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Case Report

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A Case Report of Cerebral Venous Sinus Thrombosis in Palestine: The Importance of Rapid Diagnosis and Treatment of CVST

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Abstract

Cerebral venous sinus thrombosis (CVST) is an often under-diagnosed, life-threatening condition. We report the first case of CVST in Palestine of a 39-year-old female who presented with a history of loss of consciousness preceded by severe vertigo and headache. Brain CT scan without contrast was done, in which no abnormality was detected. Further Brain MRI and MRV studies with contrast showed thrombosis of left transverse sinus with extension to left sigmoid sinus and left jugular vein. The patient was anticoagulated and admitted to the ICU for regular monitoring and frequent brain CT scans to rule out hemorrhagic transformation. The patient made a full recovery. Lab and molecular studies were carried out as an outpatient to investigate the etiology of this presentation. The aim of this case report is to demonstrate the importance of early detection and treatment of CVST for a successful outcome.

Keywords: Anticoagulants, Cerebral Venous Sinus, Vertigo, Thrombosis

Abbreviations

CT: Computed Tomography
CVST: Cerebral Venous Sins Thrombosis
GCS: Glasgow Coma Scale
ICP: Intra Cranial Pressure
JAK2: Janus Kinase2
LMWH: Low Molecular Weight Heparin
MRI: Magnetic Resonance Imaging
MRV: Magnetic Resonance Venogram
VTE: Venous Thrombo Embolism
T2W: T2 Weighted

Introduction

CVST is a rare form of venous embolism which occurs in the cerebral venous dural sinuses, or cortical veins. Increased intracranial pressure, toxic edema, seizures and possibly death can occur [1]. It represents just 0.5-1% of all brain strokes, and its outcome worsens when there is a delay in diagnosis or in the initiation of therapy [2-6]. The annual incidence of CVST is increasing due to the wider availability of imaging - ranging from 0.22 to 1.57 per 100,000 [7-9].

CVST is a multifactorial disease and its occurrence depends on the presence of multiple risk factors. Studies report 65% of patients with CVST has more than 1 risk factor [3, 4]. Approximately 75% of adult patients are women, likely due to use of oral contraceptives, pregnancy, and the peripartum period [2-6]. The main predisposing factors are genetic, acquired prothrombotic state and infection [6-10].

Table 1: Risk factors of CVST

Acquired	Inherited
Brain tumor	Protein C and S deficiency
Head trauma	Anti-thrombin III deficiency
Local head infection	Homocysteinemia
Intracranial hypertension	Factor V leiden homozygousmutation
Internal jugular vein abnormality	G20210A prothrombin gene
Neurological disorder	Methylene-Tetra-hydro-folate-reductase 677TT mutation
Lumber Puncture	Positive anti-cardiolipin or anti-phospholipid antibodies
Pregnancy	
Puerperium	
Hematological condition	
Nephrotic syndrome	
Systemic vasculitis	
Medication (Cisplatin, Methotrexate, steroids)	

The diagnosis of CVST is challenging because of variety in presentation. It rarely presents as a stroke syndrome [10]. In neonates it often appears as nonspecific neurologic signs and symptoms [11]. Young adults usually complain of headache with up to 80-90% of the first presentation being either diffuse, focal or migraine-type headaches [12-14, 4-6]. The most frequent presentations are isolated headaches, intracranial hypertension syndrome, seizures, a focal lobar syndrome, and encephalopathy [7, 15-17].

Greater availability of neuroimaging has helped physicians make more accurate diagnosis despite variability in CVST presentation. The current choice of investigation for CVST is MRI venography (MRV) and CT venography [18-21]. The cornerstone of management is dependent on recognition of the underlying cause, correction of modifiable risk factors, preventing complications, and decreasing the recurrence rate [4, 6]. Anticoagulation, typically using low molecular weight heparin (LMWH), is the first line treatment in a stable patient. In resistant cases aggressive management may necessitate local intravenous thrombolysis, mechanical thrombectomy, and decompressive hemicraniectomy [3, 4].

The prognosis of CVST has gradually improved since the 1960s [15]. During the acute phase of presentation, mortality rate is 5% [16, 17]. The International Study of CVT found 79% of patients' post-follow up had made a complete recovery, while mortality rate was 8%.

Case Presentation

A 39-year-old Palestinian lady was admitted by ambulance to Al-Wattani Hospital, Nablus at midday following an episode of loss of consciousness at home. The previous morning, she suffered from severe vertigo followed by vomiting, alongside a three-day history of a constant, progressively worsening headache not responding to painkillers. The patient had a history of known granulomatous mastitis requiring regular drainage due to recurrent abscesses, migraines from childhood, and past miscarriages. She was not on an oral contraceptive pill.

On examination vital signs were normal and she had a Glasgow coma scale (GCS) of 13/15. The patient had 4/5 MRC power in all limbs. Her pupils, fundi, and speech appeared normal. There was no evidence of any focal neurological deficit or meningism. A non-contrast brain CT carried out within an hour of admission detected no abnormality. An abdominal ultrasound and ECHO were normal.

The patient was admitted to hospital for further investigation and treatment. Complete blood count (CBC) indicated polycythemia (Hb 17.4 g/dl), thrombocytosis (platelet count 811×10^9/L), and leukocytosis (WBC 18.3×10^9/L). A treatment dose of LMWH was administered, alongside aspirin. The patient was reviewed by both hematology and rheumatology teams, and subsequently admitted to the ICU for close monitoring. She underwent two courses of venesection to treat the polycythemia. Hydroxyurea was administered to treat thrombocythemia. An immune antibody screen including anti phospholipid syndrome was negative.

Magnetic resonance venography (MRV) confirmed the presence of a thrombosis in the left transverse sinus with extension to the left sigmoid sinus and left internal jugular vein. A CT Brain demonstrated a thrombus within the anterior part of superior sagittal sinus and the left transverse sinus, and CT chest showed a small thrombus within the aortic arch. The patient's clinical condition improved during her nine day stay in ICU, receiving ongoing anticoagulation. She was discharged from hospital thirteen days post-admission with a complete resolution of symptoms. Bone marrow biopsy results indicated essential thrombocythemia, and molecular studies confirmed the patient has JAK2 mutation.

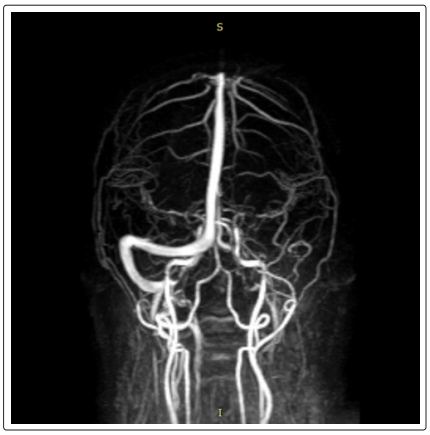

Figure 1: Coronal Magnetic Resonance Venography Shows Filling Defect In The Left Transverse And Sigmoid Sinuses Extended To Left Internal Jugular Represents Venous Thrombosis

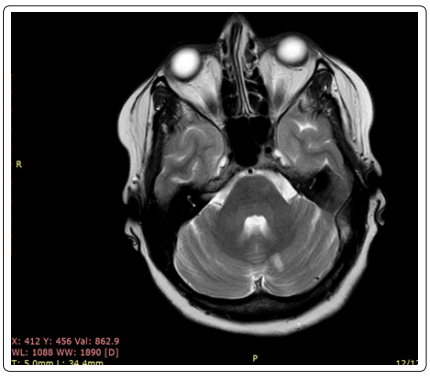

Figure 2: T2W_TSE_AX1/MRI: Left Cerebellar Ischemia Shown On As Hyperintense Area

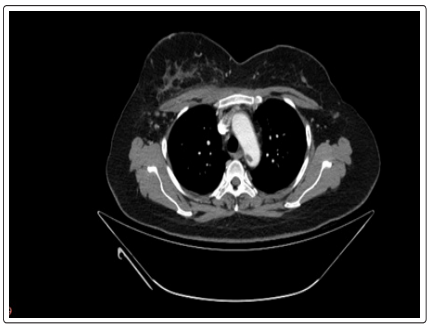

Figure 3: Chest and neck CT with Contrast Shows Small Thrombus on Arch of Aorta

Discussion

This was the first reported case of CVST in Palestine. There is currently no official data regarding the incidence of CVST in the developing world. Worldwide incidence ranges from 0.22 to 1.57 per 100,000 [7-9]. This patient presented within the classic demographic group for CVST, with women known to be at three times greater risk than men, and 78% of all cases affecting those aged below 50 years old as reported in the international study on CVST [14]. It differs from typical strokes, affecting a much younger population.

One study found the median age of presentation of CVST to be 37 years old [21-23].

Predisposing risk factors are present in 80% of patients diagnosed with CVST [24]. There is often more than one risk factor present, as in our case. The patient was JAK2 mutation positive, and had known granulomatous mastitis. The patient suffered from raised hemoglobin, platelet count, and raised hematocrit, which can be explained by the presence of JAK2 mutation confirmed with molecular studies. JAK2 mutation can cause essential thrombocythemia, polycythemia rubra Vera, myelofibrosis, as well as other myeloproliferative disorders. CVST was the first presentation of this patient's myeloproliferative disorder. The internal jugular vein receives cerebral venous blood through the dural sinuses. The sagittal sinus is more susceptible to thrombus formation than other areas of the cerebral venous sinuses, with 62% of cases reported to occur within it [10]. However, in this case the thrombus was found in the left transverse sinus with extension to the left sigmoid sinus and left jugular vein.

The wide variation in anatomy of the brain's venous system has resulted in an incomplete understanding of CVST pathogenesis [25]. The clinical presentation of CVST can occur due to different mechanisms. Cerebral venous occlusion causes infarction, and edema within the brain. Venous occlusion also leads to raised intracranial pressure (ICP), with cerebrospinal fluid collecting within the superior sagittal sinus unable to drain to the left jugular vein [10]. In this case there was evidence of left cerebellar ischemia on MRI, but no indication of ICP.

The patient underwent a series of urgent investigations to determine the cause of her presentation. A third of CVST cases present with intracranial hemorrhage seen on imaging [12]. As the initial CT Brain without contrast ruled out ICH, prophylactic LMWH was promptly administered in light of the patient's hypercoagulable clinical picture. MRI Brain with contrast showed left transverse sinus thrombosis with left cerebellar ischemic insult. MRV confirmed the presence of thrombosis of the left transverse sinus with extension to the left sigmoid sinus and left internal jugular vein. MRV is the preferred and most sensitive diagnostic investigation for CVST, which allows venous occlusion to be identified along with any consequences such as cerebral edema and areas of venous infarction [26]. However, Bousser and Ferro suggested that MRV should be conducted in conjunction with MRI, as it cannot alone distinguish thrombosis from hypoplasia [3].

A whole-body CT scan was conducted to investigate for further thrombus formation, and underlying malignancy. A small thrombus was seen in the aortic arch. Frequent repeat CT Brain imaging was conducted to monitor for possible transformative hemorrhage and ischemic changes. Initial management of patients with confirmed CVST should include stabilization and the prevention of any cerebral herniation [5]. Despite the risk of hemorrhage into venous infarcts, anticoagulation forms the mainstay of treatment [27]. In this case standard hospital protocol suggests use of prophylactic LMWH following a VTE risk assessment on all inpatients. However, given the clinical indication of a hypercoagulable state, a therapeutic dose of LMWH was administered. It is likely the patient benefited from this early anticoagulation, before a formal diagnosis of CVST was made.

A prospective study demonstrated that 79% of patients recovered following treatment with LMWH [23]. Our patient showed marked clinical improvement in the days following the commencement of anticoagulation. The patient was eventually switched from LMWH to warfarin to prevent the risk of long term CVST recurrence. Recurrence rates are estimated to be between 2-4%. Long term anticoagulation also reduces risk of extra cerebral venous thrombosis, which is seen in 4-7% of patients with following CVST [23, 28-30].

The patient was prescribed hydroxyurea during admission as a myeloproliferative disease was suspected given her clinical presentation [31]. This treatment was continued as hydroxyurea is the only agent which has been shown to reduce the rates of thrombosis in ET when compared to other choices of treatment [32-69]. In cases where prognosis appears poor, endovascular thrombolysis, with agents such as urokinase and tissue plasminogen activator, may be used [10]. Other treatment options include the use of therapeutic lumbar punctures, oral acetazolamide and surgery in treating intracranial hypertension which were not required in our case.

Conclusion

CVST is an uncommon condition and diagnosis is challenging due to its variable presentation. Doctors should maintain a high index of suspicion for CVST, especially in young or female adults presenting with headaches, refractory to other treatment. CVST may be the first presentation for some patients with JAK2 mutation. Neuroimaging is integral to providing a rapid diagnosis with MRI and MRV the most effective choices of investigation. Early administration of anticoagulation is the mainstay of treatment to aid recovery and prevent complications. Long term anticoagulation is important to prevent recurrence, reducing mortality, and morbidity.

References

1. Ameri A, MG Bousser (1992) Cerebral venous thrombosis. Neurologic Clinics 10: 87-111.
2. Beer-Furlan A, de Almeida CC, Noleto G, Paiva W, Ferreira AA, et al. (2013) Dural sinus and internal jugular vein thrombosis complicating a blunt head injury in a pediatric patient. Child's Nervous System 29: 1231-1234.
3. Bousser MG, JM Ferro (2007) Cerebral venous thrombosis: an update. The Lancet Neurology 6: 162-170.
4. Bentley JN, RE Figueroa, JR Vender (2009) From presentation to follow-up: diagnosis and treatment of cerebral venous thrombosis. Neurosurgical focus 27: E4.
5. Crassard I, MG Bousser (2004) Cerebral venous thrombosis. J Neuroophthalmol 24: 156-163.
6. Filippidis A, Kapsalaki E, Patramani G, Fountas KN (2009) Cerebral venous sinus thrombosis: review of the demographics, pathophysiology, current diagnosis, and treatment. Neurosurgical focus 27: E3.
7. Ferro JM, Correia M, Pontes C, Baptista MV, Pita F (2001) Cerebral vein and dural sinus thrombosis in Portugal:1980-1998. Cerebrovasc Dis 11: 177-182.
8. Coutinho JM, Zuurbier SM, Aramideh M, Stam J (2012) The incidence of cerebral venous thrombosis: a cross-sectional study. Stroke 43: 3375-3377.
9. Devasagayam S, Wyatt B, Leyden J, Kleinig T (2016) Cerebral Venous Sinus Thrombosis Incidence Is Higher Than Previously Thought: A Retrospective Population-Based Study. Stroke 47: 2180-2182.
10. Stam J (2005) Thrombosis of the cerebral veins and sinuses. New England Journal of Medicine 352: 1791-1798.
11. DeVeber G, Andrew M, Adams C, Bjornson B, Booth F, et al.

(2001) Cerebral sinovenous thrombosis in children. N Engl J Med 345: 417-423.
12. Pongmoragot J, Saposnik G (2012) Intracerebral Hemorrhage from Cerebral Venous Thrombosis. Current atherosclerosis reports 14: 382-389.
13. De Bruijn SF, J Stam, LJ Kappelle (1996) Thunderclap headache as first symptom of cerebral venous sinus thrombosis. CVST Study Group. Lancet 348: 1623-1625.
14. Saposnik G, Barinagarrementeria F, Brown RD Jr, Bushnell CD, Cucchiara B, et al. (2011) Diagnosis and management of cerebral venous thrombosis: a statement for healthcare professionals from the American Heart Association/American Stroke Association. Stroke 42: 1158-1192.
15. Canhão P, Ferro JM, Lindgren AG, Bousser MG, Stam J, et al. (2005) Causes and predictors of death in cerebral venous thrombosis. Stroke 36: 1720-1725.
16. Borhani Haghighi A, Edgell RC, Cruz-Flores S, Feen E, Piriyawat P, et al. (2012) Mortality of cerebral venous-sinus thrombosis in a large national sample. Stroke 43: 262-264.
17. Coutinho JM, Zuurbier SM, Stam J (2014) Declining mortality in cerebral venous thrombosis: asystematic review. Stroke 45: 1338-1341.
18. Sébire G, Tabarki B, Saunders DE (2005) Cerebral venous sinus thrombosis in children: risk factors, presentation, diagnosis and outcome. Brain 128: 477-489.
19. Barnes C, Newall F, Furmedge J (2004) Cerebral sinus venous thrombosis in children. J Paediatr Child Health 40: 53-55.
20. Teksam M, Moharir M, deVeber G (2008) Frequency and topographic distribution of brain lesions in pediatric cerebral venous thrombosis. AJNR Am J Neuroradiol 29: 1961-1965.
21. Medlock MD, Olivero WC, Hanigan WC (1992) Children with cerebral venous thrombosis diagnosed with magnetic resonance imaging and magnetic resonance angiography. Neurosurgery 31: 870-876.
22. Alvis-Miranda HR, Castellar-Leones SM, Alcala-Cerra G, Moscote-Salazar LR (2013) Cerebral sinus venous thrombosis. J Neurosci Rural Pract 4: 427-438.
23. Ferro JM, Canhão P, Stam J, Bousser MG, Barinagarrementeria F, et al. (2004) Prognosis of cerebral vein and dural sinus thrombosis: results of the International Study on Cerebral Vein and Dural Sinus Thrombosis (ISCVT). Stroke 35: 664-670.
24. Bousser MG, Russell RR (1997) Cerebral venous thrombosis. London: WB Saunders
25. Coutinho JM (2015) Cerebral venous thrombosis. J Thromb Haemost 13: S238-S244.
26. Lafitte F, Boukobza M, Guichard J, Hoeffel C, Reizine D, et al. (1997) MRI and MRA for diagnosis and follow-up of cerebral venous thrombosis (CVT). Clin Radiol 52: 672-679.
27. Villringer A, Einhaupl KM (1997) Dural sinus and cerebral venous thrombosis. New Horizons 5: 332-341.
28. Martinelli I, Bucciarelli P, Passamonti SM, Battaglioli T, Previtali E, et al. (2010) Long-term evaluation of the risk of recurrence after cerebral sinus-venous thrombosis. Circulation 121: 2740-2746.
29. Miranda B, Ferro JM, Canhão P, Stam J, Bousser MG, et al. (2010) Venous thromboembolic events after cerebral vein thrombosis. Stroke 41: 19011906.
30. Dentali F, Poli D, Scoditti U, Di Minno MN, De Stefano V, et al. (2012) Long-term outcomes of patients with cerebral vein thrombosis: a multicenter study. J Thromb Haemost 10: 1297-1302.
31. Cortelazzo S, Finazzi G, Ruggeri M, Vestri O, Galli M, et al. (1995) Hydroxyurea for patients with essential thrombocythemia and a high risk of thrombosis. N Engl J Med 332: 1132-1136.
32. Brown A (2012) Preventing venous thromboembolism in hospitalized patients with cancer: Improving compliance with clinical practice guidelines. Am J Health Syst Pharm 69: 469-481.
33. Dobbs TD, Barber ZE, Squier WL, Green AL (2012) Cerebral venous sinus thrombosis complicating traumatic head injury. J Clin Neurosci 19: 1058-1059.
34. Krishnan A, Karnad DR, Limaye U, Siddharth W (2004) Cerebral venous and dural sinus thrombosis in severe falciparum malaria. J Infect 48: 86-90.
35. Jia M, Xiong N, Huang J, Wang Y, Zhang X, et al. (2012) Japanese encephalitis accompanied by cerebral venous sinus thrombosis: A case report. BMC Neurol 12: 43.
36. Mazzoleni R, Piette T, Lucas C, Seeldrayers P (2012) Cerebral venous thrombosis during acute herpes simplex menigoradicultis. What is the pathophysiological mechanism? Rev Neurol (Paris) 168: 379-380.
37. Price K, Wilson L, Tsegaye M (2012) A case of craniocervical abscess with sinus thrombosis in Lemierre's syndrome. Br J Neurosurg 26: 426-428.
38. Costa P, Del Zotto E, Giossi A, Volonghi I, Poli L, et al. (2012) Headache due to spontaneous intracranial hypotension and subsequent cerebral vein thrombosis. Headache 52: 1592-1596.
39. Fabricius J, Klotz JM, Hofmann E, Behr R, Neumann-Haefelin T (2012) Cerebral venous thrombosis and subdural haematoma: Complications of spontaneous intracranial hypotension. Fortschr Neurol Psychiatr 80: 599-601.
40. Mao YT, Dong Q, Fu JH (2011) Delayed subdural hematoma and cerebral venous thrombosis in a patient with spontaneous intracranial hypotension. Neurol Sci 32: 981-983.
41. Seiler R, Hamann GF (2009) Sinus venous thrombosis as complication of a spontaneous intracranial hypotension. Nervenarz 80: 963-966.
42. Tian C, Pu C (2012) Dural enhancement detected by magnetic resonance imaging reflecting the underlying causes of cerebral venous sinus thrombosis. Chin Med J (Engl) 125: 1513-1516.
43. Yoon KW, Cho MK, Kim YJ, Lee SK (2011) Sinus thrombosis in a patient with intracranial hypotension: A suggested hypothesis of venous stasis. A case report. Interv Neuroradiol 17: 248-251.
44. Eudo C, Petit A, Mondon K, Rippault H, Dardaine V, et al. (2011) Cerebral venous thrombosis in an individual with multiple myeloma treated with lenalidomide. J Am Geriatr Soc 59: 2371-2372.
45. May T, Rabinowe SN, Berkowitz RS, Goldstein DP (2011) Cerebral venous sinus thrombosis presenting as cerebral metastasis in a patient with choriocarcinoma following a non-molar gestation. Gynecol Oncol 122: 199-200.
46. Miki Y, Tomiyama M, Arai A, Kimura T, Suzuki C, et al. (2010) Cerebral venous thrombosis with dural arteriovenous fistulas and antiphospholipid syndrome: A case report. Neurol Sci 31: 237-238.
47. Beslow LA, Abend NS, Smith SE (2009) Cerebral sinus venous thrombosis complicated by cerebellar hemorrhage in a child with acute promyelocytic leukemia. J Child Neurol 24: 110-114.
48. Giordano P, Cecinati V, Grassi M, Del Vecchio GC, Dicuonzo F, et al. (2011) Magnetic resonance imaging screening of cerebral thromboembolic events in children with acute lymphoblastic leukemia: A pilot study. Neuropediatrics 42: 55-59.

49. Godfrey AL, Higgins JN, Beer PA, Craig JIO, Vassiliou GS (2011) In situ thrombolysis for cerebral venous thrombosis complicating anti-leukemic therapy. Leuk Res 35: 1127-1129.
50. González García H, Sacoto Erazo G, Moreno Gómez E, Blanco Quirós A, Fernández Abril MC, et al. (2013) Cerebral sinovenous thrombosis in a girl with acute lymphoblastic leukaemia carrying the prothrombin G20210A variant. An Pediatr (Barc) 78: 263-267.
51. Wang TY, Yen HJ, Hung GY, Hsieh MY, Tang RB (2011) A rare complication in a child undergoing chemotherapy for acute lymphoblastic leukemia: Superior sagittal sinus thrombosis. J Chin Med Assoc 74: 183-187.
52. Friemel SP, Mackey DW, Fenves AZ, Hise JH, Cheung EH, et al. (2002) Nephrotic syndrome presenting as dural sinus thrombosis. Am J Med 113: 258-260.
53. Al Fakeeh KN, Al Rasheed SA (2000) Cerebral venous thrombosis in the nephrotic syndrome. Saudi J Kidney Dis Transpl 11: 59-63.
54. Costa P, Biscoito L, Vieira M, Marçal M, Camilo C, et al. (2010) Endovascular thrombolysis for massive cerebral venous thrombosis in a teenager with nephrotic syndrome. Acta Med Port 23: 1141-1146.
55. Xu H, Chen K, Lin D, Dai L, Chen H, et al. (2010) Cerebral venous sinus thrombosis in adult nephrotic syndrome. Clin Nephrol 74: 144-149.
56. Lizarazo-Barrera JC, Jacobelli S, Mellado P, González S, Massardo L (2010) Extensive cerebral vein thrombosis as first manifestation of Behçet's disease. Report of one case. Rev Med Chil 138: 746-751.
57. Teresa Sartori M, Briani C, Munari M, Amistà P, Pagnan A, et al. (2006) Cerebral venous thrombosis as a rare onset of Churg-Strauss syndrome. Thromb Haemost 96: 90-92.
58. Ahbeddou N, Benomar A, Rasmouni K, Quessar A, Ouhabi H, et al. (2010) Cerebral venous thrombosis and acute polyradiculoneuritis revealing systemic lupus erythematosus. Rev Neurol (Paris) 166: 458-463.
59. Karam C, Koussa S (2008) Cerebral dural sinus thrombosis following cisplatin chemotherapy. J Clin Neurosci 15: 1274-1275.
60. Casado-Menéndez I, Uría DF, Jiménez L (2011) Cerebral venous thrombosis as a complication following a diagnostic lumbar puncture. Rev Neurol 52: 252-253.
61. Ferrante E, Spreafico C, Regna-Gladin C, Protti A (2009) Images from Headache. Cerebral venous thrombosis complicating lumbar puncture. Headache 49: 276-277.
62. Pfeilschifter W, Neumann-Haefelin T, Hattingen E, Singer OC (2009) Cortical venous thrombosis after a diagnostic lumbar puncture. Nervenarzt 80: 1219-1221.
63. Presicci A, Garofoli V, Simone M, Campa MG, Lamanna AL, et al. (2013) Cerebral venous thrombosis after lumbar puncture and intravenous high dose corticosteroids: A case report of a childhood multiple sclerosis. Brain Dev 35: 602-605.
64. Skeik N, Stark MM, Tubman DE (2012) Complicated cerebral venous sinus thrombosis with intracranial hemorrhage and mastoiditis. Vasc Endovascular Surg 46: 585-590.
65. Alvis JS, Hicks RJ (2012) Pregnancy-induced acute neurologic emergencies and neurologic conditions encountered in pregnancy. Semin Ultrasound CT MR 33: 46-54.
66. Gao H, Yang BJ, Jin LP, Jia XF (2011) Predisposing factors, diagnosis, treatment and prognosis of cerebral venous thrombosis during pregnancy and postpartum: A case-control study. Chin Med J (Engl) 124: 4198-4204.
67. Munira Y, Sakinah Z, Zunaina E (2012) Cerebral venous sinus thrombosis presenting with diplopia in pregnancy: A case report. J Med Case Rep 6: 336.
68. Guo XB, Fu Z, Song LJ, Guan S (2013) Local thrombolysis for patients of severe cerebral venous sinus thrombosis during puerperium. Eur J Radiol 82: 165-168.
69. McCaulley JA, Pates JA (2011) Postpartum cerebral venous thrombosis. Obstet Gynecol 118: 423-425.