

DIAGNOSIS & MANAGEMENT OF ACUTE NEUROLOGICAL DISORDERS IN PREGNANCY AND POSTPARTUM

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Acute neurological symptoms in pregnant and post-partum women could be caused by

1. Exacerbation of a pre-existing neurological condition.

2. The initial presentation of a non-pregnancy-related problem or

3. A new acute-onset neurological problem that is either unique to or occurs with increased frequency during or just after pregnancy

- **Pregnant and postpartum patients with headache and neurological symptoms are often diagnosed with pre-eclampsia;**
- **however, a range of other causes must also be considered.**

THESE INCLUDE:

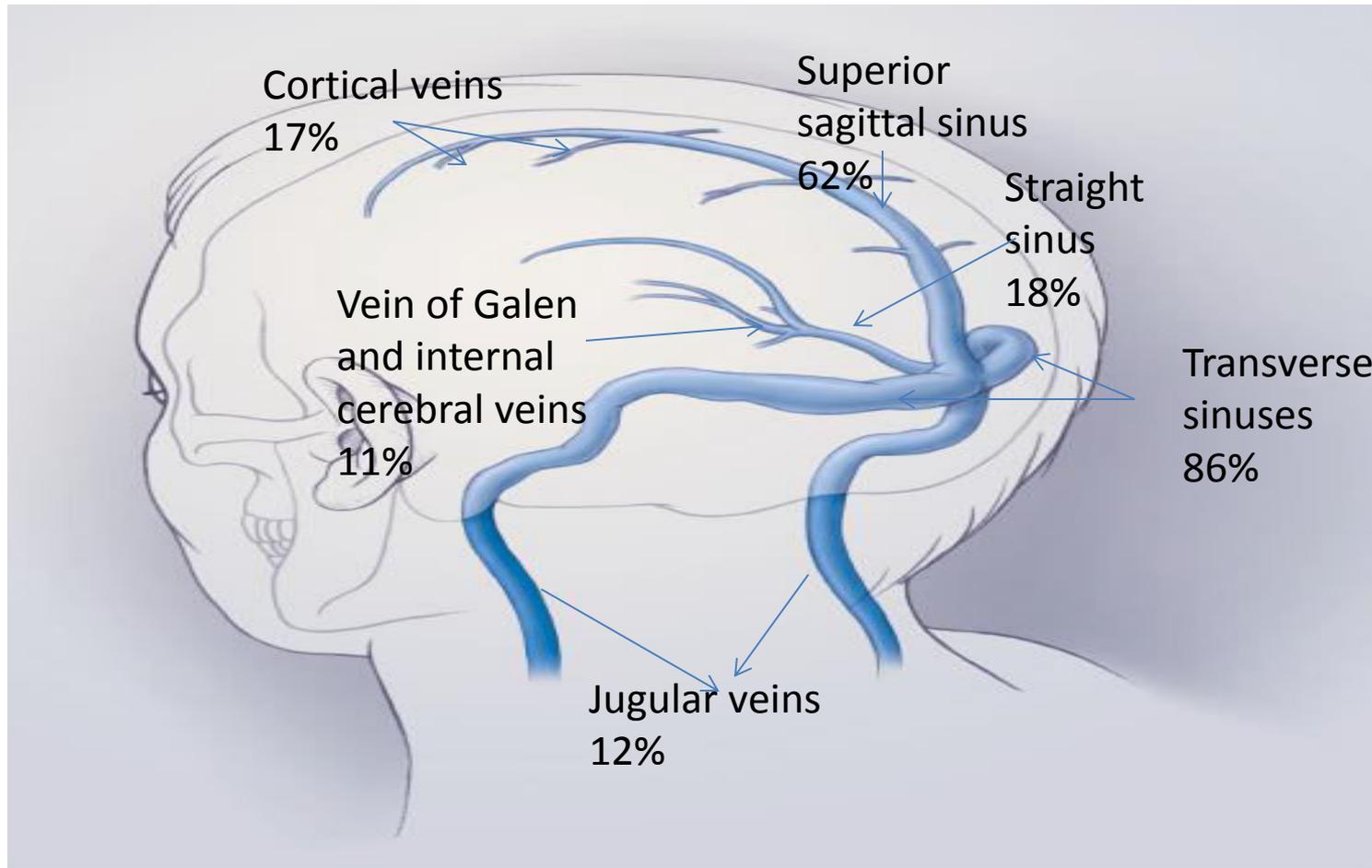
- 1. Cerebral venous sinus thrombosis**
- 2. Reversible cerebral vasoconstriction syndrome**
- 3. Posterior reversible encephalopathy syndrome**

	PRES	RCVS	CVT	Eclampsia
Mode of onset	Rapid (hours), usually post partum	Abrupt, usually post partum	Third trimester or post-partum, symptoms often progress over days	Antepartum, intrapartum, or post partum (10–50%)
Key findings	symptoms (eg, stupor, visual loss, and visual hallucinations) usually accompany seizures; headache dull and throbbing, not thunderclap	Thunderclap headache, multiple episodes; seizures occur but are less common than in PRES; transient focal deficits (could become permanent in cases with intracerebral haemorrhage or infarction)	Headache nearly universal at onset, generally progressive and diffuse, thunderclap in small minority; seizures occur in roughly 40% of patients; focal signs might develop later	Seizures, frequent visual symptoms, abdominal pain, hyper-reflexia, hypertension, and proteinuria

	PRES	RCVS	CVT	Eclampsia
Evolution over time	If blood pressure is controlled, symptoms resolve within days to weeks	Dynamic process over time; generally, headaches common during first week, intracerebral haemorrhage during second week, and ischaemic complications during third week	Evolves over several days, non-arterial territorial infarcts and haemorrhages might develop	Can evolve (from pre-eclampsia) gradually or abruptly
CSF findings	Usually normal, might have slightly raised protein	Often normal (unless complicated by subarachnoid haemorrhage), but 50% of patients have slight pleocytosis and protein increases	Opening pressure raised in about 80% of patients; roughly 35–50% will have slightly raised protein or cell counts	Usually normal unless complicated by haemorrhage

	PRES	RCVS	CVT	Eclampsia
Imaging aspects	<p>CT positive in about 50% of patients; MRI shows prominent T2-weighted and FLAIR abnormalities nearly always in parieto-occipital lobes, but can involve other brain regions; intracerebral haemorrhage in about 15% of patient</p>	<p>CT usually normal (if no subarachnoid haemorrhage); 20% show localised convexal subarachnoid haemorrhage on MRI; CT angiogram and magnetic resonance angiogram usually show typical string-of-beads constriction of cerebral arteries; digital subtraction angiogram is more sensitive; might have associated cervical arterial dissection; initial arteriogram might be negative</p>	<p>CT often negative; MRI might show non-arterial territorial infarcts; haemorrhage common; MRV shows intraluminal clot flow voids; although MRV is preferred, CT venogram is also sensitive</p>	<p>Same as for PRES, some patients have coincident acute ischaemic stroke or intracerebral haemorrhage</p>

Cerebral venous sinus thrombosis



- About 75 percent of the adult patients are women.
- More than 75% of cases of CVT are post partum.
- The frequency of peripartum and postpartum sinus thrombosis is about 12 cases per 100,000 deliveries.

- occlusion of the cerebral veins, can cause localized edema of the brain and venous infarction.
- Two different kinds of cerebral edema can develop.
- The first, cytotoxic edema, is caused by ischemia, which damages the energy-dependent cellular membrane pumps, leading to intracellular swelling. The second type, vasogenic edema, is caused by a disruption in the blood–brain barrier and leakage of blood plasma into the interstitial space.
- Vasogenic edema is reversible if the underlying condition is treated successfully

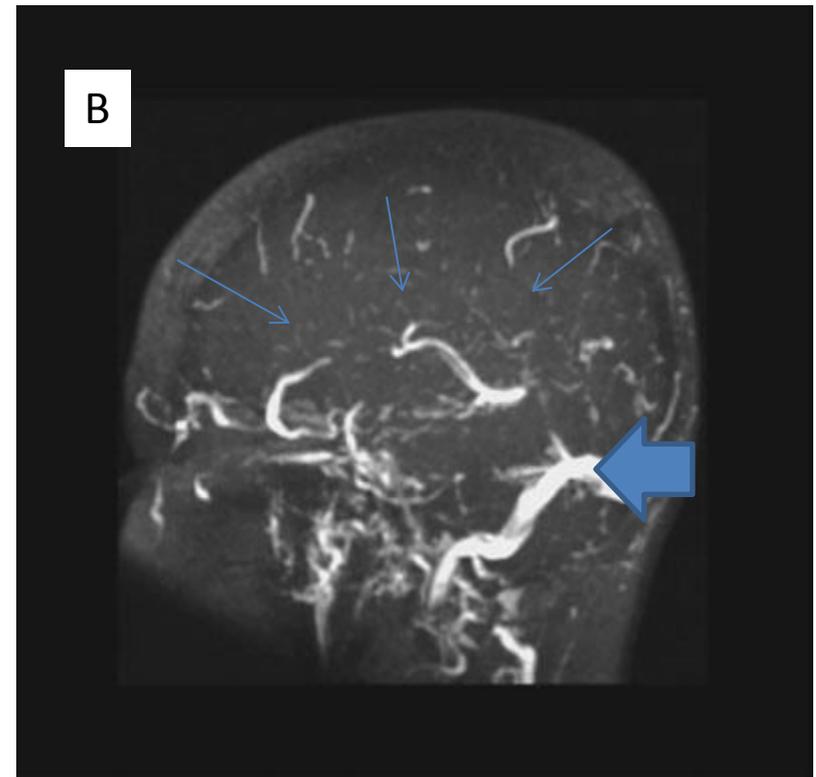
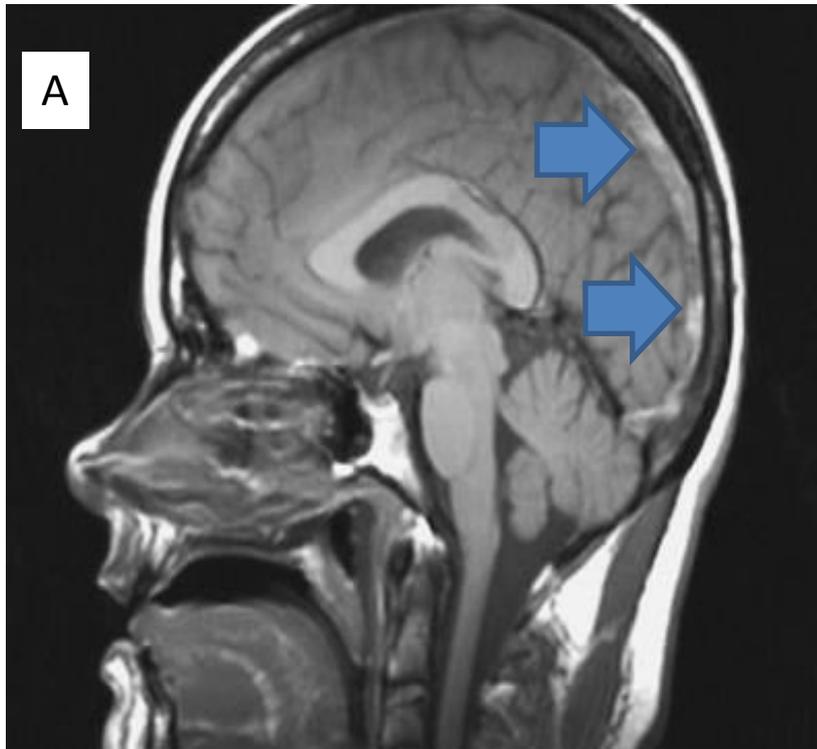
- the development of intracranial hypertension as the result of occlusion of the major venous sinuses.
- Normally, the cerebrospinal fluid is transported from the cerebral ventricles through the subarachnoid spaces at the base and surface of the brain to the arachnoid villi, where it is absorbed and drained into the superior sagittal sinus.
- Thrombosis of the sinuses leads to increased venous pressure, impaired absorption of cerebrospinal fluid, and consequently, increased intracranial pressure.

- Risk factors include
- caesarean section, dehydration, traumatic delivery, anaemia, raised homocysteine concentrations, and low CSF pressure due to dural puncture from a neuraxial anaesthesia.

- The clinical presentation is highly variable
- The most frequent but least specific symptom of sinus thrombosis is severe headache present in more than 90 percent of patients
- Usually increases gradually over a couple of days but can also start in a split second
- Seizures occur in about 40 percent of patients, Seizures are limited and focal in 50 percent of these patients but may generalize to a life-threatening status epilepticus
- .

- **Cortical lesions on both sides of the superior sagittal sinus:**
unilateral hemispheric symptoms such as hemiparesis or aphasia, followed within days by symptoms from the other hemisphere
- **Thrombosis of the deep venous system — the straight sinus and its branches:**
causes centrally located, often bilateral thalamic lesions, with behavioral symptoms such as delirium, amnesia, and mutism
- **Infectious cavernous sinus thrombosis:** headache, fever, and eye symptoms such as periorbital edema, proptosis, chemosis, and paralysis of eye movements due to involvement of the oculomotor, abducent, or trochlear nerves.
- large unilateral infarcts or hemorrhages compress the diencephalon and brain stem, patients may become comatose

- Patients with isolated intracranial hypertension have headache but no other neurologic symptoms, with the exception of diplopia due to involvement of the sixth nerve
- The most sensitive examination technique is MRI in combination with magnetic resonance venography
- T1-weighted and T2 -weighted MRI will show a hyperintense signal from the thrombosed sinuses
- The combination of an abnormal signal in a sinus and a corresponding absence of flow on magnetic resonance venography confirms the diagnosis of thrombosis.
- If the diagnosis is still uncertain after MRI or CT venography has been performed cerebral angiography may be indicated.



In Panel A, a T1-weighted MRI scan obtained with the spin–echo technique provides a sagittal view of a hyperintense signal in the thrombosed superior sagittal sinus (arrows).

In Panel B, a magnetic resonance venogram obtained without the administration of contrast material reveals the absence of a signal in the superior sagittal sinus (upper arrows) and a normal flow signal in the transverse and sigmoid sinuses (lower arrow) as well as in a number of veins

- The combination of acutely increased intracranial pressure and large venous infarcts is dangerous, and patients may die within hours from cerebral herniation.
- Impaired consciousness and cerebral hemorrhage are associated with a poor outcome
- The priority of treatment in the acute phase is to stabilize the patient's condition and to prevent or reverse cerebral herniation.
- This may require the administration of intravenous mannitol, surgical removal of the hemorrhagic infarct, or decompressive hemicraniectomy.

- **anticoagulation**
- The most obvious treatment option is anticoagulation with heparin to arrest the thrombotic process
- Most neurologists now start treatment with heparin as soon as the diagnosis is confirmed, even in the presence of hemorrhagic infarcts.
- In patients who have symptoms of chronic intracranial hypertension only, the first priority is to rule out a space-occupying process and to investigate whether sinus thrombosis is indeed the cause.

- If there are no contraindications, such as large infarcts or hemorrhages, a lumbar puncture is then performed to measure the cerebrospinal fluid pressure.
- This is also the start of treatment, the objective of which is to lower the intracranial pressure, to relieve headache, and to reduce papilledema.
- Oral acetazolamide (500 to 1000 mg daily) may reduce the intracranial pressure. Often, if effective and tolerated, this agent must be continued for weeks to months, as demonstrated among patients with idiopathic intracranial hypertension.
- If repeated lumbar punctures and treatment with acetazolamide do not control the intracranial pressure within about two weeks, surgical drainage of the cerebrospinal fluid is indicated, usually by a lumboperitoneal shunt.
- If the visual fields deteriorate, fenestration of the optic-nerve sheath should be considered.

Reversible cerebral vasoconstriction syndrome

- Reversible cerebral vasoconstriction syndrome (RCVS) is characterised by severe headaches, with or without other acute neurological symptoms, and diffuse segmental constriction of cerebral arteries that resolves spontaneously within 3 months.
- Manifestations are attributed to a transient disturbance of the regulation of cerebral arterial tone.
- Thunderclap headache—severe pain peaking in seconds—is usually the first symptom and typically recurs for 1–2 weeks.
- Ischaemic and haemorrhagic stroke are the major complications of the syndrome.

- In 2007, Calabrese and colleagues proposed the name RCVS
- **Previous names for reversible cerebral vasoconstriction syndrome**
 - • Isolated benign cerebral vasculitis
 - • Acute benign cerebral angiopathy
 - • Reversible cerebral segmental vasoconstriction
 - • Call or Call-Fleming syndrome
 - • CNS pseudovasculitis
 - • Benign angiopathy of the CNS
 - • Post-partum angiopathy
 - • Migraine angiitis
 - • Primary thunderclap headache

- RCVS has been reported in people aged from 10 to 76 years, but occurrence peaks at around 42 years and
- the syndrome is more common in women than in men
- **More than half the cases occur post partum**

- Clinical manifestations typically follow an acute and self-limiting course without new symptoms after 1 month.
- Headache is the main symptom and often remains the only manifestation of RCVS.
- Onset is acute with thunderclap headache—extreme head pain peaking in less than 1 min, mimicking that of a ruptured aneurysm
- Typical headache is bilateral (although it can be unilateral), with posterior onset followed by diffuse pain.
- Nausea, vomiting, photophobia, and phonophobia frequently occur.

- By contrast with the headaches associated with ruptured aneurysms, the severe pain of RCVS is short lived (usually lasting 1–3 h).
- A single attack is possible, but usually patients have a mean of four attacks, during 1–4 week
- all noteworthy headaches are generally gone 3 weeks after onset.
- Associated neck pain should prompt investigations for cervical artery dissection
- Focal deficits, which can be transient or persistent, and seizures have been reported in 8–43% and 1–17%, respectively

- Transient focal deficits are present in slightly more than 10% of patients, last from 1 min to 4 h, and are most frequently visual, but sensory, dysphasic, or motor deficits can also occur.
- Persistent deficits, including hemiplegia, aphasia, hemianopia, or cortical blindness, suggest a stroke.
- A third of patients have surges in blood pressure during acute headaches because of the pain, the syndrome itself, or an associated disorder.

- **Laboratory investigations:**
- The results of blood counts, measurements of ESR and concentrations of serum electrolytes, and liver and renal function tests are usually normal in patients with RCVS.
- Tests for angiitis, including measurements of rheumatoid factor, antinuclear and antineutrophil cytoplasmic antibodies, and tests for Lyme disease are generally negative.
- Urinary concentrations of vanillylmandelic acid and 5-hydroxy indoleacetic acid should be measured to exclude a diagnosis of pheochromocytoma.
- Serum and urine toxicology screens should be done to check for drug use.

- Slight abnormalities of CSF are reported
- an excess of white blood cells (5–35 per μL), red blood cells with or without visible subarachnoid blood on an MRI scan
- increased protein concentrations of as much as 100 mg/dL
- If the white blood cell count exceeds 10 cells per μL or the protein concentration exceeds 80 mg/dL, or if both measures are exceeded, analysis of CSF should be repeated after a few weeks to ensure that concentrations have returned to normal.

- **Neuroimaging**
- Brain scans of many patients with RCVS look healthy despite the presence of diffuse vasoconstriction on concomitant cerebral angiograms.
- Lesions include —
- convexity subarachnoid haemorrhage,
- intracerebral haemorrhage,
- cerebral infarction and
- reversible brain oedema.

- **Convexity subarachnoid haemorrhage**
- Convexity subarachnoid haemorrhages are nonaneurysmal, usually mild, unilateral or bilateral, and
- manifest as a hyperintense signal on fluid-attenuated inversion recovery (FLAIR) MRI and a hypointense signal on T2*-weighted MRI in a few sulcal spaces near the convexity.
- Convexity subarachnoid haemorrhage is usually diagnosed within the first week of headache onset, sometimes after an initial normal MRI.
- About 50% of cases are associated with another type of stroke, either at onset or later in the course of the disorder.

- **Focal intracerebral haemorrhage**
- Parenchymal haemorrhages are of variable volume,
- more frequently single than multiple and lobar than deep, and more often associated with another type of stroke.
- They occur early in the course of RCVS and are revealed mostly by a persisting focal deficit concomitant with thunderclap headache.
- Haemorrhagic forms of RCVS seem to be more common in women than in men and in people with migraine than in those without.

- **Cerebral infarction**
- Infarctions occur mainly in arterial watershed regions of the cerebral hemispheres, often between the posterior circulation and the carotid territories.
- most patients with infarctions present with a focal deficit (transient or persistent).
- Ischaemic strokes usually occur later than do haemorrhagic strokes in the course of RCVS.
- **Reversible brain oedema**
- Oedema is an early manifestation of RCVS and is usually diagnosed within a few days of clinical onset.
- It is more frequently associated with at least one variety of stroke than isolated

- Oedema is better seen on MRI than on CT scans, with symmetrical FLAIR hyperintensities showing a distribution similar to that of posterior reversible encephalopathy syndrome.
- Oedema usually totally reverses within 1 month of clinical onset, much earlier than does vasoconstriction.
- **Cerebral angiography**
- To diagnose RCVS, direct (transfemoral) or indirect (CT or magnetic resonance) cerebral angiography is needed to show segmental narrowing and dilatation (string of beads) of one or more arteries.

- Narrowing of arteries is not fixed; a repeat angiogram after a few days might show resolution of some vessels, with eventual new constrictions often affecting more proximal vessels.
- Furthermore, the patient's first angiogram, irrespective of type, might be normal if it is done early—ie, within a week of clinical onset—even in the presence of haemorrhage or brain oedema.

- **Ultrasonography**
- Cervical ultrasonography is normal except in cases of RCVS associated with cervical arterial dissection.
- Transcranial doppler ultrasonography can be useful in monitoring cerebral vasoconstriction

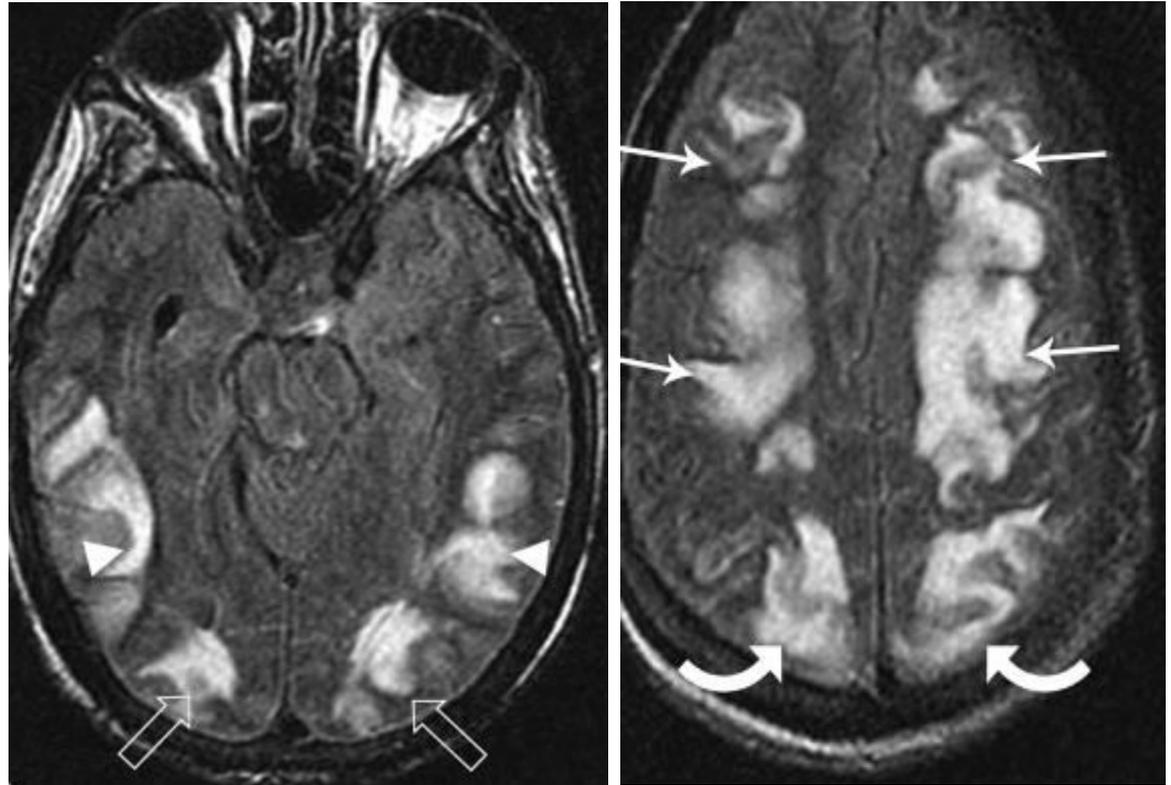
- **Diagnostic criteria for reversible cerebral vasoconstriction syndrome**
- I. • Acute and severe headache (often thunderclap) with or without focal deficits or seizures
- II. • Uniphasic course without new symptoms more than 1 month after clinical onset
- III. • Segmental vasoconstriction of cerebral arteries shown by indirect (eg, magnetic resonance or CT) or direct catheter angiography
- IV. • No evidence of aneurysmal subarachnoid haemorrhage
- V. • Normal or near-normal CSF (protein concentrations <100 mg/dL, <15 white blood cells per μ L)
- VI. • Complete or substantial normalisation of arteries shown by follow-up indirect or direct angiography within 12 weeks of clinical onset

- All patients need symptomatic management, which is primarily based on the identification and elimination of any precipitating or aggravating factors.
- Patients should be told to rest and advised to avoid- physical exertion, Valsalva manoeuvres, and other headache triggers for a few days to a few weeks, depending on initial severity.
- Any vasoactive drugs should be stopped and avoided even after disease resolution.
- Treatment should include analgesics, antiepileptic drugs , monitoring of blood pressure.

- Clinicians should treat hypertension according to the guidelines for patients with acute stroke, but should keep in mind that hypotension in the setting of cerebral vasoconstriction is potentially more dangerous.
- Drugs targeted at vasospasm can be considered when cerebral vasoconstriction has been assessed.
- Nimodipine, verapamil, and magnesium sulphate have been used to relieve arterial narrowing.
- Nimodipine was given orally at the dose used for the prevention of vasospasm in aneurysmal subarachnoid haemorrhage. (60 mg/4th hourly).
- Duration of treatment ranged from 4 to 12 weeks.
- Although nimodipine seemed to reduce the number and intensity of headaches, prospective and retrospective large studies suggest that it does not affect the timecourse of cerebral vasoconstriction.

Posterior Reversible Encephalopathy Syndrome

Axial MR images (fluid-attenuated inversion recovery) demonstrate extensive vasogenic edema in the frontal lobes (*arrows*), parietal region (*curved arrows*), occipital lobes (*open arrows*), and temporal lobes (*arrowheads*), bilaterally, consistent with PRES.



- PRES is characterized by variable associations of seizure activity, consciousness impairment, headaches, visual abnormalities, nausea/vomiting, and focal neurological signs.
- The cerebral imaging abnormalities are often symmetric and predominate in the posterior white matter.
- the main abnormality is cerebral vasogenic edema, the pathogenesis of which is still under debate.
- PRES is typically reversible once the cause is removed.
- PRES has been reported in patients aged 4 to 90 years, although most cases occur in young to middle-aged adults, the mean age ranging from 39 to 47 years. There is a marked female predominance.

- PRES is a clinicoradiological entity. The intensity and severity of clinical manifestations vary and may require ICU admission.
- The combination of suggestive clinical manifestations and radiological criteria establishes the diagnosis of PRES.

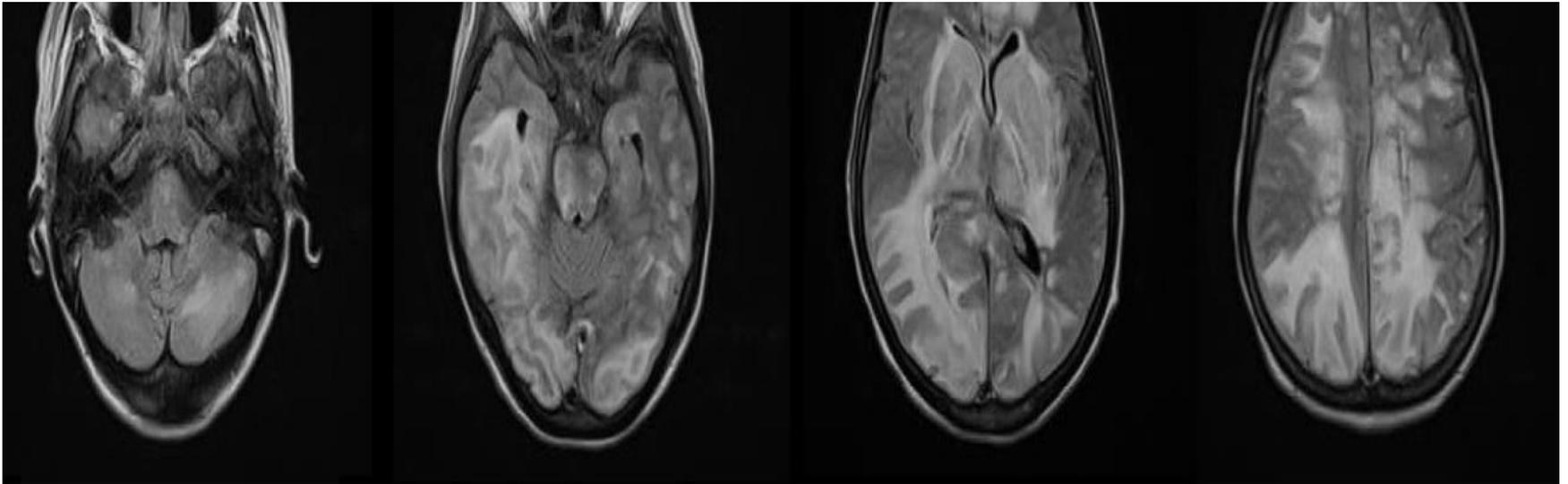
- The typical features of PRES consist of consciousness impairment, seizure activity, headaches, visual abnormalities, nausea/vomiting, and focal neurological signs.
- Consciousness impairment may range in severity from confusion, somnolence, and lethargy to encephalopathy or coma.
- Seizure activity occurs in up to 92 % of cases. The seizures are rarely isolated (23 %-28 %) . Secondary generalized seizures are common.
- Visual abnormalities were found in 26 % to 67 % of patients and consisted of blurred vision (7 % -18 %), visual neglect (4 % -27 %), homonymous hemianopsia (4 % -20 %), visual hallucinations (3 % -5 %), and cortical blindness (8 % -33 %).

- Headaches and nausea/vomiting were reported in 26 % to 53 % of patients.
- Focal neurological signs were either not mentioned at all or reported in only 3 % to 17 % of cases.
- Acute hypertension is not usually described among the main signs of PRES. However, hypertension has been reported in most studies , in 67 %to 80 % of patients.
- Acute hypertensive emergency was not significantly associated with the intensity of the clinical or radiological manifestations of PRES .
- Therefore, high mean blood pressure is often observed in PRES but its level is not correlated to the severity of PRES.

Radiological Characteristics of PRES

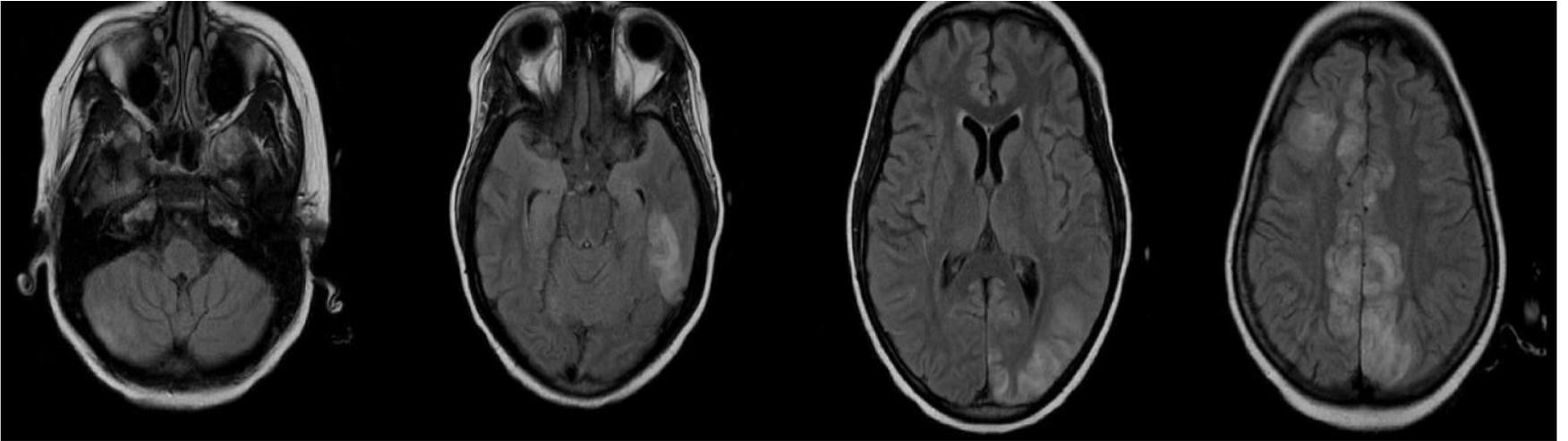
- **The four radiological patterns of PRES**
 - a. Holohemispheric watershed pattern (23 %)
 - b. Superior frontal sulcus pattern (27 %)
 - c. Dominant parietal-occipital pattern (22 %)
 - d. Partial or asymmetric expression of the primary patterns (28 %)

Holohemispheric watershed pattern (23 %)



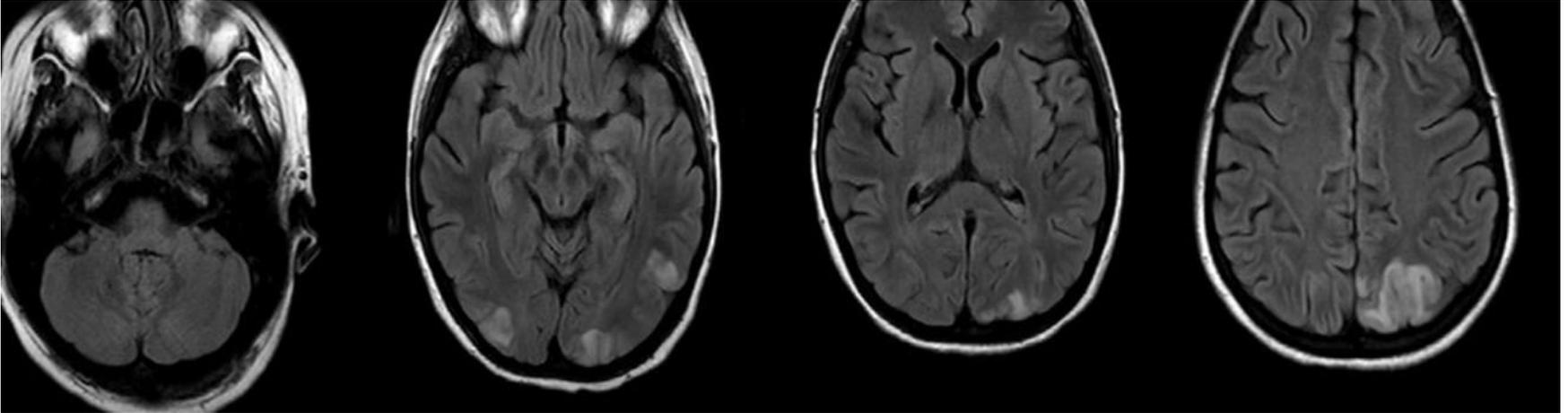
A swath of confluent vasogenic edema extends through the frontal, parietal, and occipital lobes. Involvement of the temporal lobes is less marked. This topography matches the watershed zone between the anterior and posterior cerebral arteries, on the one hand, and the middle cerebral artery, on the other

Superior frontal sulcus pattern



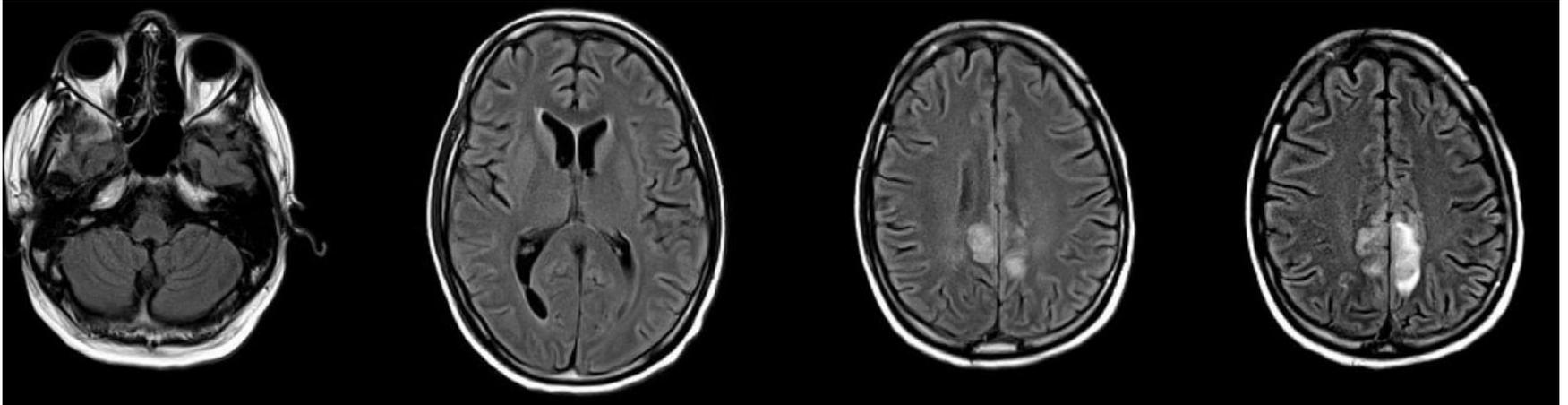
Patchy edema predominates in the frontal lobes along the superior frontal sulci. The parietal and occipital lobes are variably involved

Dominant parietal-occipital pattern



In this pattern previously thought to be typical of PRES, the posterior part of the parietal and occipital lobes is predominantly involved

Partial or asymmetric expression of the primary patterns



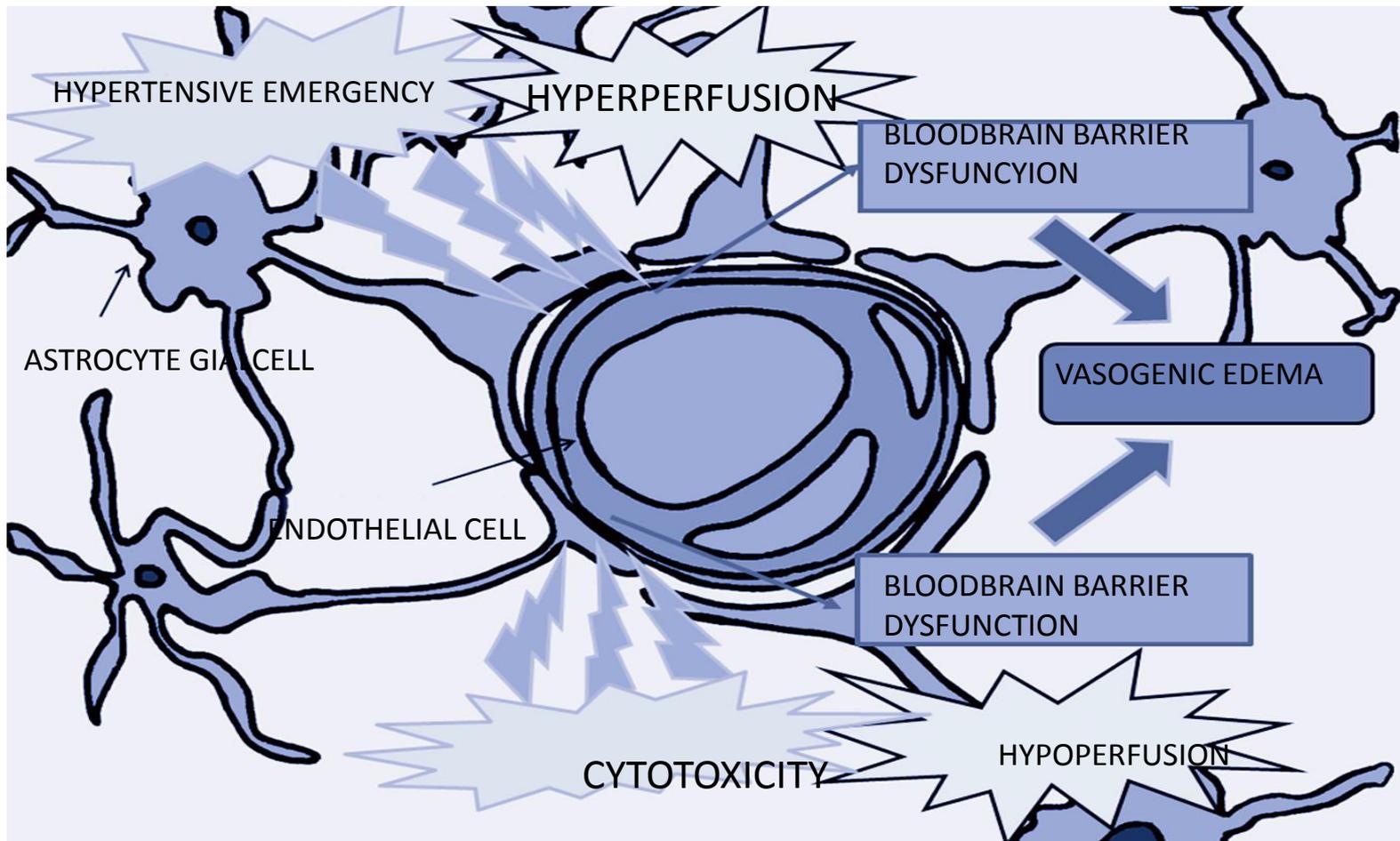
The partial form is defined as bilateral absence of edema in either the parietal or the occipital lobes. The frontal lobes are often involved. The asymmetric form is characterized by unilateral absence of edema in either a parietal or an occipital lobe. Finally, in the partial and asymmetric form, there is both absence of involvement of either the parietal or the occipital lobes and asymmetric abnormalities in the affected parietal or occipital lobes

- MRI: Cerebral MRI is the key investigation for the diagnosis of PRES.
- Fluid-attenuated inversion recovery (FLAIR) sequences have been shown to improve the diagnosis of PRES and the detection of subcortical and cortical lesions in PRES.

Complications

- **Cerebral ischemia:** Cerebral infarction is among the early signs of non-reversible damage associated with adverse outcomes.
- In this setting, every effort must be taken to exclude a reversible cerebral vasoconstriction syndrome defined as at least two narrowings per artery on two different cerebral arteries at brain magnetic resonance angiography (MRA) or at conventional angiography.
- **Cerebral hemorrhage:** Cerebral hemorrhage is uncommon in PRES
- Cerebral hemorrhage may be more common among patients with allogeneic bone marrow transplantation or anticoagulant treatment.
- significant association has been reported between edema severity on FLAIR sequences and bleeding risk.
- **Cerebral herniation:**
- Posterior edema, particularly when located in the cerebellum and brainstem, may cause transtentorial cerebral herniation.

- PATHOPHYSIOLOGY:
- The pathophysiology of PRES remains controversial
- One involves impaired cerebral autoregulation responsible for an increase in cerebral blood flow.
- other involves endothelial dysfunction with cerebral hypoperfusion.
- the result of the cerebral blood perfusion abnormalities is bloodbrain barrier dysfunction with cerebral vasogenic edema.



- **Cerebral Hyperperfusion Results in Vasogenic Edema by Exceeding the Capacity for Autoregulation of Perfusion Pressure:**
- When mean arterial blood pressure (MAP) is within the 60–120 mmHg range,
- cerebral autoregulation via variations in vasoconstriction and vasodilatation keeps the CBF at about 50 ml/100 g/min in healthy individuals.
- MAP must exceed 170 mmHg (systolic/diastolic bloodpressure of 220/110 mmHg). However, a smaller MAP increase of only 50 mm Hg
- (systolic/diastolic blood pressure of 160/100 mmHg) in a patient with *de novo*
- hypertension is sufficient to trigger severe vasoconstriction

- Cerebral hyperperfusion leads to the release of the vasodilators nitric oxide(NO) and prostacyclin under the influence Of endothelial agonists such as acetylcholine, norepinephrine, and substance P.
- Concomitantly, there is overproduction of catecholamines, vasopressin, thromboxane, and endothelin 1. These substances increase vasoreactivity and activate the renin-angiotensin-aldosterone system.
- Angiotensin II activates the gene expression of pro-inflammatory cytokines such as interleukin (IL)-6 and the transcription of nuclear factor-kappa B (NF-κB), leading to direct cytotoxic effects on the blood vessel wall

- **Cerebral Hypoperfusion Related to Disruption of the Blood-brain Barrier Results in Vasogenic Edema:**
- Not all patients with PRES have hypertension. In patients with PRES and normal blood pressure, cytotoxicity has been hypothesized to be the mechanism underlying the brain edema.
- Causes of PRES without hypertension include cyclosporine toxicity, and infection/sepsis/septic shock.
- The Immune system (T-cell) activation leads to endothelial cell activation with the release of various mediators such as histamine, free radicals, NO, bradykinin, and arachidonic acid .
- These mediators activate the production of pro-inflammatory cytokines (e.g., tumor necrosis factor[TNF]- α , IL-1, IL-6, and interferon [IFN]- γ) .

- All these changes result in vascular instability with vasoconstriction and downstream hypoperfusion. Blood-brain barrier dysfunction occurs, leading to vasogenic cerebral edema.

Conditions Most Commonly Associated With PRES

- **Preeclampsia/Eclampsia**
- Pre-eclampsia/eclampsia was present in 7 % to 20 % of patients with PRES. The outcome was usually favorable. Hypertension was a prominent feature at presentation. PRES onset occurred from 28 weeks' gestational age to day 13 postpartum.
- **Toxic Agents**
- Exposure to toxic agents is the most common condition associated with PRES, exposure to cancer chemotherapy and/or immunosuppressive therapy.

- **Hypertension**
- Hypertension is the second most common condition associated with PRES, being present in 6 % to 72 % of cases.
- **Infection/Sepsis/Septic Shock**
- Infections have been reported in 8 % to 24 % of cases . The most common situation was PRES onset within 2 weeks after a Gram-positive bloodstream infection, often with hypertension at diagnosis.
- **Autoimmune Disease**
- Autoimmune disease has been encountered in 8 % to 10 % of cases.
- PRES has been reported in patients with systemic lupus erythematosus , systemic sclerosis , polyarteritis nodosa , Wegener's granulomatosis , thrombotic microangiopathy

- **General measures**
- Patients with PRES require the symptomatic measures usually taken in the ICU.
- The need for upper airway protection should be evaluated continuously in patients with marked consciousness impairment or seizure activity
- Antiepileptic treatment, appropriate for the electrical and clinical pattern in the patient, should be initiated on an emergency basis and according to current guidelines. Patients with persistent seizure activity at ICU admission should be given intravenous benzodiazepines
- ANTIHYPERTENSIVES: The aim is not to normalize the blood pressure but rather to decrease the MAP by 20–25 % within the first 2 hours and to bring the blood pressure down to 160/100 mmHg within the first 6 hours.
- Correction of the underlying cause of PRES

STROKE

- **Definition:** Stroke is an acute neurological impairment that follows interruption of blood supply to a specific part of the brain.
- Blood supply may be interrupted by thrombosis (either arterial or venous) or embolism, or in a smaller proportion of women by hemorrhage.
- Most (up to 90%) strokes occur peripartum and up to a few weeks after the birth

Risk factors for stroke

- ***Physiological risk factors***
- Progressive physiological changes occurring throughout pregnancy predispose to stroke including
- 1.increasing hypercoagulability,
- 2. venous stasis and vascular wall changes.
- 3. Pushing in the active phase of the second stage of labor involves episodes of significantly increased intrathoracic pressure (Valsalva) and elevation of cerebral perfusion pressure, which may lead to changes in cerebral blood flow – particularly where cerebral autoregulation or anatomy is disordered.

- ***Obstetric risk factors***

- The main obstetric factor associated with an increased risk of stroke is pre-eclampsia and eclampsia, in particular uncontrolled systolic hypertension.
- Age more than 35 years, greater parity and multiple gestation are all risk factors for stroke.

- ***Co-morbidity risk factors***
- Women who become pregnant may have comorbidity that increases the risk of vascular events including stroke; such factors include:
 - 1.obesity (BMI >30 kg/m²),
 2. diabetes,
 - 3.pre-existing hypertension,
 - 4.renal and heart disease,
 - 5.vasculopathies such as sickle cell disease,
 - 6.vasculitis and collagen or atherosclerotic disease.
 - 7.Alcohol, tobacco and cocaine use may cause vasculopathy or hypertension.

- Stroke presents as in the non-pregnant woman and clinical features may suggest either infarction or hemorrhage, neuroimaging is required to confirm the diagnosis.
- The possibility of stroke should be considered in any woman who presents with any of the following symptoms:
 - Sudden weakness or numbness of face, arm or leg, especially if on one side of the body
 - Sudden confusion
 - Trouble speaking or understanding
 - Sudden trouble seeing in one or both eyes without a prior history of migraines
 - Sudden trouble walking
 - Sudden loss of balance or coordination not readily attributable to pregnancy
 - Sudden severe headache with no known cause

- Cincinnati Stroke Scale:
- **Three elements**
- **1. Facial droop**
 - a. Have the patient smile and assess for facial droop
 - i. Normal: both sides of face move equally
 - ii. Abnormal: one side of face does not move
- **2. Arm drift**
 - a. Have the patient hold both arms out and up with palms facing upwards
 - i. Normal: both arms move equally
 - ii. Abnormal: one arm drifts compared with the other
- **3. Speech**
 - a. Have the patient repeat a sentence
 - i. Normal: patient uses correct words with no slurring
 - ii. Abnormal: slurred or inappropriate words or mute
- If any of the three elements in the scale or any other neurologic findings are newly abnormal, the possibility of acute stroke is high and the patient should have urgent imaging

- all patients with symptoms suggestive of stroke require prompt neuroimaging to determine whether they have had an ischemic stroke that may benefit from the use of thrombolytic therapy.
- Have a neurologic assessment and head CT performed within 25 min of presentation
- Have the head CT scan read with determination of whether they are candidates for fibrinolytic therapy
- within 45 min of presentation Receive fibrinolytic therapy within 60 min of presentation to the A&E and no longer than 180 min since the time of onset of symptoms.
- The use of thrombolysis should be considered for pregnant and postpartum women with severe acute cerebral non-hemorrhagic infarction if it can administered within 180 minutes of onset of the neurologic deficit.
- Thrombolysis is well tolerated by the fetus in pregnancy and does not seem to increase the risk of placental abruption, so should not be withheld if the maternal condition is life-threatening.
- The tPA administered in a dose of 0.9 mg/kg, 10 percent of which was given as an initial bolus, followed by an infusion of the remainder over 1 h. A dose of 90 mg is not exceeded, this being lower than the dose used for myocardial infarction.
- The relative improvement in neurologic state came at the expense of a 6 percent risk of symptomatic cerebral hemorrhage.

- It is not recommended to use thrombolytics for acute ischemic stroke in the setting of probable or confirmed preeclampsia.
- Low dose aspirin is the mainstay of treatment for acute ischemic stroke.
- Unfractionated or lowmolecular weight heparin is not recommended for acute stroke or stroke prevention except in the case of stroke from cardioembolism, arterial dissection or large artery intraluminal thrombus.
- Warfarin is teratogenic and usually avoided in pregnancy.

- Perhaps the most challenging and common differential diagnosis for stroke in the obstetric population is migrainous aura. Migrainous auras are typically brief and more likely to be positive (the alteration of a sensory perception) rather than negative (the absence of a perception), e.g. wavy lines in vision versus no vision or 'pins and needles' versus numbness.
- Visual or sensory symptoms should be one sided, gradually progress and last between 5 and 60 minutes. If more than one aura symptom is present, symptoms should occur in succession rather than simultaneously.
- If there is doubt about whether a patient's symptoms represent stroke/transient ischemic attack or migrainous aura, an evaluation by a neuroimaging is advisable.

- ***Intracranial hemorrhage:***
- Most intracranial hemorrhage occurring during an otherwise normal pregnancy is the result of aneurysmal SAH and arteriovenous malformation (AVM).
- Hypertension, smoking, alcohol and family history are all risk factors.
- The incidence of SAH from aneurysmal rupture is 3–11 per 100,000 pregnancies, but 50% of all aneurysmal rupture in women below 40 occurs in the context of pregnancy.

- Presentation of intracranial hemorrhage is the same as in the non-pregnant woman.
- Symptoms are dominated by the sudden onset of headache, often described as “the worst headache of my life.”

- Meningeal irritation (due to blood spreading through the cerebrospinal fluid),
- altered consciousness, collapse or vomiting at onset, and the absence of lateralizing neurologic findings are features that are characteristic of SAH but not universal.
- CT scan is very sensitive for SAH in the first 12 hours after the event, but is less sensitive with smaller bleeds and as the days go by after the initial event.
- Lumbar puncture is recommended in patients with a history suggestive of SAH who have a normal CT scan, especially if more than a day has passed since the onset of their symptoms.
- The presence of xanthochromia on cerebrospinal fluid is highly suggestive of a SAH but will not be present until 2–6 hours after the acute event.

- the use of FLAIR and T2 sequences with MRI may be as good or better than CT at identifying an early SAH.
- Ruptured aneurysmal SAH may be complicated by rebleeding – with an associated mortality rate of 50–70%.
- Vasospasm, cerebral infarction, hydrocephalus, increased intracranial pressure, seizures and hyponatremia are other possible complications.
- Medical treatment usually involves intravenous fluids, bed rest, compression stockings, analgesia, laxatives and nimodipine 60 mg 4-hourly.
- Once the diagnosis is established, the etiology for the SAH must be determined with cerebral angiography, CT angiography or MR angiography.

- Definitive treatment usually involves endovascular coiling or surgical clipping and the
- timing of these interventions will be decided by the neurosurgeon.
- In most cases, treatment of the mother is the primary concern, although near to or during labor, in some cases the baby may be delivered first.
- Women who have had a previous aneurysm completely obliterated by clipping or coiling may consider vaginal delivery. Use of epidural anesthesia is advised.

- In general, management of unruptured aneurysms should be the same as in the non-pregnant state, and guided by ISUIA (the International Study on Unruptured Intracranial Aneurysms).
- most clinicians would recommend early good pain control, ensuring blood pressure remains less than 140/90 mmHg and limiting the active phase of the second stage of labor.

THANK YOU