Original Article

Spectrum of Neurological Complications of Pregnancy on Magnetic Resonance Imaging

Sheema Ahmad Posh, Suhail Rafiq[1], Beenish Jeelani, Saima Wani

Department of Gynecology and Obstetrics, SKIMS, [1]Department of Radiodiagnosis and Imaging, GMC, Srinagar, Jammu and Kashmir, India

Abstract

Introduction: Catastrophic neurological decline, although exceedingly rare, takes a huge toll on pregnant population. In view of varied symptomatology and risks to the fetus, diagnosis and management of the neurological disorders in pregnancy is always a challenging task. The evaluation and management should be performed in a stepwise fashion and requires a multidisciplinary approach. Radiological imaging, especially magnetic resonance imaging (MRI), has revolutionized the diagnosis of these disorders, thereby exacerbating maternal and fetal outcome. **Aim:** The present study was conducted with the aim to characterize some of the significant neurological disorders complicating pregnancy and puerperium and to study the role of imaging, especially MRI, in differentiation and exclusion of various neurologic conditions, which helps an obstetrician to point to a specific diagnosis and management. **Materials and Methods:** Our study was an observational study conducted in the Department of Obstetrics and Gynecology in collaboration with the Department of Radiodiagnosis of GMC, Srinagar, from June 2018 to January 2020. The images were obtained with MRI and subjected for radiological interpretation. **Results:** A total of 750 patients were included in the study out of which 25 patients had neurological complications. Out of 25 patients, 13 were in the antenatal period and 12 were in the puerperium. There were 10 (40.0%) cases of posterior reversible encephalopathy syndrome (PRES), 6 (24%) cases of cerebral venous thrombosis (CVT), 3 (12.0%) cases of embolic infarcts, 2 (8.0%) cases of status epilepticus, 2 (8.0%) cases of pituitary apoplexy, 1 (4.0%) case of Wernicke encephalopathy, and 1 (4.0%) case of metastasis to brain (choriocarcinoma). **Conclusion:** Diagnosis of neurological complications of pregnancy and postpartum plays a crucial role in reducing fetomaternal morbidity and mortality. MRI stands above all imaging modalities in early diagnosis of these neurological complications, simultaneously taking care of fetal safety as well. The most common neurological complications that cause increased maternal mortality are PRES and CVT. Hence, early imaging can help in early and appropriate management of serious pregnancy-related neurological catastrophes.

Keywords: Magnetic resonance imaging, neurological disorder, posterior reversible encephalopathy syndrome, puerperium

Introduction

A unique challenge to an obstetrician in pregnancy is the early diagnosis and evaluation of neurologic disorders, which pose a serious threat to the life of the mother as well as the fetus. Hormonal changes, increased blood volume, changes in sleep, higher risk of clotting during pregnancy, as well as changes in mechanical pressure due to an enlarged uterus can worsen preexisting neurologic conditions and introduce new neurologic symptoms. The central nervous system (CNS) and the peripheral nervous system do experience these alterations. The neurologic symptoms seen in pregnant and postpartum women may be due to exacerbation of a preexisting medical condition, such as multiple sclerosis or a seizure disorder; the initial manifestation of a primary CNS-related problem as brain neoplasm, or acute ischemic stroke; or a neurologic problem unique to pregnancy and the postpartum period, such as posterior reversible encephalopathy syndrome (PRES), eclampsia, postpartum cerebral angiopathy, Sheehan syndrome, and lymphocytic adenohypophysitis.[1]

During the course of pregnancy, one of the most common symptoms encountered due to intracranial disease is headache. An acute-onset headache must be taken into

Access this article online

Quick Response Code:

Website: www.matrixscimed.org

DOI: 10.4103/mtsm.mtsm_9_21

Address for correspondence: Dr. Suhail Rafiq, Department of Radiodiagnosis, GMC, Srinagar, Jammu and Kashmir, India. E-mail: suhailrafiq777@gmail.com

How to cite this article: Posh SA, Rafiq S, Jeelani B, Wani S. Spectrum of neurological complications of pregnancy on magnetic resonance imaging. Matrix Sci Med 2022;6:53-9.

Received: 22-04-2021, **Accepted:** 07-10-2021, **Published:** 12-04-2022

serious consideration.[2] Usually, the interaction of estrogen and other reproductive hormones with the trigeminovascular system produces physiologic changes that lead to headache. However, a new-onset or worsening headache, a headache with changing characteristics, multiple headaches, and any headache accompanied by vision loss or blurring or a neurologic deficit must be identified with great vigilance.[3,4] Other neurologic disorders that can occur in pregnancy and puerperium include PRES, cerebral vein thrombosis, status epilepticus, metastasis, embolic infarcts, Wernicke encephalopathy, and pituitary apoplexy.[5]

Magnetic resonance imaging

Magnetic resonance imaging (MRI), when available, remains the modality of choice for imaging pregnant women – mainly because it involves no radiation exposure and enables excellent soft-tissue differentiation and thus has better diagnostic accuracy. Relatively, recent data suggest that MRI performed using a 1.5-Tesla magnet is safe for performing imaging in pregnant women during any trimester.[6,7]

Materials and Methods

This hospital-based observational study was conducted in the Department of Obstetrics and Gynaecology, Sher-i-Kashmir Institute of Medical Sciences, Srinagar, along with Department of Radiodiagnosis and Imaging, GMC, Srinagar, from June 2018 to January 2020. Pregnant patients with neurological disorders who were referred for radiological evaluation and who had significant findings on MRI were taken up for the study. Twenty-five pregnant women who presented to us with neurological manifestations, irrespective of their period of gestation and pregnancy outcome, were included in the study. Most common presenting symptoms were headache, convulsions, neurosensory deficits, loss of consciousness, altered sensorium, blurring of vision, vertigo, loss of bladder, and bowel control. Magnetic resonance (MR) sequences along with time of flight were taken using 1.5-Tesla MRI.

Inclusion criteria

1. Pregnant patients presenting in the second trimester, third trimester, and postpartum
2. Pregnant patients with neurological manifestations of the CNS and pituitary gland.

Exclusion criteria

1. Refusal to participate in the study
2. Claustrophobic patients
3. Patients presenting in the first trimester
4. Contraindications to MRI – metallic implants, pacemakers, contrast allergy.

Results

A total of 750 patients were included in the study out of which 25 patients had neurological complications. Out of 25 patients, 13 were in the antenatal period and 12 were in puerperium. There were 10 (40.0%) cases of PRES [Figure 1], 6 (24%) cases of cerebral venous thrombosis (CVT) [Figure 2], 3 (12.0%) cases of embolic infarcts [Figure 3], 2 (8.0%) cases of status epilepticus, 2 (8.0%) cases of pituitary apoplexy [Figure 4], 1 (4.0%) case of Wernicke encephalopathy [Figure 5], and 1 (4.0%) case of metastasis to brain (choriocarcinoma) [Figure 6 and Table 1].

Regarding clinical profile, 19 (76%) patients presented with headache, 15 (60%) patients presented with vomiting, seizures occurred in 6 (24%) patients, neurosensory deficits occurred in 5 (20%) patients, loss of consciousness occurred in 3 (12%) patients, and visual disturbances occurred in 2 (8%) patients [Table 2].

Discussion

Pregnancy adds a unique and, at times, challenging facet to the management of neurologic disease. In both pregnancy and puerperium, the number of pathologic manifestations involves the CNS and pituitary gland. In our study, we found that the most common presenting complaint of patients in both pregnancy and postpartum phase is headache [Table 2]. A pregnant patient presenting with vomiting should never be taken lightly as it can be the first warning symptom of a CNS disease. Seizures play a significant role in increasing maternal morbidity and mortality. Loss of consciousness, neurosensory deficits, and visual disturbances, although rare, warrant specific evaluation and treatment. The most common neurological disorders that we encountered in pregnancy and puerperium are described below:

Posterior reversible encephalopathy syndrome

PRES, earlier known as reversible posterior leukoencephalopathy syndrome, is a cliniconeuroradiologic entity of heterogeneous etiologies.[8] Occurring most commonly in young, middle-aged adults with female preponderance,[9,10] the pathophysiology of PRES is based on two hypotheses. The most widely accepted theory is the impairment of cerebral autoregulation and cerebral hyperperfusion occurring due to rapidly developing

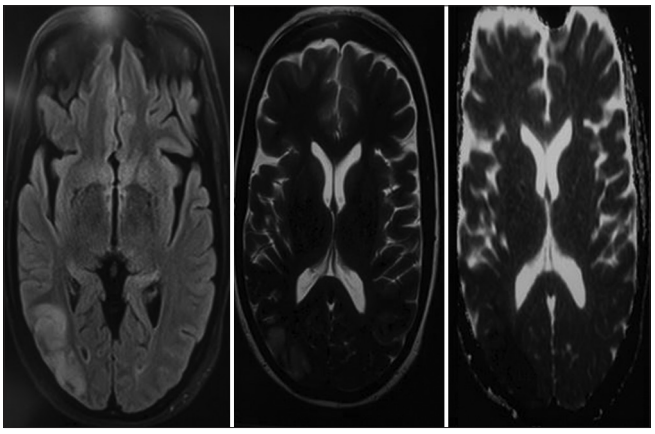

Figure 1: There is evidence of T2, FLAIR hyperintensity with diffusion restriction involving right occipital cortex, suggestive of atypical posterior reversible encephalopathy syndrome

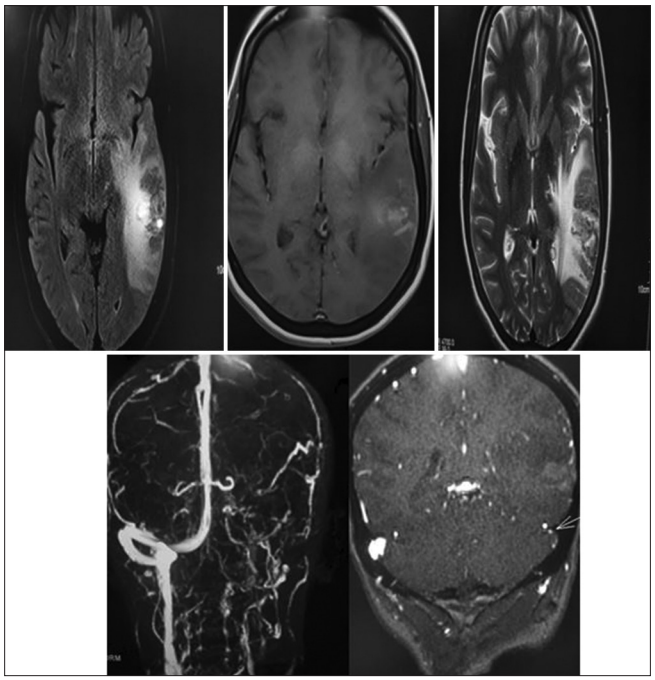

Figure 2: There is evidence of T2/FLAIR heterogenous hyperintense area in the left parietal region with areas of T1 hyperintensity corresponding to hemorrhage. Magnetic resonance venogram showing nonvisualization of left transverse, sigmoid sinus, and internal jugular vein with filling defect (arrow)

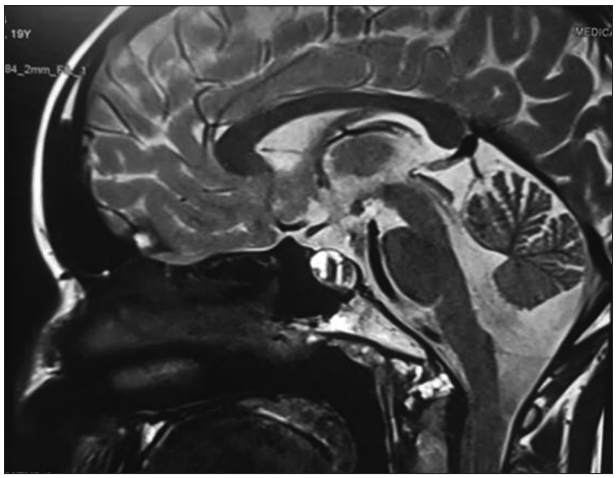

Figure 4: There is evidence of multiple FLAIR hyperintense lesions in high frontal regions with diffusion restriction, suggestive of infarcts in anterior cerebral artery territory

hypertension, leading to extravasation of protein and fluid which produces focal vasogenic edema.[11,12] The second hypothesis implicates endothelial dysfunction with cerebral hypoperfusion. Altered cerebral blood perfusion results in blood–brain barrier dysfunction and cerebral vasogenic edema.[10]

PRES has been clinically expressed by neurological manifestations derived from cortical and subcortical changes localized in the posterior regions of cerebral

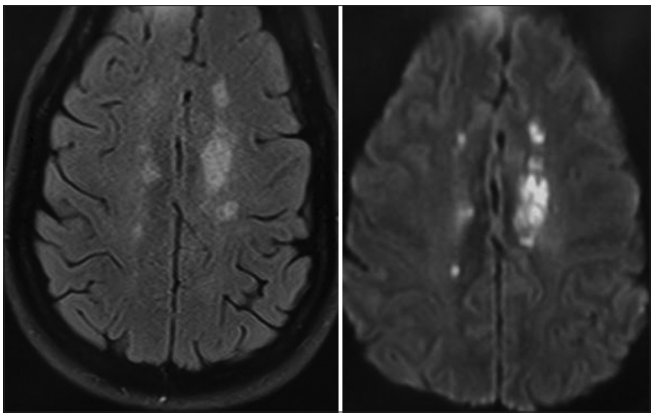

Figure 3: There is evidence of multiple FLAIR hyperintense lesions in high frontal regions with diffusion restriction, suggestive of infarcts in anterior cerebral artery territory

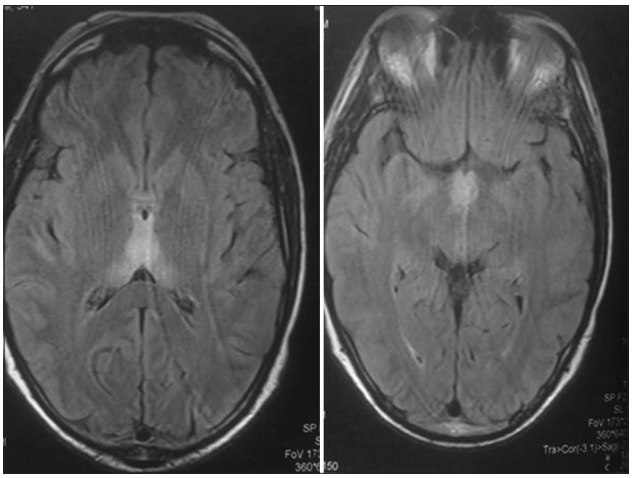

Figure 5: There is evidence of FLAIR hyperintensity involving medial thalami, mammillary bodies, and around third ventricle, suggestive of Wernicke's encephalopathy

hemispheres, cerebral trunk, and cerebellum. The causes underlying such changes in brain matter include hypertensive encephalopathy, eclampsia,[13] renal failure with hypertension, immunosuppressive agents,[14] cytotoxic drugs (cyclosporine A, interferon alpha, cisplatin, cytarabine, erythropoietin, tacrolimus), intravenous immunoglobulin, collagen vascular disorders (systemic lupus erythematosus),[15] hypercalcemia,[16] sepsis, postdural puncture and spinal anesthesia.[17-19]

The clinical profile of PRES includes headaches, seizures, focal neurological deficits, and visual disturbances, impairment of consciousness (confusion, somnolence, lethargy, and coma). PRES is reversible.[9,10] MRI is the gold standard for diagnosis of PRES. There are four radiological patterns of PRES, according to distribution of edema: holohemispheric watershed pattern, superior frontal sulcus pattern, and dominant parieto-occipital pattern.[10]

Depending on the region of involvement, two types of PRES have been described:

- Typical PRES: Involves parieto-occipital lobes.

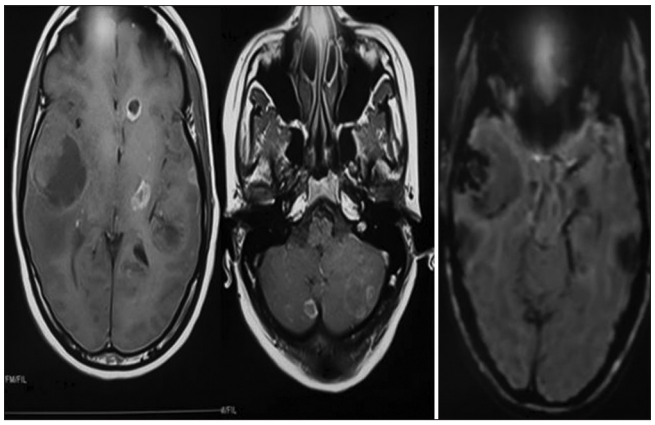

Figure 6: There is evidence of multiple supratentorial and infratentorial T1 hyperintense lesions with areas of blooming on susceptibility-weighted images in a known case of choriocarcinoma, suggestive hemorrhagic metastases

- Atypical PRES: When areas of brain other than parieto-occipital region are predominantly involved along with internal hemorrhage or diffusion restriction on diffusion-weighted imaging (DWI), the syndrome is called atypical PRES.

The management of PRES requires a multidisciplinary approach.[12] General measures include supportive treatment and maintenance of ABC of the patient. Treatment involves antihypertensives (for arterial hypertension), magnesium sulfate or phenytoin (for eclamptic seizures), vasodilators such as nitroglycerin to widen cerebral arteries, anti-edema therapy (mannitol), and withdrawal of the offending agent.[20]

Cerebral venous thrombosis

CVT is any thrombosis occurring in intracranial veins and sinuses. CVT is typically multifactorial as well as a rare disorder affecting five persons per million. The clinical presentation of CVT being diverse can pose a diagnostic challenge to the treating obstetrician; hence, early diagnosis, mostly by MRI, is crucial but often difficult, particularly during postpartum period because of the numerous causes of headache that may occur after delivery.[21] Most common sinuses involved by thrombosis are superior sagittal sinus and lateral sinus.

CVT accounts for 27%–57% of all pregnancy-related strokes.[22,23] CVT associated with pregnancy and puerperium has a more acute onset and better prognosis than thrombosis due to other causes. The hypercoagulability of pregnancy makes the environment favorable for CVT.

Increased levels of platelet count, platelet adhesiveness, fibrinogen, VII, VIII, and X, decreased levels of inhibitors of coagulant proteins S, rise in inhibitors of protein C levels, and Factor V Leiden and Factor II gene mutations are the predisposing factors for CVT in pregnancy and puerperium.[24]

The gold standard is the combination of MRI, which localizes the thrombus with MR venography, which shows

Table 1: Neurological disorders in pregnancy and puerperium

Diagnosis	Number of cases, *n* (%)
PRES	10 (40.0)
Cerebral venous thrombosis	6 (24)
Embolic infarcts	3 (12.0)
Status epilepticus	2 (8.0)
Pituitary apoplexy	2 (8.0)
Wernicke's encephalopathy	1 (4.0)
Metastasis (choriocarcinoma)	1 (4.0)

PRES: Posterior reversible encephalopathy syndrome

Table 2: Clinical presentation of neurological disorders in pregnancy and puerperium

Patient	*n*=25, *n* (%)
Headache	19 (76)
Vomiting	15 (60)
Seizure	6 (24)
Neurosensory deficits	5 (20)
Loss of consciousness	3 (12)
Visual disturbances	2 (8)

the nonvisualization of the same vessel. On MRI, usually, it shows the high-signal intensity of the venous sinuses with all routine sequences (usually on T1-weighted [T1W], T2-weighted, and FLAIR). On contrast-enhanced T1W, it usually shows high-signal intensity with a corresponding filling defect after gadolinium enhancement may develop within the 1st week after clinical onset. Early detection can be done with MRI within 7 days of clinical onset. Although not recommended in pregnancy, whole-brain CT perfusion may assist in establishing the diagnosis of CVT by detecting perfusion abnormalities that do not correspond with arterial territories.[25,26] The "empty delta sign" seen on CT venography has a reported sensitivity of 95% compared to DSA as the gold standard.

The most common symptom of CVT that occurs in 95% of patients is headache. Other manifestations are focal seizures, paresis, papilledema, altered consciousness, and isolated intracranial hypertension.[27]

Embolic infarcts

Embolic infarcts can occur from dissections due to prolonged difficult labor, cardiac valvular disease, and dilated peripartum cardiomyopathy. Dissections and obstetric hemorrhage can cause watershed infarcts. Frontal and parietal regions are the most common site of infarcts.[28] The use of DWI-MRI improves the accuracy of the subtype diagnosis of stroke. On the basis of the DWI findings, three types of lesions can be identified: (1) single subcortical lesion (diameter <15 mm); (2) large and scattered lesions in 1 vascular territory (≥15 mm; scattered small lesions <15 mm or confluent scattered lesions ≥15 mm); and (3) multiple lesions in multiple vascular territories as defined in recent studies.[29,30]

Status epilepticus

Status epilepticus is defined as an acute and prolonged seizure that persists for a sufficient length of time or is repeated frequently enough that recovery between attacks does not occur. It is associated with high fetomaternal morbidity and mortality. A widely accepted seizure duration to qualify as status epilepticus is 5 min in length.[31] In pregnancy, status occurs almost always due to eclampsia. However, other causes include stroke, hypoxia, metabolic derangement, toxicity (e.g., drugs), encephalitis, alcohol intoxication or withdrawal, and infections accompanied by fever.[32,33] MRI is the gold standard of diagnosis. The most common changes seen are increased T2 signal (best seen on FLAIR) with some swelling. These regions may demonstrate corresponding diffusion abnormalities, with increased DWI signal and in some instances reduced ADC values.[34,35] Postcontrast T1 imaging is extremely variable, ranging from no enhancement to marked enhancement, which may be gyriform or leptomeningeal in distribution.[34,36]

Pituitary apoplexy

Pituitary apoplexy is a rare but potentially life-threatening disorder, resulting from acute hemorrhage, infarction, or hemorrhagic infarction of the pituitary gland, causing significant morbidity and mortality if not promptly recognized and managed.[37] Causes include hypertension, major surgery (coronary artery bypass grafting), dynamic pituitary testing using gonadotropin-releasing hormone (GnRH), thyrotropin-releasing hormone, corticotropin-releasing hormone, insulin tolerance test, anticoagulation therapy, coagulopathies, estrogen therapy, initiation or withdrawal of dopaminergic therapy, radiation therapy and head trauma, and most importantly pregnancy.[38] Clinical manifestations include headache, nausea, vomiting, visual field defects, impairment of consciousness, hyponatremia, and central diabetes insipidus.[39-41] In pregnancy, pituitary apoplexy is often accompanied by endocrine abnormalities, among which Adrenocorticotropic hormone (ACTH) deficiency is the most relevant clinically because it causes life-threatening adrenal insufficiency.[39,42,43]

MRI is an essential diagnostic tool, confirming the diagnosis of pituitary apoplexy in over 90% of the patients.[38,44] On T1W images, hemorrhage usually manifests with hyperintensity compared with the rest of the brain.[45] MRI and MR angiogram techniques also help to distinguish an aneurism from pituitary apoplexy.[46] Pregnancy is not an absolute contraindication to MRI, and available data have not conclusively documented any deleterious effects of MRI on the developing fetus, with no special consideration for the first trimester versus any other trimester of pregnancy.[47] Moreover, MRI remains preferable to any imaging study using ionizing radiation. Initial treatment of acute pituitary apoplexy consists of preservation of electrolyte and fluid balance, alongside prompt replacement of deficient hormones (because of high risk of hypopituitarism), especially glucocorticoids, to treat both adrenal insufficiency and the effects of edema on suprasellar structures.[39,48]

Wernicke encephalopathy

It is also referred to as Wernicke-Korsakoff syndrome. It is a form of thiamine (Vitamin B1) deficiency and is typically seen in alcoholics. It is characterized by a triad of acute confusion, ataxia, and ophthalmoplegia. Thiamine deficiency results from malnutrition or malabsorption, which can occur for a number of reasons, especially by hyperemesis gravidarum in pregnancy. On imaging, it is commonly seen on MRI as areas of symmetrical increased T2/FLAIR signal involving the mammillary bodies, dorsomedial thalami, tectal plate, periaqueductal area, and/or around the third ventricle.[49,50] Treatment of acute Wernicke-Korsakoff syndrome is with intravenous thiamine hydrochloride, along with other vitamins/minerals, and treatment of the underlying cause (e.g., alcohol cessation). Untreated, there is high mortality of up to 20%.[51,52]

Choriocarcinoma

Choriocarcinoma is an aggressive, highly vascular tumor. When it is associated with gestation, it is often considered part of the spectrum of gestational trophoblastic disease; it is then termed gestational choriocarcinoma.[53] In the classic case of gestational choriocarcinoma, the tumor is derived from chorionic epithelium. It commonly arises in reproductive organs – uterus, cervix,[54] ovary, and testes. Metastasis occurs mostly by hematogenous route and sites include brain, lungs, pulmonary arteries,[55] stomach,[56,57] small intestine, and pancreas.[56] MRI again plays a very important role in diagnosis. MRI is preferred because of proper tissue resolution, and the use of magnetic field is safe and does not affect the fetus.[58]

Conclusion

Pregnancy involves physiological changes that can trigger peripheral neurological and/or CNS pathologies. Central nervous disorders are more complex, and a precise diagnosis must be made to improve perinatal outcomes, provide correct management and treatment, and prevent acute and long-term complications. It is possible to achieve a precise diagnosis, management, and treatment of neurological disorders during pregnancy, but these require a multidisciplinary approach, crucial to improve perinatal outcomes. MRI has proved to be a boon in timely diagnosis of these disorders, keeping in view its preference of use over other imaging modalities, due to least risk of ionization to the fetus.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Edlow JA, Caplan LR, O'Brien K, Tibbles CD. Diagnosis of acute neurological emergencies in pregnant and post-partum women. Lancet Neurol 2013;12:175-85.
2. James AH, Bushnell CD, Jamison MG, Myers ER. Incidence and risk factors for stroke in pregnancy and the puerperium. Obstet Gynecol 2005;106:509-16.

3. Von Wald T, Walling AD. Headache during pregnancy. Obstet Gynecol Surv 2002;57:179-85.
4. Goadsby PJ, Goldberg J, Silberstein SD. Migraine in pregnancy. BMJ 2008;336:1502-4.
5. Gupta S, Rohatgi A, Sharma SK, Gurtoo A. A study of neurological disorders during pregnancy and puerperium. Ann Indian Acad Neurol 2006;9:152-7.
6. Tremblay E, Thérasse E, Thomassin-Naggara I, Trop I. Quality initiatives: Guidelines for use of medical imaging during pregnancy and lactation. Radiographics 2012;32:897-911.
7. De Wilde JP, Rivers AW, Price DL. A review of the current use of magnetic resonance imaging in pregnancy and safety implications for the fetus. Prog Biophys Mol Biol 2005;87:335-53.
8. Hinchey J, Chaves C, Appignani B, Breen J, Pao L, Wang A, *et al*. A reversible posterior leukoencephalopathy syndrome. N Engl J Med 1996;334:494-500.
9. Burnett MM, Hess CP, Roberts JP, Bass NM, Douglas VC, Josephson SA. Presentation of reversible posterior leukoencephalopathy syndrome in patients on calcineurin inhibitors. Clin Neurol Neurosurg 2010;112:886-91.
10. Legriel S, Pico F, Azoulay E. Understanding posterior reversible encephalopathy syndrome. In: Vincent JL, editor. Annual Update in Intensive Care and Emergency Medicine. Berlin, Heidelberg: Springer; 2011. p. 631-53.
11. Lubarsky SL, Barton JR, Friedman SA, Nasreddine S, Ramadan MK, Sibai BM. Late postpartum eclampsia revisited. Obstet Gynecol 1994;83:502-5.
12. Ehtisham S, Hashmi HA. Posterior reversible encephalopathy syndrome. J Coll Physicians Surg Pak 2012;22:398-400.
13. Thackeray EM, Tielborg MC. Posterior reversible encephalopathy syndrome in a patient with severe preeclampsia. Anesth Analg 2007;105:184-6.
14. Stott VL, Hurrell MA, Anderson TJ. Reversible posterior leukoencephalopathy syndrome: A misnomer reviewed. Intern Med J 2005;35:83-90.
15. Vaughan CJ, Delanty N. Hypertensive emergencies. Lancet 2000;356:411-7.
16. Kastrup O, Maschke M, Wanke I, Diener HC. Posterior reversible encephalopathy syndrome due to severe hypercalcemia. J Neurol 2002;249:1563-6.
17. Ho CM, Chan KH. Posterior reversible encephalopathy syndrome with vasospasm in a postpartum woman after postdural puncture headache following spinal anesthesia. Anesth Analg 2007;105:770-2.
18. Eran A, Barak M. Posterior reversible encephalopathy syndrome after combined general and spinal anesthesia with intrathecal morphine. Anesth Analg 2009;108:609-12.
19. Servillo G, Apicella E, Striano P. Posterior reversible encephalopathy syndrome (PRES) in the parturient with preeclampsia after inadvertent dural puncture. Int J Obstet Anesth 2008;17:88-9.
20. Pedraza R, Marik PE, Varon J. Posterior reversible encephalopathy syndrome: A review. Crit Care Shock 2009;12:135-43.
21. Bousser MG, Ferro JM. Cerebral venous thrombosis: An update. Lancet Neurol 2007;6:162-70.
22. Liang CC, Chang SD, Lai SL, Hsieh CC, Chueh HY, Lee TH. Stroke complicating pregnancy and the puerperium. Eur J Neurol 2006;13:1256-60.
23. Cantu-Brito C, Arauz A, Aburto Y, Barinagarrementeria F, Ruiz-Sandoval JL, Baizabal-Carvallo JF. Cerebrovascular complications during pregnancy and postpartum: Clinical and prognosis observations in 240 Hispanic women. Eur J Neurol 2011;18:819-25.
24. Leys D, Cordonnier C. Cerebral venous thrombosis: Update on clinical manifestations, diagnosis and management. Ann Indian Acad Neurol 2008;11:S79-87.
25. Mokin M, Ciambella CC, Masud MW, Levy EI, Snyder KV, Siddiqui AH. Whole-brain computed tomographic perfusion imaging in acute cerebral venous sinus thrombosis. Interv Neurol 2016;4:104-12.
26. Rodallec MH, Krainik A, Feydy A, Hélias A, Colombani JM, Jullès MC, *et al.* Cerebral venous thrombosis and multidetector CT angiography: Tips and tricks. Radiographics 2006;26 Suppl 1:S5-18.
27. Shah AK, Whitty JE. Brain MRI in peripartum seizures: Usefulness of combined T2 and diffusion weighted MR imaging. J Neurol Sci 1999;166:122-5.
28. Zak IT, Dulai HS, Kish KK. Imaging of neurologic disorders associated with pregnancy and the postpartum period. Radiographics 2007;27:95-108.
29. Baird AE, Lövblad KO, Dashe JF, Connor A, Burzynski C, Schlaug G, *et al.* Clinical correlations of diffusion and perfusion lesion volumes in acute ischemic stroke. Cerebrovasc Dis 2000;10:441-8.
30. Roh JK, Kang DW, Lee SH, Yoon BW, Chang KH. Significance of acute multiple brain infarction on diffusion-weighted imaging. Stroke 2000;31:688-94.
31. Lowenstein DH, Bleck T, Macdonald RL. It's time to revise the definition of status epilepticus. Epilepsia1999;40:120-2.
32. Walker M. Status epilepticus: An evidence based guide. BMJ 2005;331:673-7.
33. Cherian A, Thomas SV. Status epilepticus. Ann Indian Acad Neurol 2009;12:140-53.
34. Kim JA, Chung JI, Yoon PH, Kim DI, Chung TS, Kim EJ, *et al.* Transient MR signal changes in patients with generalized tonicoclonic seizure or status epilepticus: Periictal diffusion-weighted imaging. AJNR Am J Neuroradiol 2001;22:1149-60.
35. Finelli PF. Diagnostic approach to restricted-diffusion patterns on MR imaging. Neurol Clin Pract 2012;2:287-93.
36. Katramados AM, Burdette D, Patel SC, Schultz LR, Gaddam S, Mitsias PD. Periictal diffusion abnormalities of the thalamus in partial status epilepticus. Epilepsia 2009;50:265-75.
37. Wakai S, Fukushima T, Teramoto A, Sano K. Pituitary apoplexy: Its incidence and clinical significance. J Neurosurg 1981;55:187-93.
38. Rajasekaran S, Vanderpump M, Baldeweg S, Drake W, Reddy N, Lanyon M, *et al*. UK guidelines for the management of pituitary apoplexy. Clin Endocrinol (Oxf). 2011 Jan;74(1):9-20.
39. de Heide LJ, van Tol KM, Doorenbos B. Pituitary apoplexy presenting during pregnancy. Neth J Med 2004;62:39-6.
40. Lamberts SW, Klijn JG, de Lange SA, Singh R, Stefanko SZ, Birkenha¨ger JC. The incidence of complications during pregnancy after treatment of hyperprolactinemia with bromocriptine in patients with radiologically evident pituitary tumors. Fertil Steril 1979;31:614-9.
41. Ohtsubo T, Asakura T, Kadota K, Takasaki K, Uchimura K, Makiuchi T, *et al.* A report of a transsphenoidal operation during pregnancy for a pituitary adenoma. No Shinkei Geka 1991;19:867-70.
42. Rosen SG, Kharlip J. Pituitary apoplexy during pregnancy. Endocr Rev 2011;32:P1-438.
43. Janssen NM, Dreyer K, van der Weiden RM. Management of pituitary tumour apoplexy with bromocriptine in pregnancy. JRSM Short Rep 2012;3:43.
44. Ayuk J, McGregor EJ, Mitchell RD, Gittoes NJ. Acute management of pituitary apoplexy – Surgery or conservative management? Clin Endocrinol (Oxf) 2004;61:747-52.
45. Lee JS, Park YS, Kwon JT, Nam TK, Lee TJ, Kim JK. Radiological apoplexy and its correlation with acute clinical presentation, angiogenesis and tumor microvascular density in pituitary adenomas. J Korean Neurosurg Soc 2011;50:281-7.
46. Karaka Z, Tanriverdi F, Unluhizarci K, Kelestimur E. Pregnancy and pituitary disorders. Eur J Endocrinol 2010;162:453-75.
47. Expert Panel on MR safety, Kanal E, Barkovich AJ, Bell C, Borgstede JP, Bradley WG Jr., *et al.* ACR guidance document on MR safe practice. J Magn Reson Imaging 2013;37:510-30.
48. Nawar RN, Abdel-Mannan D, Selma WR, Arafah BM. Pituitary tumor apoplexy: A review. J Intensive Care Med 2008;23:75-90.
49. Thomson AD, Marshall EJ. The natural history and pathophysiology of Wernicke's encephalopathy and Korsakoff's psychosis. Alcohol Alcohol 2006;41:151-8.
50. Hegde AN, Mohan S, Lath N, Lim CC. Differential diagnosis for bilateral abnormalities of the basal ganglia and thalamus. Radiographics 2011;31:5-30.
51. Thomson AD, Guerrini I, Marshall EJ. Wernicke's encephalopathy: Role of thiamine. In: Practical Gastroenterology. June 2009.
52. Sabatini JS, Schutz-Pereira GL, Feltrin F, Teive HA, Camargo CH. Wernicke's encephalopathy with chorea: Neuroimaging findings. Dement Neuropsychol 2016;10:370-2.

53. Yahata T, Kodama S, Kase H, Sekizuka N, Kurabayashi T, Aoki Y, *et al.* Primary choriocarcinoma of the uterine cervix: Clinical, MRI, and color doppler ultrasonographic study. Gynecol Oncol 1997;64:274-8.
54. Bazot M, Cortez A, Sananes S, Buy JN. Imaging of pure primary ovarian choriocarcinoma. AJR Am J Roentgenol 2004;182:1603-4.
55. Trübenbach J, Pereira PL, Huppert PE, Farnsworth C, Mayer R, Feine U, *et al.* Primary choriocarcinoma of the pulmonary artery mimicking pulmonary embolism. Br J Radiol 1997;70:843-5.
56. Coşkun M, Ağildere AM, Boyvat F, Tarhan C, Niron EA. Primary choriocarcinoma of the stomach and pancreas: CT findings. Eur Radiol 1998;8:1425-8.
57. Bateman HE, Kasimis BS, Yook CR, Hiremath V. Case report: Primary choriocarcinoma of the stomach. N J Med 1995;92:459-62.
58. Schwaighofer BW, Hesselink JR, Healy ME. MR demonstration of reversible brain abnormalities in eclampsia. J Comput Assist Tomogr 1989;13:310-2.