Radiology Case Reports 18 (2023) 844–849

Available online at www.sciencedirect.com

ScienceDirect

journal homepage: www.elsevier.com/locate/radcr

Case Report

Reversible cerebral vasoconstriction syndrome associated with probable drug poisoning ☆,☆☆

Arthur de Oliveira Veras, MD[a], *Felipe Micelli Noleto, MD*[b], *Michelle Zonkowski Ribas, MD*[c,*], *Gabriela Ferreira Paticcié, MD*[d], *Raphael Palomo Barreira, MD*[e], *Beatriz Medeiros Correa, MD*[e], *Thomas Zurga Markus Torres, MD*[e], *Vanessa Moraes Rossette, MD*[e], *Natalia Figueiredo Miranda, MD*[e], *Clara Kimie Miyahira, MD*[e], *Júlio César Claudino dos Santos, MSc*[f]

[a] *Hospital das Clínicas da Faculdade de Medicina de Ribeirão Preto - USP, Ribeirão Preto, SP, Brasil*
[b] *Faculdade de Medicina, Centro Universitário Christus, UNICHRISTUS, Fortaleza, CE, Brasil*
[c] *Universidade Federal do Paraná, UFPR, Rua XV de Novembro, 1299 – Centro, Curitiba, Paraná CEP 80060-000, Brasil*
[d] *Faculdade de Medicina, Universidade Federal de Juiz de Fora (UFJF), Juiz de Fora, MG, Brasil*
[e] *Serviço de Neurologia da Santa Casa de Santos, Santos, SP, Brasil*
[f] *Programa de Pós-Graduação em Ciências Morfofuncionais, Universidade Federal do Ceará - UFC, Fortaleza, CE, Brazil*

ARTICLE INFO

Article history:
Received 9 October 2022
Revised 14 November 2022
Accepted 16 November 2022

Keywords:
Reversible cerebral vasoconstriction syndrome
Headache
Cannabinoid
Marijuana

ABSTRACT

Reversible cerebral vasoconstriction syndrome (RCVS) is a clinical condition characterized by arterial involvement of the intracranial vessels, manifesting with vasospasm. The most common clinical manifestation related to the syndrome is the thunderclap headache, which consists of a severe headache that reaches the peak of pain within minutes. The imaging study assumes a leading role in the complementary investigation. Laboratory tests and cerebrospinal fluid analysis are often nonspecific and without significant diagnostic importance. Non-contrast studies of the brain parenchyma reveal variable results that can often be normal. Angiographic findings, which initially may not reveal any changes, allow the visualization of diffuse narrowing of the vessels, with the posterior cerebral circulation being preferentially affected. The present study reports the case of a 19-year-old woman with no relevant medical history, except that she was a regular user of marijuana and a drug based on chloroform and ether. The patient showed clinical and imaging signs compatible with RCVS, and the narcotics used by her were considered precipitating factors.

☆ Funding: The authors declare that no funds, grants, or other support were received during the preparation of this manuscript.
☆☆ Competing Interests: None.
* *Corresponding author.*
E-mail address: michelle.z.ribas@gmail.com (M.Z. Ribas).
https://doi.org/10.1016/j.radcr.2022.11.044

Introduction

Reversible cerebral vasoconstriction syndrome (RCVS), currently described as a synonym for Call-Fleming Syndrome, is a clinical-radiological condition representing a group of clinical entities with reversible vasoconstriction of brain vessels [1]. Clinically, it is characterized by the presence of thunderclap headaches, present in 95%-100% of patients [2], associated or not with other neurological signs and symptoms, such as epileptic seizures and focal deficits [3,4]. In neuroimaging, there is multifocal vasoconstriction of the cerebral arteries, with a resolution between 3 months, which denotes the characteristic reversibility of the condition [2,4–6].

It mainly affects individuals between 20 and 50 years of age [2,3], with a peak around 40 years of age and a higher incidence in females, reaching rates of 10 females for every male affected [1,3–5,7,8]. Regarding the pathophysiology, evidence suggests that the etiologic cause of cerebral vasoconstriction is a disturbance in the control of vascular tone, which can be triggered by endothelial dysfunction or hyperstimulation of the sympathetic autonomic nervous system. This mechanism has not yet been fully elucidated, but its origin is known to be multifactorial and associated with hormonal, immunological, and biochemical factors [9].

Studies indicate that about 60%-80% of patients have some secondary cause [3,7,10]. Several factors can act as precipitants of RCVS, such as puerperium, eclampsia, porphyria, use of antidepressants, immunosuppressants, and neurosurgical procedures [1,2,4,10]. In addition, drugs with vasoactive properties stand out, mainly marijuana, cocaine, and heroin [2,10]. Marijuana induces cerebral ischemia through vasospasm that is generated by increased oxidative stress in the release of reactive oxygen species and by the state of cellular adrenergic hyperstimulation. There is also a systemic reduction in the supply of oxygen secondary to the increase in carboxyhemoglobin levels after inhalation of the drug. Although the molecular pathophysiology is not well understood, studies point to a complex interaction between the active ingredients of marijuana, mainly delta-9-tetrahydrocannabinol, and the endocannabinoid and autonomic nervous systems, in addition to other mediating molecular pathways [11]. In addition, it is known that cannabis exerts a procoagulant effect through its interaction with CB1 and CB2 cannabinoid receptors on platelet cell membranes and, therefore, can trigger transient vasoconstriction in the peripheral vascular system [12,13].

In Brazil, another widely used narcotic is a clandestine mixture based on chloroform and ether, used only for recreational purposes. It is a drug snorted multiple times in a short time, given that it takes effect in seconds to minutes, with a maximum duration of 40 minutes. Its effects on the central nervous system involve 4 phases: arousal phase, with euphoria; depression, with mental confusion; deepening of depression, with reduced alertness, incoordination, and hallucinations; and, finally, late-onset depression, which can manifest with hypotension, lowered level of consciousness, epileptic seizures, and even coma. As it is a clandestine mixture, its complete composition is unknown, with few reports on its actual effects [14].

The diagnosis of RCVS is based on clinical features combined with neuroimaging findings [2]. It is a difficult diagnosis, given that thunderclap headache may be the only symptom in 76%-85% of patients, generating a broad spectrum of differential diagnoses, such as subarachnoid hemorrhage, cerebral venous thrombosis, primary angiitis of the central nervous system, posterior reversible encephalopathy syndrome, among others [3,10]. Thus, neuroimaging becomes essential, in which the presence of recurrent thunderclap headaches with normal cranial computed tomography (CT) and magnetic resonance imaging (MRI) has a high positive predictive value for the possibility of RCVS [4]. Angiography is the gold standard method, but due to the degree of invasion and risk of complications inherent to the method [2,3,7], it is usually replaced by magnetic resonance angiography or CT angiography [3], which are less invasive and more available tests, but which may not detect abnormalities when performed early [2].

Given the importance of the diagnosis of RCVS and the numerous triggering factors described, we present the case of a 19-year-old female patient, with a clinical picture suggestive of RCVS attributable to the use of marijuana and narcotic based on chloroform and ether. In addition, we highlight the importance of radiological findings for diagnosis, as demonstrated by the images in this report.

Case description

In 2019, a 19-year-old female with no relevant medical history, except for being a regular user of marijuana and a narcotic based on chloroform and ether, reported an onset of an acute holocranial headache a week earlier. She reported an insidious clinical course with recurrent and intense episodes, which evolved with behavioral changes and a lowering of the level of consciousness. She was found to be unresponsive and admitted to the hospital with a generalized tonic-clonic epileptic seizure. Neurological examination on admission found the patient ventilated by invasive mechanical ventilation through an endotracheal tube, under intravenous sedoanalgesia, with a Richmond Agitation Sedation Scale of -2. Trunk reflexes were hypoactive. General laboratory tests were performed, which did not show any changes. Non-contrast CT of the skull was performed, which revealed hyperdense foci in the topography of the splenium of the corpus callosum and the cingulate gyrus, with no other findings. Diagnostic cerebral arteriography was abnormal, with diffuse narrowing of intracranial arteries, more evident in the P1 and P2 segments of the posterior cerebral arteries (Fig. 1). The diagnostic hypothesis was Reversible Cerebral Vasoconstriction Syndrome. After 15 days, a neurological improvement was observed, and a new angiographic study documented the reversal of arterial narrowing.

Discussion

The diagnosis of RCVS is related to multifactorial aspects, which range from a detailed anamnesis of the patient to high-

Table 1 – Imaging tests chosen for diagnostic investigation and respective imaging findings found in the present case and 22 others reported in the scientific literature in the last 10 years concerning patients with RCVS.

Authors and year	Imaging diagnostic method	Imaging findings
Present case	CT; Cerebral arteriography	CT: hyperdense focus on the topography of the splenium of the corpus callosum and the cingulate gyrus; Arteriography: presence of diffuse narrowing of intracranial arteries, being more evident in the P1 and P2 segments of the posterior cerebral arteries.
Yancy et al., 2013	MR angiography	MR angiography: multifocal areas narrowing bilaterally involving the middle cerebral, anterior cerebral, and posterior cerebral arteries.
Abkur et al., 2014	MR angiography; CT	MR angiography: focal narrowing in the intracerebral arteries. CT: Normal.
Sheikh et al., 2014	MR angiography; CT	CT: Normal; MR angiography: diffuse segmental stenosis.
Bernard et al., 2015	MR angiography; CT	MR angiography: multiple areas of segmental narrowing of the anterior cerebral vasculature without evidence of infarction or hemorrhage; CT: Normal
Sorensen et al., 2015	MR; CT;	MR: bilateral cortical and subcortical edema
Kulkarni et al., 2015	MR angiography; MR	MR: multiple acute infarctions bilaterally in the territory between the anterior and middle cerebral arteries; MR angiography: diffuse vasospasm of the A1 segment of the territory of the bilateral anterior cerebral artery and branches of the bilateral middle cerebral arteries.
Roongpiboonsopit et al., 2016 (1)	MR angiography; CT	CT: subarachnoid hemorrhage in the right superior frontal sulcus; MR angiography: multifocal segmental narrowing of both middle cerebral arteries, left anterior cerebral artery, and right posterior cerebral artery.
Roongpiboonsopit et al., 2016 (2)	MR angiography; CT	CT: subarachnoid hemorrhage in the left frontal lobe; MR: subarachnoid hemorrhage in the left frontal lobe; MR angiography: multiple vascular irregularities in middle and posterior cerebral arteries and superior cerebellar artery branches.
Roongpiboonsopit et al., 2016 (3)	MR angiography; CT	CT: intraparenchiamatous hemorrhage in the right frontal lobe; MR angiography: diffuse irregularity, with moderate-severe multifocal stenoses of the intracranial arteries.
Perdices et al., 2016	MR angiography; CT	CT: Normal; MR Angiography: irregular narrowing of the anterior and middle cerebral branches.
Manning et al., 2021	MR angiography	MR angiography: diffuse concentric narrowing of cerebral vessels, in addition to the typical "beading" sign.
Lee et al., 2013	MR angiography	MR angiography: narrowing of the left posterior communicating artery segment.
Cholet et al., 2018	MR angiography	MR angiography: diffuse arterial spasms mainly affecting the posterior cerebral arteries. The anterior arteries and especially the middle cerebral arteries are also involved.
Laeeq et al., 2019	MR angiography	MR angiography: diffuse vasoconstriction
Howart et al., 2020	MR angiography	MR angiography: irregular caliber of the left anterior and middle proximal cerebral arteries, mainly in the A2 and M1 segments.
Bouvy et al., 2020	MR angiography	MR angiography: stenosis of the A1 segment of the right anterior cerebral artery and the M2 segment of the right middle cerebral artery.
Hiashy et al., 2021	MR	MR: edema in the basal nuclei and right occipital lobe.
Mikami et al., 2021	CT; MR angiography	CT: hypodensity in bilateral semi-oval centers, with greater involvement on the left; MR angiography: diffuse narrowing of cerebral arteries, especially anterior cerebral arteries, without occlusions.
Zeitoune et al., 2021	MR; CT angiography;	MR: small infarcts in border zones of posterior and middle cerebral arteries on the right; CT angiography: multiple areas of moderate anterior and posterior circulation narrowing.
Mansoor et al., 2021	MR; CT angiography;	MR: bilateral gyral hypersignal of asymmetric pattern, predominantly in the parieto-occipital and frontal regions, on T2; CT angiography: basilar artery narrowing.
Bliss et al., 2021	MR; CT angiography;	MR: diffuse areas of leptomeningeal abnormality in both cerebral hemispheres; CT angiography: narrowing of the basilar artery and right vertebral artery and irregularities in segments A1, M1, and P1.
Lin et al., 2021	MR; MR angiography	MR: bilateral cytotoxic edema in parietal and frontal lobes; MR angiography: multiple areas of stenosis and partial occlusions involving the anterior, middle, and posterior cerebral arteries bilaterally.

CT, computed tomography; MR, magnetic resonance.

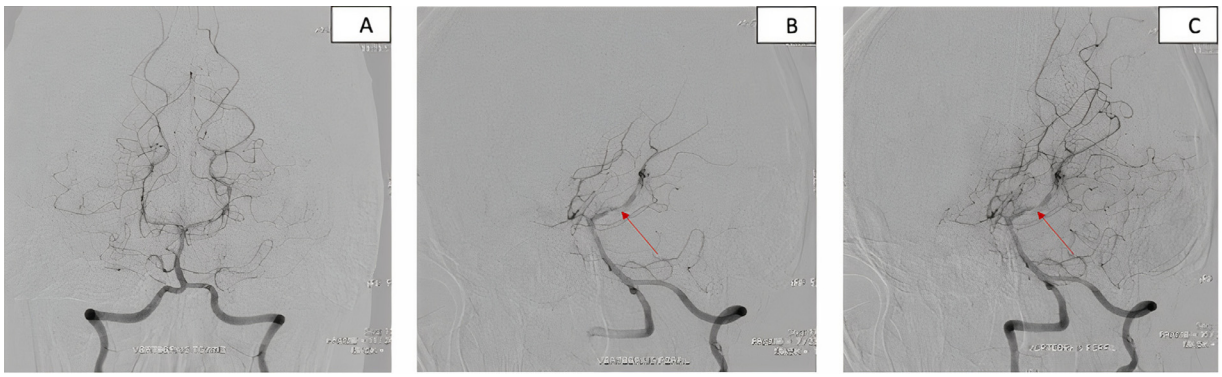

Fig. 1 – Diagnostic arteriography of vertebrobasilar circulation in frontal (A), left posterior oblique early phase (B), and left posterior oblique late phase (C) views revealed diffuse narrowing of intracranial arteries, being more evident in P1 and P2 segments of posterior cerebral arteries.

quality imaging tests. The medical literature demonstrates that factors such as chronic medications, use of narcotics, and pre-existing comorbidities are coadjuvants in the RCVS [15]. Regarding the medical history before the syndrome diagnosis, chronic migraine and psychiatric diseases were considered more prevalent [9]. Depression, anxiety, reflux, obesity, diabetes mellitus, and postpartum changes appear to be in fewer numbers and, consequently, in a lower level of interaction with RCVS [16].

A cohort study published in 2018 regarding the epidemiological aspects of RCVS found that 60% of the patients had precipitating factors, of which 33% were related to marijuana use. In a second analysis, marijuana was the identifiable narcotic with the highest prevalence of cases in patients with RCVS. However, although it is a known predisposing factor for the syndrome, studies examining this relationship are lacking [17].

Marijuana for recreational use has been the subject of extensive political debate and has already been approved in several countries. Unfortunately, due to this broad legalization, a false impression was created, on the part of many users, that the use is safe. In this regard, the population should know the association between marijuana use and RCVS. Although most patients with the syndrome fully recover, some may present permanent deficits and even die [17,18]. In the present case, the patient used marijuana recreationally and deliberately, and its use was related to the diagnosis of RCVS.

Regarding diagnosis, RCVS is considered underdiagnosed and should be more considered in interpreting clinical-imaginological findings in health services. The main characteristic observed in the clinical profile of patients with RCVS is a sudden-onset headache and, more specifically, a thunderclap sensation often described as large-scale pain [19]. Cases with specific alterations may also be related to the syndrome under discussion. Some of these changes, such as executive dysfunction, nausea associated with vomiting, changes in sensitivity, disorientation, and behavioral changes, appear to recur in RCVS cases. The clinical and neurological examinations of the case reported in the present study demonstrated a relationship with RCVS. The patient in question presented with recurrent thunderclap headaches, a presentation with high specificity for diagnosing RCVS [20]. In addition, she evolved with other symptoms such as epileptic seizures, behavioral changes, and lowering of the level of consciousness that, as seen, can occur in the context of the on-screen diagnosis.

As for the diagnostic investigation, brain parenchyma exams, such as CT and MRI of the skull, and the analysis of the vascular structure through CT Angio, AngioMRI, and Angiography, occupy a prominent position in the approach to the patient with suspected RCVS [19]. Regarding the evaluation of the brain parenchyma, when compared to other arteriopathies, the RCVS shows greater normality in the results of the exams and, when the investigation reveals any alteration, presents fewer cerebral infarctions [20,21]. Rocha et al. (2019) comparatively studied patients with arteriopathies and found that those who did not have RCVS accumulated more lesions in a serial investigation. Subarachnoid hemorrhage, in turn, was more frequently found in patients with RCVS, and non-aneurysmal convexity SAH was found exclusively in patients with RCVS [20].

As for the study of vessels, in an initial moment, the angiographic investigation can be normal in up to 33% in the first week, considering the natural history of the syndrome [22]. The smaller and more distal arteries are usually affected first, and this alteration can go unnoticed in an initial evaluation [21]. More proximal arteries are affected by the evolution of vasoconstriction, altering the imaging evaluation [22,23]. The findings typically include an alternation between narrowing and dilating the affected vessels due to the vasoconstriction inherent to the syndrome [24,25]. Regarding the vascular territory affected, it appears that the involvement of the intracranial segment of the internal carotid artery (ICA) is usually preserved in patients with RCVS and is more frequently affected in other arteriopathies, such as moyamoya disease and atherosclerotic disease. In turn, the involvement of the posterior arterial circulation and the pattern of diffuse involvement is more related in patients with RCVS [20].

A comparative analysis was performed between the cases exposed in the present study with 22 others reported in the

scientific literature in the last 10 years referring to patients diagnosed with RCVS. Graphs 1 and 2 bring together relevant epidemiological characteristics regarding the prevalence distribution by sex of RCVS considering different age groups and the reported precipitating factors. Table 1, in turn, shows which imaging technique was used for diagnostic investigation in each of these cases and the imaging findings obtained [2–5,7,9,10,13,15,16,18,19,24,26–32].

Considering the information presented in Table 1, one can see the wide variety of findings that can be found in patients with RCVS undergoing investigation with non-contrast imaging tests, which may often prove to be normal or, when altered, be less frequent, the finding of brain parenchymal infarction. In addition, when analyzing the data presented in Table 1, it is possible to observe the diffuse pattern of arterial involvement typical of RCVS. Such data follow the scientific literature, as previously explained [20–25].

It is also worth noting the complete absence of reports of involvement of the intracranial segment of the ICA in all 23 cases presented in the table. This is a relevant factor to be considered in the differential diagnosis between RCVS and other medical conditions that may have a similar clinical presentation. In addition to being in line with the medical literature, such information is of singular importance when considering the RCVS2 diagnostic score. From the total of 10 points used to calculate the probability of the RCVS diagnosis, in which the higher the score, the greater the probability of the diagnosis of the syndrome, removes a value of 2 points when there is the involvement of the intracranial segment of the ICA [20].

Conclusion

In the present report, we describe a rare case of RCVS associated with the use of illicit drugs: marijuana and a narcotic based on chloroform and ether. The association between RCVS and marijuana is described in the literature. However, more studies are needed to investigate the association between RCVS and chloroform and ether-based narcotics since no other study in the literature besides the present report has demonstrated this association. As for the diagnosis, it is essential to know the variety of clinical and imaging patterns of RCVS to recognize a suspected patient quickly and exclude differential diagnoses.

Patient consent

Written informed consent for the publication of this case report was obtained from the patient.

REFERENCES

[1] Song TJ, Lee KH, Li H, Kim JY, Chang K, Kim SH, et al. Reversible cerebral vasoconstriction syndrome: a comprehensive systematic review. Eur Rev Med Pharmacol Sci 2021;25(9):3519–29.
[2] Sheikh HU, Mathew PG. Reversible cerebral vasoconstriction syndrome: updates and new perspectives. Curr Pain Headache Rep 2014;18(5):414.
[3] Abkur TM, Saeed M, Alfaki NO, O'Connor M. Idiopathic reversible cerebral vasoconstriction syndrome (RCVS). BMJ Case Rep 2014;2014:bcr2014206913.
[4] Kulkarni M, Chauhan V, Shetty S. Reversible cerebral vasoconstriction syndrome. J Assoc Physicians India 2016;64(6):76–8.
[5] Perdices M, Herkes G. Reversible cerebral vasoconstriction syndrome. Neuropsychol Rehabil 2018;28(2):223–33.
[6] Patel SD, Topiwala K, Saini V, Patel N, Pervez M, Al-Mufti F, et al. Hemorrhagic reversible cerebral vasoconstriction syndrome: a retrospective observational study. J Neurol 2021;268(2):632–9.
[7] Yancy H, Lee-Iannotti JK, Schwedt TJ, Dodick DW. Reversible cerebral vasoconstriction syndrome. Headache 2013;53(3):570–6.
[8] Patel SD, Topiwala K, Otite Oliver F, Saber H, Panza G, Mui G, et al. Outcomes among patients with reversible cerebral vasoconstriction syndrome: a nationwide United States analysis. Stroke 2021;52(12):3970–7.
[9] Manning T, Bartow C, Dunlap M, Kiehl R, Kneale H, Walker A. Reversible cerebral vasoconstriction syndrome associated with fluoxetine. J Acad Consult Liaison Psychiatry 2021;62(6):634–44.
[10] Bernard KR, Rivera M. Reversible cerebral vasoconstriction syndrome. J Emerg Med 2015;49(1):26–31.
[11] Singh A, Saluja S, Kumar A, Agrawal S, Thind M, Nanda S, et al. Cardiovascular complications of marijuana and related substances: a review. Cardiol Ther 2018;7(1):45–59.
[12] Goyal H, Awad HH, Ghali JK. Role of cannabis in cardiovascular disorders. J Thorac Dis 2017;9(7):2079–92.
[13] Bliss L, McGowan B, Rahman A. A rare pediatric case of marijuana-induced reversible cerebral vasoconstriction syndrome. Pediatr Neurol 2021;120:33–5.
[14] Centro Brasileiro de Informações sobre Drogas. Livreto informativo sobre drogas psicotrópicas. Livreto informativo sobre drogas psicotrópicas; 2007. Unifesp. São Paulo.14–16.
[15] Cholet C, Dormont D, Law-Ye B. MR angiography of reversible cerebral vasoconstriction syndrome. Diagn Interv Imaging 2018;99(9):525–6.
[16] Bouvy C, Ackermans N, Maldonado Slootjes S, Rutgers MP, Gille M. Reversible cerebral vasoconstriction syndrome revealed by fronto-callosal infarctions. Acta Neurol Belg 2020;120(6):1467–9.
[17] Jensen J, Leonard J, Salottolo K, McCarthy K, Wagner J, Bar-Or D. The epidemiology of reversible cerebral vasoconstriction syndrome in patients at a Colorado Comprehensive Stroke Center. J Vasc Interv Neurol 2018;10(1):32–8.
[18] Mikami T, Obata R, Steinberg DI, Skliut M, Boniece I. Marijuana-related reversible cerebral vasoconstriction syndrome. Intern Med 2021;60(5):795–8.
[19] Hayashi R, Hayashi S, Machida S. Ophthalmological symptoms in a patient with reversible cerebral vasoconstriction syndrome: a case report. J Med Case Rep 2021;15(1):175.
[20] Rocha EA, Topcuoglu MA, Silva GS, Singhal AB. RCVS2 score and diagnostic approach for reversible cerebral vasoconstriction syndrome. Neurology 2019;92(7):e639–47.
[21] Ducros A, Bousser MG. Reversible cerebral vasoconstriction syndrome. Pract Neurol 2009;9:256–67.
[22] Ducros A, Fiedler U, Porcher R, Boukobza M, Stapf C, Bousser MG. Hemorrhagic manifestations of reversible cerebral vasoconstriction syndrome: frequency, features, and risk factors. Stroke 2010;41:2505–11.

[23] Ducros A, Boukobza M, Porcher R, Sarov M, Valade D, Bousser MG. The clinical and radiological spectrum of reversible cerebral vasoconstriction syndrome. A prospective series of 67 patients. Brain 2007;130:3091–101.
[24] Roongpiboonsopit D, Kongbunkiat K, Phanthumchinda K. Reversible cerebral vasoconstriction syndrome: a report on three cases. J Med Assoc Thai 2016;99(1):97–105.
[25] Kushkuley J, Feroze AH, Choudhri OA. Diffuse intracranial and extracranial vascular involvement in reversible cerebral vasoconstriction syndrome. Clin Neuroradiol 2015;25:301–4.
[26] Sorensen DM. Reversible cerebral vasoconstriction syndrome. JAMA Neurol 2016;73(2):232–3.
[27] Lee R, Ramadan H, Bamford J. Reversible cerebral vasoconstriction syndrome. J R Coll Physicians Edinb 2013;43(3):225–8.
[28] Laeeq R, Berman JS, Khalid U, Lakkis NM, Tabbaa R. Reversible cerebral vasoconstriction syndrome associated with coronary artery vasospasm. Tex Heart Inst J 2019;46(2):139–42.
[29] Howarth H, Mandal AKJ, Boyd E, Missouris CG. Reversible cerebral vasoconstriction syndrome: perhaps not so reversible? Am J Med 2020;133(8):928–9.
[30] Zeitouni D, Parish JM, Smith M, Stetler WR, Bernard JD. Reversible cerebral vasoconstriction syndrome successfully treated by intrathecal nicardipine. Clin Neurol Neurosurg 2021;206:106705.
[31] Mansoor T, Alsarah AA, Mousavi H, Khader Eliyas J, Girotra T, Hussein O. COVID-19 associated reversible cerebral vasoconstriction syndrome successfully treated with nimodipine and aspirin. J Stroke Cerebrovasc Dis 2021;30(7):105822.
[32] Lin B, Wang C, Lu N, Zhang L, Jiang B. Reversible cerebral vasoconstriction syndrome with cerebral infarction caused by acute high-level vapor exposure of ethylene oxide: a case report. BMC Neurol 2021;21(1):391.