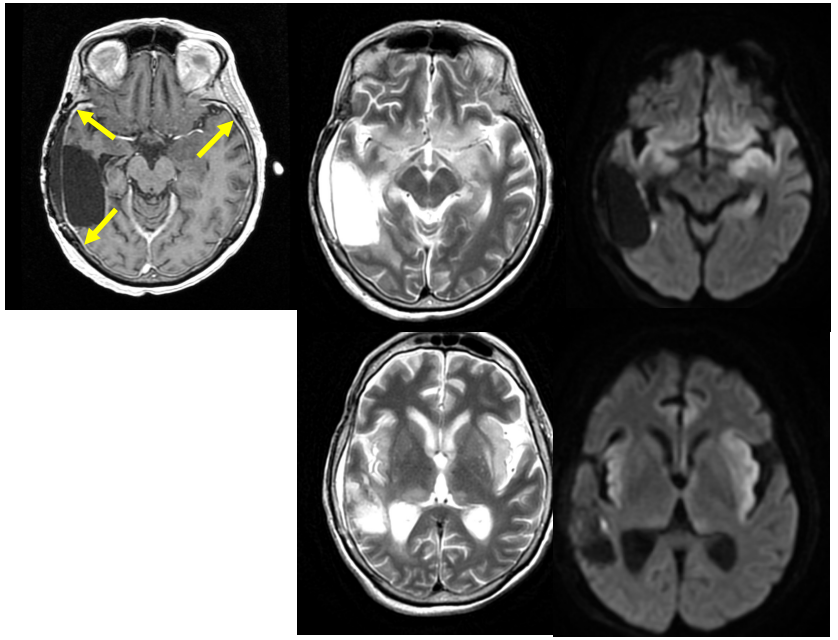
Radiology case reports

Anamnesis: 60-year-old female patient with a history of glioblastoma surgery, followed by a course of radiotherapy (29 fractions of 2Gy each) and concurrent temozolomide (100mg/day) between July and September. After surgery, she was ambulatory and self-sufficient. No residual tumor was visible on follow-up imaging. In September, she became difficult to arouse on one day, was disoriented, and had fever.
Status: Glasgow Coma Scale (GCS): 4-2-4. Neck stiffness. She withdraws limbs in response to painful stimuli. Babinski reflex positive on the left side.
EEG: Normal.
Laboratory findings: WBC: 0.39G/l, Lymphocytes: 0.04 G/l, Neutrophils: 0.27G/l, RBC: 3.08T/l, Platelets: 121G/l, CRP: 190mg/l, PCT: 4.0ng/ml
Lumbar puncture: WBC: 0M/l, RBC: 0M/l, Protein: 0.55g/l, Glucose: 6.36mmol/l CSF PCR: Positive for Herpes simplex I

Diagnosis: ***Herpes simplex encephalitis***
Although the immunosuppressive effect of temozolomide is generally less compared to other alkylating agents, it can occasionally cause immunosuppression and predispose individuals to opportunistic infections.
Note: There are now WBC in CSF because of the severe lyphopenia.



Pre- and postoperative post-contrast T1-weighted MR images of the right temporal lobe glioblastoma.



On the left-sided post-contrast T1-weighted image, there is no evidence of recurrent tumor in the surgical cavity; however, there is pronounced dural enhancement around the temporal lobes (indicated by arrows).
The T2 and diffusion-weighted images obtained at the level of the midbrain and thalamus demonstrate bilateral, asymmetric hyperintensity and diffusion restriction in the temporal poles, orbitofrontal cortex, and insula, which are characteristic of herpes simplex encephalitis.

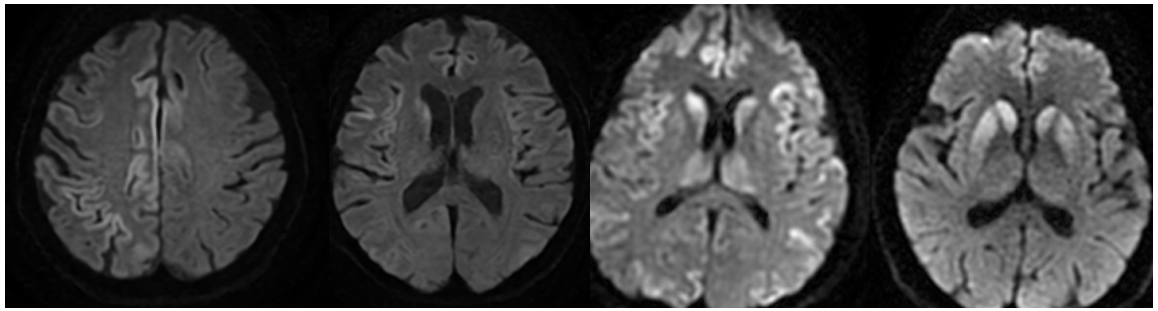
Herpes simplex encephalitis MR images:
- *Localization:* Bilateral, asymmetric involvement of the temporal lobes, insula, and limbic cortex. T
- 2-weighted imaging: Hyperintense signal.
- Diffusion-weighted imaging (DWI): Restricted diffusion.
- Enhancement: Variable (leptomeningeal, gyriform).

Radiológiai Klinika SZTE

Radiology case reports

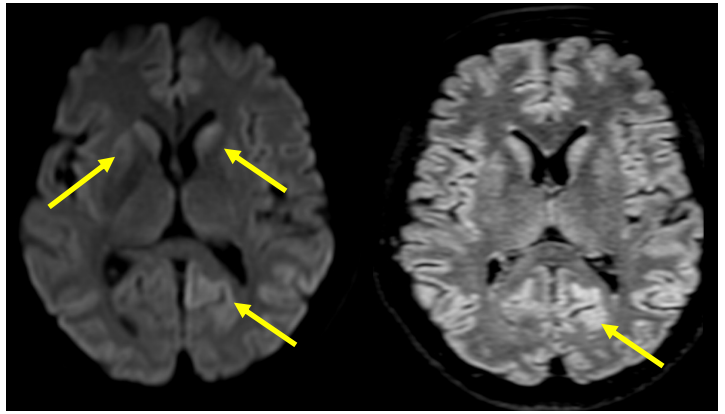
Anamnesis: 59-year-old male. Complaints began about 3-4 months earlier when he started experiencing visual impairment. Later, he began experiencing visual hallucinations. At the time of admission, the patient reports seeing only lights and outlines, but he can catch a ball thrown to him. His memory has significantly declined, and he cannot recall past events. His gait has become unsteady, requiring assistance. His wife occasionally observes muscle twitches.
Status: Severe visual impairment, but no visual field defects confirmed. Rigidity with left-sided dominance. Left-sided polykinetic Achilles reflex. Four-limb ataxia.
Liquor analysis: Protein: 0M/l, WBC: 4M/l, total protein: 0.47g/l, Glucose: 4.31mmol/l (Serum Glucose: 8.86mmol/l)
EEG: Generalized sharp wave sequences with a frequency of 2-3Hz, dominant on the left side.
14-3-3 protein test: Positive.

Diagnosis: **Creutzfeldt-Jakob disease**



Diffusion-weighted images from other CJD patients show prominent hyperintensity in the basal ganglia and cortex.

CDC's Diagnostic Criteria for Creutzfeldt-Jakob Disease 2018



Axial diffusion-weighted and FLAIR images reveal bilateral hyperintensity in the caudate nuclei, right-sided putamen, and occipital cortex.

Creutzfeldt-Jakob disease
Diagnostic criteria
- Rapidly progressive dementia At least two of the following:
  - Myoclonuses
  - Vizuális or cerebellaris symptoms
  - Pyramidalis/extrapyramidalis symptoms
  - Akinetic mutizm
- At least one of the following
  - EEG: periodic sharp waves
  - 14-3-3 protein in the CSF
  - DWI/FLAIR hyperintensity in the caudate/putamen or et least in two cortical region (temporal, parietal, occipital)

Radiology case reports

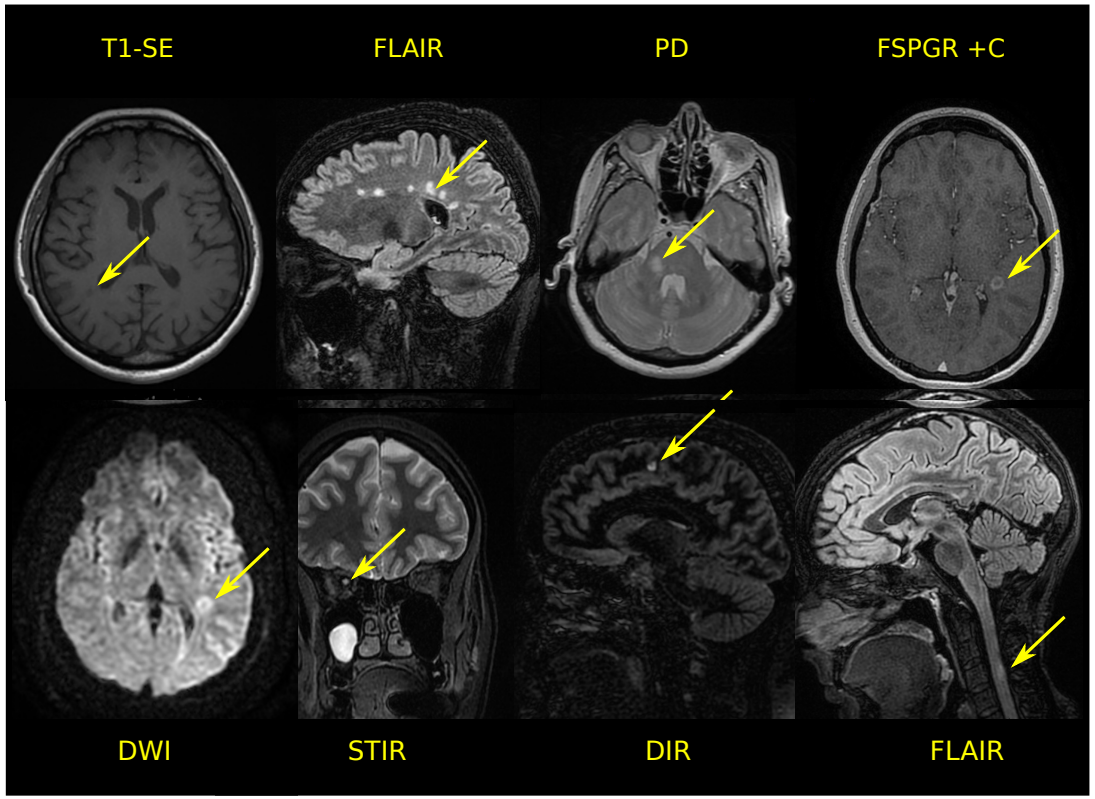
Anamnesis: 35-year-old Swedish female. Six months ago, she experienced blurred vision in her right eye, which gradually improved over weeks. One week before the MRI examination, she developed sensory disturbances in her right side of the body.
Status: Reduced contrast sensitivity on the right side. Positive Babinski reflex on the right side, with polyclonic Achilles reflex.

Diagnosis: **Multiple sclerosis (relapsus-remission, active)**

Spatial dissemination:
Presence of at least one lesion in at least two different regions among the following:
- Periventricular
- (Juxta)cortical
- Infratentorial
- Spinal

Temporal dissemination:
- Appearance of new T2 hyperintense lesions on two consecutive MRI scans
- Presence of both enhancing and non-enhancing lesions on a single MRI scan

Diagnosis of multiple sclerosis: 2017 revisions of the McDonald criteria



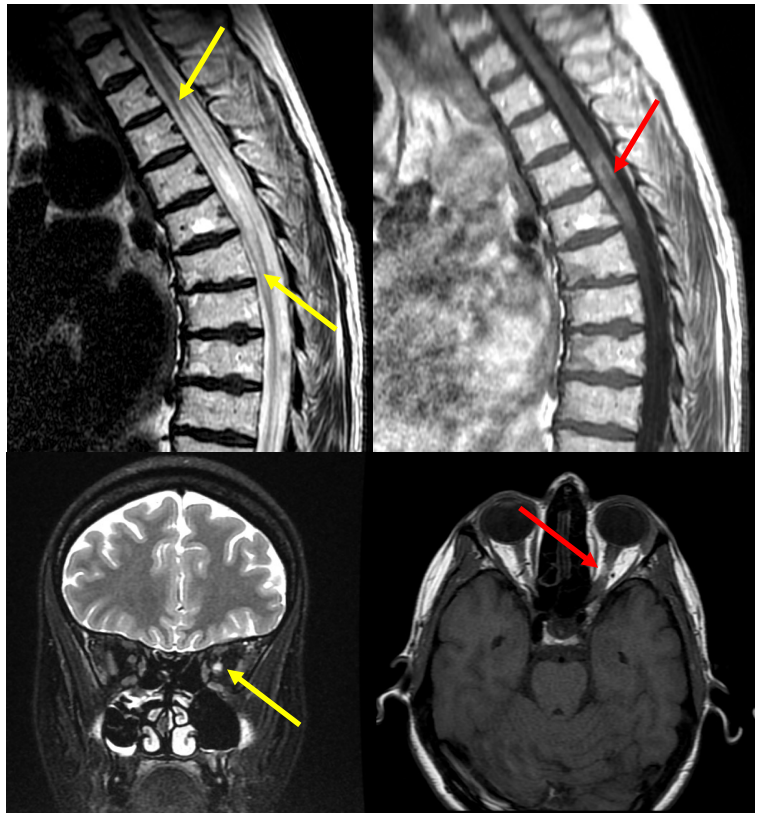
On sagittal FLAIR images, multiple periventricular T2 hyperintense lesions are visible (Dawson's fingers). On axial proton density (PD) images, an infratentorial lesion is observed. On coronal STIR images, increased signal intensity is noted in the right optic nerve. On double inversion recovery (DIR) images, a juxtacortical T2 hyperintense lesion is seen. On the lower right sagittal FLAIR image, a T2 hyperintense lesion is also visible in the CIII segment of the spinal cord. A "black-hole" is depicted on spin-echo T1-weighted sequences, indicating severe, irreversible tissue damage. On post-contrast T1-weighted images, a ring-enhancing lesion is observed. These lesions typically enhance over 2-3 weeks after formation. Diffusion restriction is also present, but it is not a definitive sign of lesion age.
Based on these findings, the radiological image suggests spatially and temporally disseminated demyelinating disease.

Radiology case reports

Anamnesis: She began to feel numbness symmetrically in his toes, and by the next day, it had spread up to his knees. Two days later, he felt numbness up to the level of his breasts. Her legs were weak. She felt as if electric current was running through her spine.
The thoracic MRI showed long thoracic myelon lesion.
Three month later she experienced blurred vision on the right.
Status: Paraparesis (4/5), hypaesthesia below Th.VI. level (predominantly loss of vibration and joint position), bilateral Babinski sign, ataxia in the lower extremety. Urinary retention.
CSF: No remarkable.
SSEP: Normal responses from the median nerve, but small amplitudes from the tibial nerve.
VEP: No response identifiable on the right.
Aquaporin-4 antibodies (CSF): positive

Diagnosis: **Neuromyelitis optica spectrum disorder**

Most common radiological presentation of NMOSD
- Optic neuritis (> half of the optic nerve length)
- Long transverse myelitis (> 3 vertebrae)
- Area postrema syndrome (dorsal medulla)



Upper row: T2 and postcontrast T1 weighted images from the thoracic spine. The yellow arrows indicate a long transverse myelitis. In the same lesion there is a ring enhancement (red arrow).
Lower row: coronal T2 and axial postcontrast T1 weighted images. The left optic nerve is hyperintense (yellow arrow) and shows contrast enhancement (red arrow).

Update on the diagnosis and treatment of NMOSD (NEMOS)

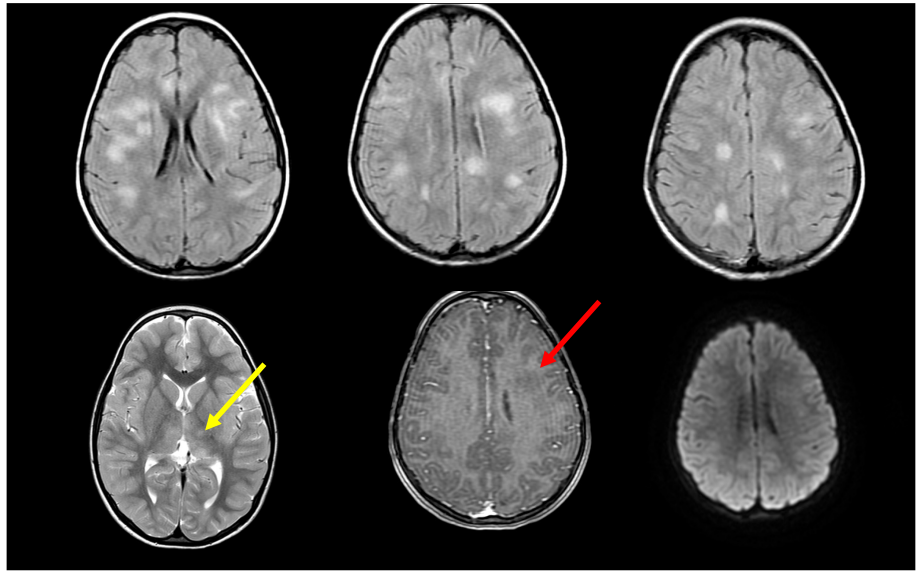
Radiology case reports

Anamnesis: 5 years old boy. After a week-long upper respiratory tract infection, he developed headache,he become slightly confused and somnolent.
Status: Somnolent, mild ataxia with all four extremities, he is not able to perform tandem-walk.
Lab: CRP 6.8mg/l, PCT: neg. WBC: 14.3G/l
CSF: WBC: 15 M/l (75% lympocytes, RBC: 5 M/l)
Microbiliogical examinations: neg.

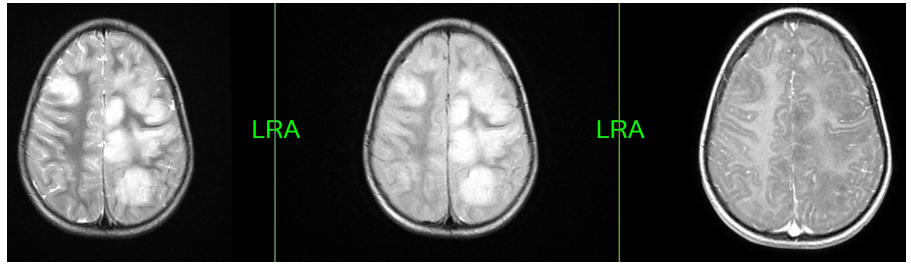
Diagnosis: **Acute disseminated encephalomyelitis**

ADEM diagnostic criteria

- Polyfocal CNS event
- Encephalopathy
- No new MR or clinical findings in the 3 months or more after onset.
- Abnormal brain MRI:
  - Diffuse, poorly demarcated large lesions in the WM
  - T1 hypointense lesions are rare
  - Deep grey matter lesions might be present



Upper row: Multplex, large, poorly demarcated white matter lesions on the FLAIR images.
Lower row: The yellow arrow indicate T2 hyperintense lesions in the bilateral thalami. The red arraow indicate slight hypointensity on the postcontrast T1 weighted image according to the large T2 hyperintensity (see image above). Note, there was no abnormal contrast enhancement. There is now restricted diffusion on the lower right image.
The white matter lesions completely resolved on the control MR one month after the first.



Images from another ADEM patients. Large, poorly demarcated, non-enhancing white matter lesions.

[IPMSG criteria for pediatric MS and immune mediated CNS demyelinating disorders]

Radiology case reports

Anamnesis: 28 years old male. He was admitted urgently for evaluation of memory impairment, left hemiparesis. According to the patient's mother, her colleagues found her behaviour odd following a prolonged upper respiratory tract infection. He was working three shifts abroad; hence any abnormal behaviour was attributed to fatigue.

Status: Left hemiparesis (4/5), left Babinski reflex, ataxic left upper extremity. Short-term memory deficit.

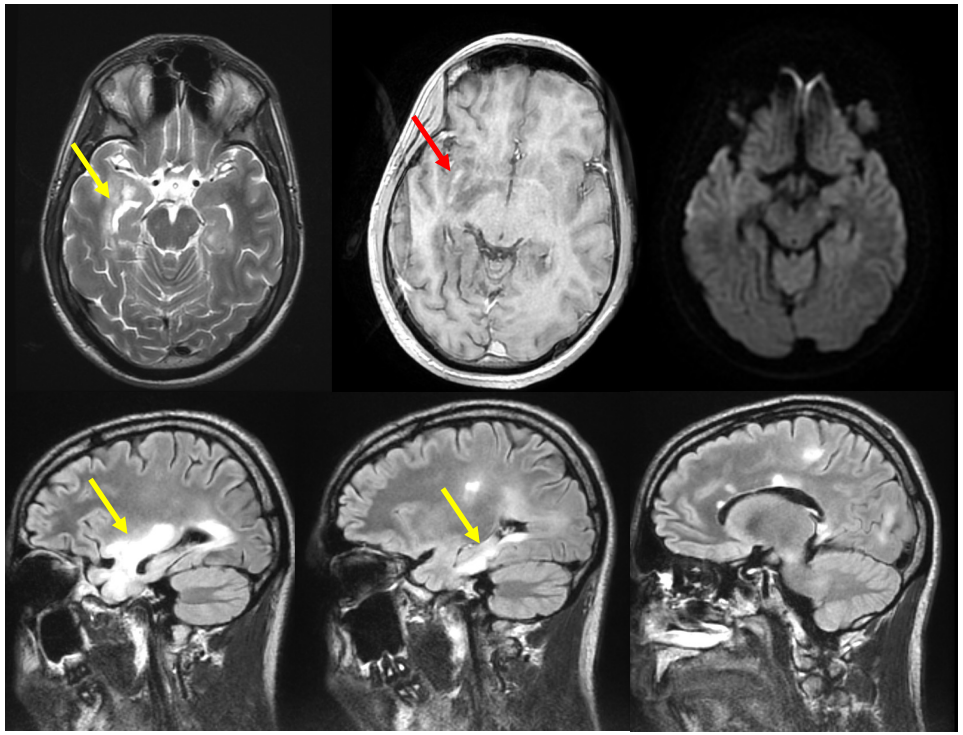
EEG: Bilateral temporal slow waves.

CSF: WBC: 17M/l, RBC: 1M/l, protein: 0.3g/l, Glu: 3.11mmol/l 90% lymphocytes.

FDG-PET: neg.

Autoantibodies (CSF): anti-NMDR: pos.

Diagnosis: **Autoimmune encephalitis**



Upper row: T2, postcontrast T1 and diffusion weighted images.
Lower row: Sagittal FLAIR images
Note the right dominant bilateral mesio-temporal hyperintensities, with no diffusion restriction and only a slight contrast enhancement on the right (red arrow).

Autoimmune encephalitis

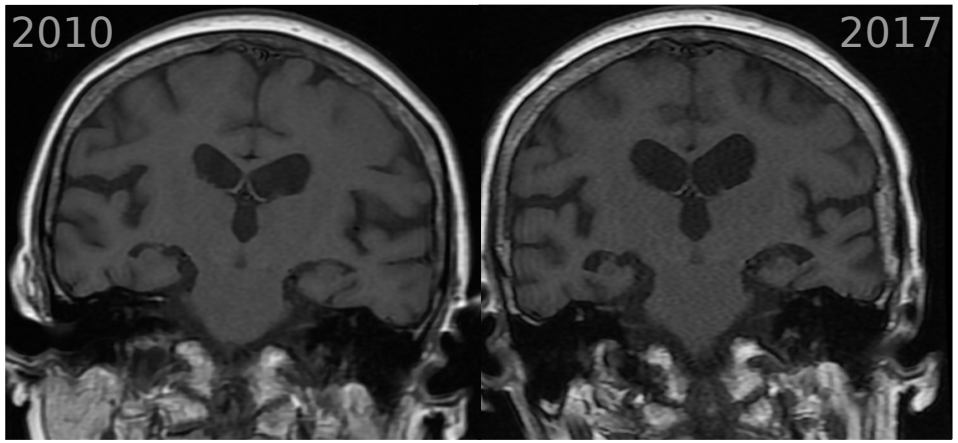
- Subacute onset (rapid progression of less than 3 months) of working memory deficits (short-term memory loss), altered mental status, or psychiatric symptoms
- At least one of the following:
  - New focal CNS findings
  - Seizures not explained by a previously known seizure disorder
  - CSF pleocytosis (white blood cell count of more than five cells per mm3)
  - MRI features suggestive of encephalitis
- Reasonable exclusion of alternative causes

A clinical approach to diagnosis of autoimmune encephalitis

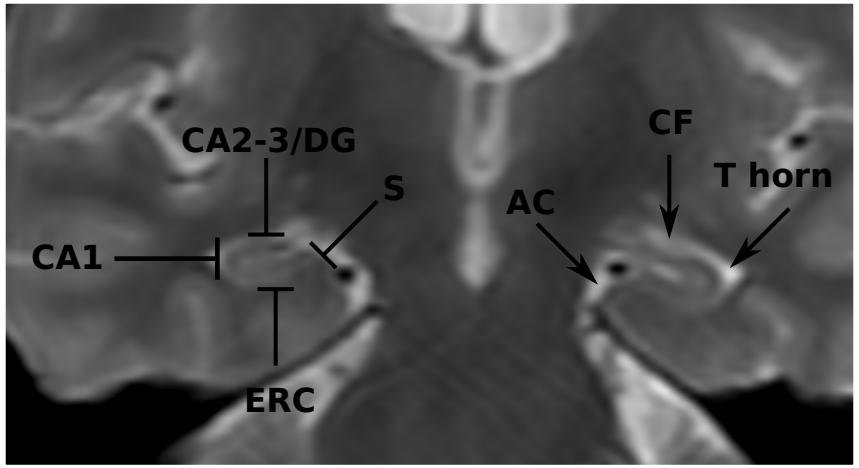
Radiology case reports

Anamnesis: 82-year-old male. Investigation started due to memory problems. According to the family, his memory problems have been worsening for 5-6 years. He does not recognize acquaintances, often asks for clarification, and occasionally has difficulty finding words. Naming objects is also problematic for him. His orientation is not as good as before. He is experiencing more conflicts with those around him. Irritability and verbal aggressiveness occur occasionally. Sleep is normal. He is active and exercises. MRI examination was performed 10 years ago due to headaches.

Diagnosis: **Alzheimer's disease**



On the coronal T1-weighted images, significant hippocampal atrophy over 7 years is clearly visible.



The MR anatomy of the hippocampus and surrounding structures on high-resolution T2-weighted image. AC: ambient cistern, CF: choroid fissure, Thorn: temporal horn, DG: dentate gyrus, S: subiculum, ERC: entorhinal cortex

Scoring of medial temporal lobe atrophy:

0: No significant CSF space around the hippocampus
1: Mild dilatation of the choroid fissure
2: Moderate dilatation of the choroid fissure, mild dilatation of the temporal horn, and mild decrease in the height of the hippocampus
3: Significant dilatation of the choroid fissure, moderate dilatation of the temporal horn, and moderate decrease in the height of the hippocampus
4: Significant dilatation of the choroid fissure and temporal horn, significant decrease in the height of the hippocampus, and loss of its internal structure.

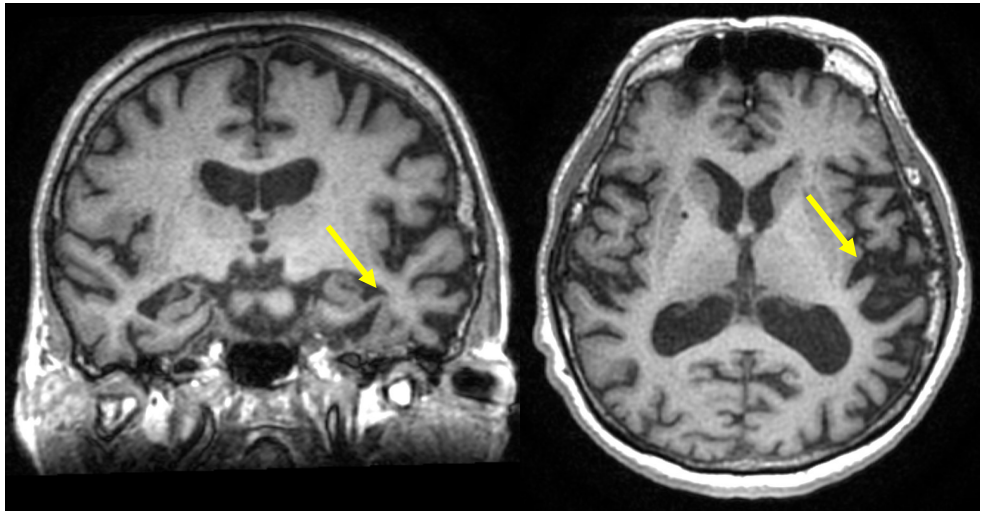
Radiology case reports

Anamnesis: A 69-year-old male patient. Six years ago, following a knee surgery, his family noticed a slowing down of his speech, decreased verbal output, and occasional confusion. After the surgery, his speech became more hesitant, and he had difficulty expressing himself, although he remained self-sufficient. Later on, significant deterioration in his speech ability and spatial orientation was observed. Five years later, substantial cognitive decline was noted, with the most prominent symptom being his speech impairment, particularly affecting speech comprehension. At the time of the MRI examination, he was unable to speak at all.
Status: Sensomotor aphasia, hypokinesis.

Diagnosis: **Primary progressive aphasia**

Primary progressive aphasia
Diagnostic criteria:
- The most significant difficulty is related to language use. This impairs daily functioning.
- At the onset of symptoms, aphasia should be the most significant deficit.
- Other diseases causing the symptoms must be ruled out.

Subtypes: Agrammatic, semantic, logopenic forms.
Imaging: Predominantly left-sided insular (anterior or posterior) or posterior perisylvian and parietal atrophy (MRI), hypoperfusion, hypometabolism (SPECT/PET).



On coronal and axial native T1-weighted images, left temporal lobe atrophy is observed. Compared to the opposite side, the areas marked with arrows show widened sulci and narrower gyri.

Classification of primary progressive aphasia and its variants. Gorno-Tempini et al. Neurology, 2011

Radiology case reports

Neuroradiology

Anamnesis: 72-year-old male. Over the months preceding the examination, his memory gradually deteriorated, and he developed gait disturbances, unable to control his urine.

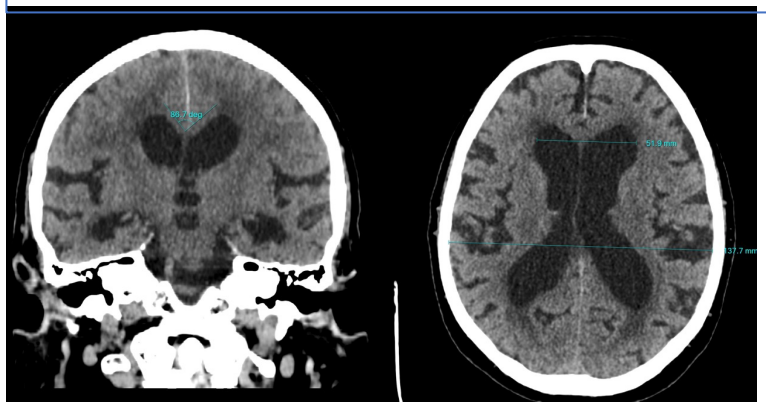
Status: Broad-based gait, able to take only a few steps without a walker. Mini-Mental State Examination (MMS) score: 24, urinary incontinence.

Diagnosis: **Normal pressure hydrocephalus**

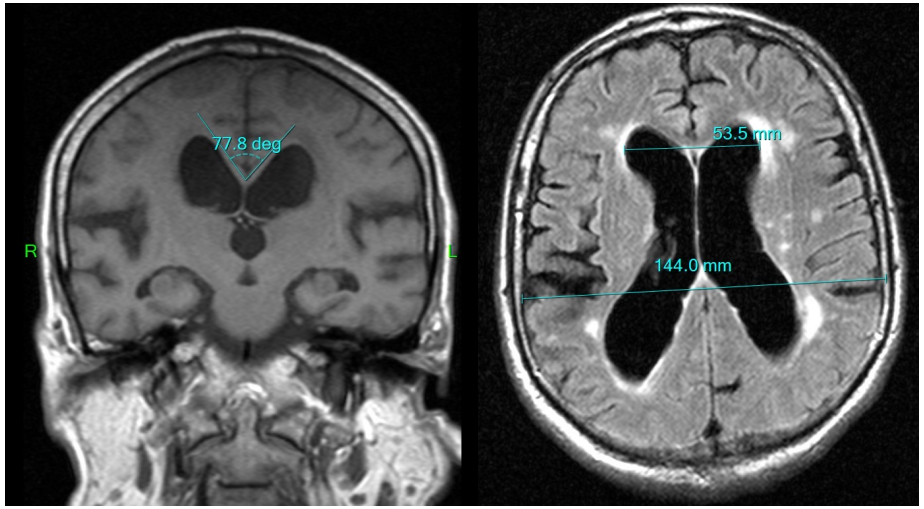
Normal Pressure Hydrocephalus

Diagnostic criteria: Hakim's triad:

- Gait disturbance,
- cognitive impairment,
- urinary incontinence
- CSF pressure: <200 mm H2O
- Disproportionately enlarged subarachnoid spaces (DESH)
- Improvement with spinal tap test.



A CT scan of another patient with normal pressure hydrocephalus shows similar abnormalities.



Coronal T1-weighted and axial FLAIR images. The callosal angle is less than 90 degrees, and the Evans index is greater than 0.3. The sulci are narrow on the convexity, while the basal brain regions show widened cerebrospinal fluid spaces (DESH). Around the frontal and occipital horns of the lateral ventricles, there is a cap-like "CSF percolation" visible.

Normal pressure hydrocephalus:

Radiological findings

- Evans index: The ratio of the largest diameter of the frontal horns of the lateral ventricles to the largest internal diameter of the skull (>0.3).
- DESH (Disproportionately Enlarged Subarachnoid Spaces): Narrow subarachnoid spaces on the convexity and widened spaces in the basal brain regions.
- Callosal angle: The angle formed by the corpus callosum, measured to be greater than 90 degrees.
- T2 hyperintensity around the ventricles (CSF percolation).

Guidelines for Management of Idiopathic Normal Pressure Hydrocephalus

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Radiology case reports

Neuroradiology

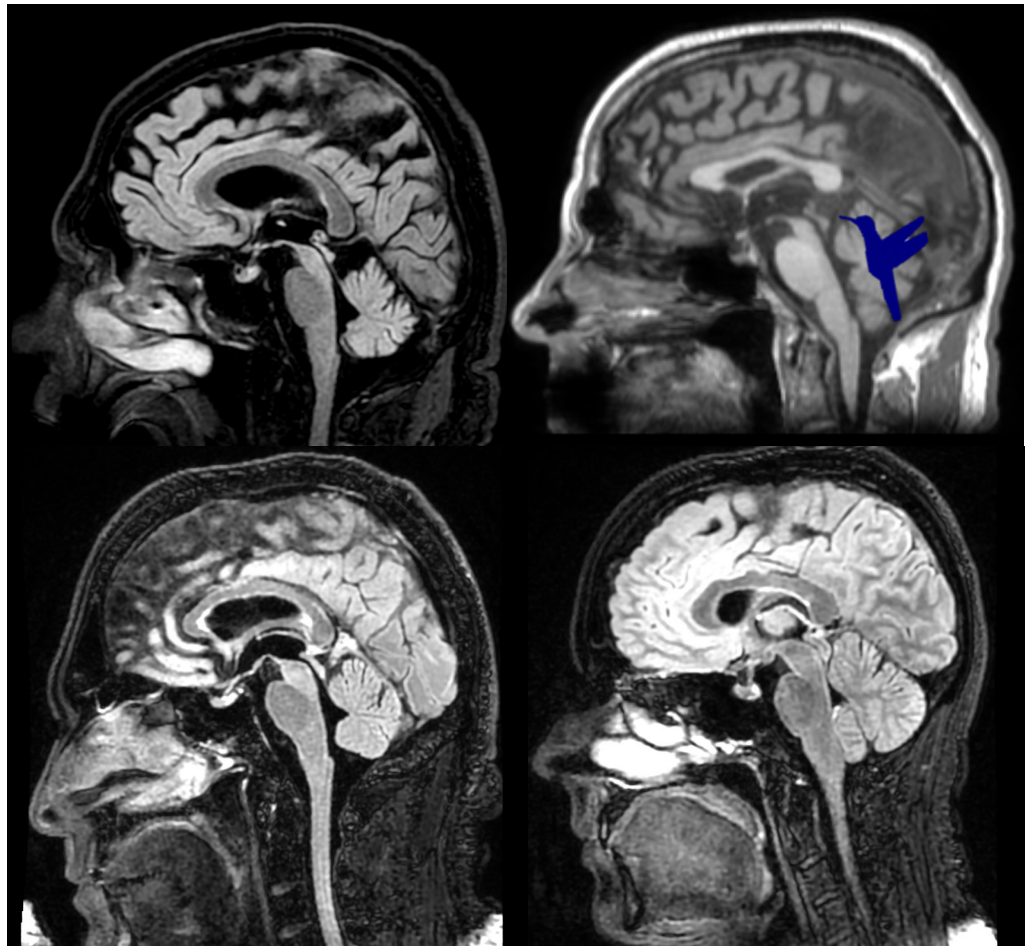
Anamnesis: A 62-year-old male. Over the past two years prior to the examination, his gait has slowed down, becoming shuffling. His balance feels uncertain. He has fallen several times. His thinking has become slower.
Status: His gait is shuffling, with small steps. There is a lack of synkinesis in the arms, and the frequency and amplitude of alternating movements are reduced. He has a disorder of vertical gaze.

Diagnosis: **Progressive supranuclear palsy**

Progressive supranuclear palsy
- 40 years old
- Vertical supranuclear gaze palsy
- Recurrent, unprovoked falls (<3 years)
- Progressive gait disturbance (freezing) (<3 years)
- Language or frontal cognitive impairment
- Levodopa resistance

PSP imaging signs:
- Mesencephalic atrophy, hypometabolism
- Tau-PET

Clinical Diagnosis of Progressive Supranuclear Palsy: The MDS criteria



Sagittal FLAIR and T1-weighted (upper right) images. The upper left image is from the 62-year-old male described in the medical history. The upper right image is from another patient suffering from PSP. The lower two images are from two healthy adults. The left side corresponds to the conditions of the two patients mentioned above, while the lower right image is from a 20-year-old individual.
On the upper two images, mesencephalic atrophy is clearly visible. The midbrain tectum is flattened. Hummingbird sign.

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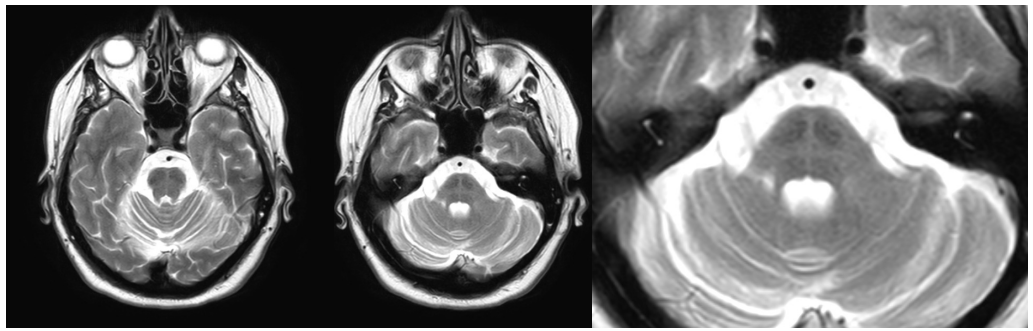
Radiology case reports

Anamnesis: 55 years old male. His symptoms began about 2 years earlier. His gait has slowed down, become unsteady, and he has developed a balance disorder. He has not experienced any falls. In recent months, his speech has become slurred. He experiences dizziness, swallowing difficulties, tremors, clumsiness in the upper limbs. He has got urinary urgency. He also feels dizziness and sense of fainting upon standing up. L-dopa treatment was not successful.
Status: Mild rigor on the upper extremities. Babinski reflex on both sides. Slight ataxia in the lower extremities and gait ataxia. Dysarthric speech. Urinary incontinence. Orthostatic hypotonia (25/10Hgmm).

Diagnosis: **Multi system atrophy**

MDS criteria for the Diagnosis of Multi System Atrophy



Axial T2 weighted images. Hot-cross-bun sign.

Multi system atrophy

- Sporadic >30 years of age
- Autonomic dysfunction (at least one):
  - Voiding difficulties (>100ml residual volume)
  - Urinary urge or incontinence
  - Orthostatic hypotension (>20/10Hgmm)
- Key neurological symptoms:
  - Poorly L-dopa responsive parkinsonism
  - Cerebellar syndrom (ataxia, dysarthria, oculomotor features)
- Supportive neurological symptoms (at least one):
  - Rapid progression within 3 years
  - Postural instability
  - Craniocervical dystonia
  - Speach impairement
  - Babinski sign
  - Jerky myoclonic or kinetic tremor
  - Postural deformities
  - Inspiratory sighs
  - Cold, discolored hands and feet
  - Erectile dysfunction
  - Pathologic laughter or crying

MRI markers of MSA

- Atrophy:
  - Putamen (hypointensity on SWI)
  - Pons
  - Cerebellum
- Hot cross bun sign
- Increased diffusivity of the putamen

Radiology case reports

Medical history: 45-year-old female patient. Examination is being conducted due to left-sided limb weakness. Symptoms have been present for a few weeks, starting relatively suddenly. Previously, she was managed for migraine with aura. Since the onset of limb weakness, the intensity and frequency of headaches have decreased. Turns out that several CADASIL patients are present in the family.
Status: Left-sided 4/5 hemiparesis, left-sided Babinski reflex, left-sided hemihypoesthesia, neglect. Psychological assessment: Mood disorder, irritability, memory impairment (MMSE: 23/30), executive dysfunction.

Cerebral autosom dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL)

Diagnostic features

Clinical criteria

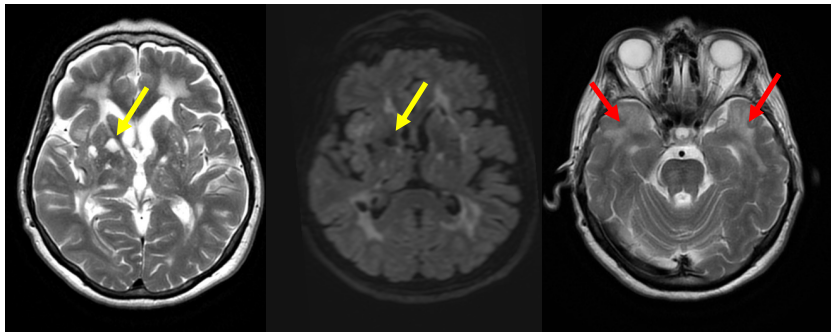
- <55 years of age at onset
- ≥ 2 from the followings:
  - Subcorticalis demencia, pyramidal signs, pseudobulber palsy
  - Stroke-like episodes with focal neurological symptoms fokális
  - Mood disorder
  - Migraine
- Autosom dominant inheritance
- White matter lesions in the temporal poles
- Other leukodystrophies can be excluded.

Genetical criteria:

- NOTCH3 mutation

Pathologic criteria:

- Granular osmiophyl material with electornmicroscopy
- NOTCH3 immunhistochemistry



Left to right: T2-weighted, FLAIR, and T2-weighted images. Multiple lacunar infarcts are visible in the area of the basal ganglia, which appear hyperintense on the T2-weighted images and hypointense on the FLAIR images (yellow arrows). Confluent T2 hyperintensities are visible in the subcortical white matter. The involvement of bilateral temporal poles is very characteristic of the disease (red arrows).

Diagnosis: **CADASIL**

New diagnostic criteria for CADASIL in Japan

Author's declaration

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