



Cerebral Venous Thrombosis Risk Factors and Their Relationship with Optic Coherence Tomography: A Case-Control Study

Hossein Ali Ebrahimi Meimand¹, Shakiba Ahmadi², Farhad Iranmanesh^{1*}, Mahdiyeh Khazanehha¹

¹Neurology Research Center, Kerman University of Medical Sciences, Kerman, Iran

²Neurology department, Afzalipour faculty, Kerman University of Medical Sciences, Kerman, Iran

*Corresponding Author: Farhad Iranmanesh, Email: fpp_farhad@yahoo.com

Abstract

Background: Cerebral venous thrombosis (CVT) is an uncommon condition with an estimated annual occurrence of five cases per million individuals. This research aimed to investigate the risk factors associated with CVT and their correlation with optical coherence tomography (OCT) findings.

Methods: The study involved 30 patients diagnosed with CVT confirmed through neuroimaging, alongside a control group of 30 healthy individuals. The patients underwent OCT assessments following their diagnosis. Healthy participants, randomly selected from hospital staff without any known health issues, were examined by an ophthalmologist. Those with normal eye examinations proceeded to OCT, where their macular thickness and retinal nerve fiber layer (RNFL) were measured.

Results: The majority of participants in the CVT group were female (80%), with a mean age of 34.98 ± 3.78 years. Headaches were reported as the most prevalent symptom (66.7%). All patients in the CVT group were pregnant, contrasting with none in the control group, indicating a significant association with CVT ($P=0.03$). Notably, the inferior temporal layer thickness was greater in the CVT group compared to controls ($P=0.04$). Additionally, patients exhibiting papilledema had a thicker superior temporal layer than those without papilledema ($P=0.02$). Women in the postpartum period showed an 18.61-fold increase in the likelihood of developing CVT ($P=0.03$).

Conclusion: Findings from this study indicate that while macular thickness tends to decrease in CVT patients, various layers of the RNFL show increased thickness. The clinical implications of these observations remain unclear.

Keywords: Vein, Thrombosis, Optic coherence tomography

Citation: Ebrahimi Meimand HA, Ahmadi S, Iranmanesh F, Khazanehha M. Cerebral venous thrombosis risk factors and their relationship with optic coherence tomography: a case-control study. *Journal of Kerman University of Medical Sciences*. 2024;31(5):233–237. doi: [10.34172/jkmu.2024.36](https://doi.org/10.34172/jkmu.2024.36)

Received: April 16, 2023, **Accepted:** August 20, 2023, **ePublished:** October 30, 2024

Introduction

Cerebral venous thrombosis (CVT) is a rare disorder. Its prevalence is about 0.5% but varies in different regions (1). Nowadays, in developed countries, CVT is considered a non-infectious disorder with diverse clinical manifestations and a mortality rate of about 10% (2). CVT Occurs when a clot forms in the venous sinuses. Venous thrombosis results in venous infarction and damage to brain tissue due to congestion and inadequate blood flow. Thrombosis is caused by an imbalance between coagulability and fibrinolysis. The three main mechanisms of this imbalance include changes in normal blood flow, damage to the walls of arteries, and hypercoagulability. Most cases of venous thrombosis of the brain occur due to hypercoagulability (3). Many disorders can be a cause or predisposing factor for CVT (4-8). The most important risk factors include being in the pregnancy or postpartum

period, using oral contraceptive pills (OCP), and having genetic and acquired prothrombotic diseases such as factor V Leiden mutation (4-8).

Headaches are the most common manifestation of CVT and are present in 80% to 90% of patients (9-11). One-third of patients may experience focal or generalized seizures (12). It is important to get a rapid diagnosis in order to start medications (13). Brain MRI is currently the standard method for CVT diagnosis (14). Generally, patients with CVT recover in 80% of cases without functional complications. Also, 5% to 10% of patients die in the acute stage due to CVT or underlying disease (15,16). In some studies, factors such as age, degree of neurological defects at hospitalization, and the occurrence of more than two seizures were found to correlate with sudden death (17). One of the most important CVT complications is visual impairment. CVT can increase intracranial pressure,



as seen in ophthalmologic examinations for conditions such as papilledema, diplopia, and blurred vision (18). In various studies, papilledema occurred in 25% to 75% of the patients with CVT (19). Optical coherence tomography (OCT) is a non-invasive imaging technique to obtain high-resolution cross-sectional images of structures behind the eye, such as the macula, optic nerve, retina, and choroid. In this method, the images are as accurate as 10 microns, so visual impairments such as papilledema, increased optic nerve layer thickness, and eye bleeding can be evaluated with OCT (20). In Iran, very limited information has been published on the incidence of venous cerebral thrombosis. The present study aimed to investigate the epidemiological factors involved in CVT and OCT changes in these patients. The epidemiological results of this study can help identify high-risk groups and improve the clinical diagnosis process. It can also improve health by identifying CVT predisposing factors and adopting long-term policies and decisions.

Methods

The present study was a cross-sectional and analytical study. The patients in the case group were all patients whose CVT disease was confirmed by brain MRI and brain MR venography with contrast. After confirmation of the diagnosis, patients underwent OCT. In this study, the parameters assessed using OCT included the thickness of the superior outer macula (SOM), inferior outer macula (IOM), temporal outer macula (TOM), nasal outer macula (NOM), superior inner macula (SIM), inferior inner macula (IIM), temporal inner macula (TIM), nasal inner macula (NIM), central foveal thickness (CFT), and the thickness of the retinal nerve fiber layer (RNFL). In the control group, healthy individuals who had no disease were randomly selected from the staff of Shafa Hospital using a convenient sampling method. An ophthalmologist examined them. If the eye examination was normal, they underwent OCT. The case and control groups were matched in terms of sex and age. A checklist was designed to collect information including age, gender, clinical signs on admission (including headache, changes in consciousness, visual changes, local neurological defects, fever, nausea and vomiting, behavioral disorders), predisposing factors (including medical causes such as hypertension and diabetes mellitus, trauma, pregnancy, taking contraceptive pills, CVT, coagulation condition, collagen vascular diseases such as Bechet's disease and antiphospholipid antibodies, previous history of stroke and infection), and the site of CVT (superior sagittal, left transverse, right transverse, left sigmoid, right sigmoid, direct, left jugular, right jugular, and superficial veins). All subjects completed the informed consent form, and the study was approved by the Ethics Committee of Kerman University of Medical Sciences (ethical code: IR.KMU.AH.REC.1400.123).

Statistical analysis

The data were processed using the Statistical Package for the Social Sciences (SPSS) version 24. Quantitative variables were expressed as mean and standard deviation, while qualitative variables were represented by frequency and percentage. A normality test was performed on continuous variables; if they met the normality criteria, a t-test was conducted. The chi-square test was applied to evaluate associations between categorical variables. Multivariate regression analysis was also employed to adjust for confounding factors. A *P* value of less than 0.05 was considered statistically significant in this study.

Results

This study was conducted on 30 patients with CVT and 30 healthy people. Of the total subjects, 12 (20%) were male, and the rest were female. The mean age of the case and control group was 35.4 ± 8.75 and 34.2 ± 10.57 years, respectively. In the patient group, 23 people (76.7%) were women and the rest were men. In the healthy group, 25 were women (83.3%), and the rest were men. There was no significant difference between the two groups regarding age ($P=0.07$) or gender ($P=0.051$). In the CVT group, 43.3% had CVT in the superior sagittal sinus, the most common site of thrombosis, followed by transverse sinus thrombosis (23.3%). Headache (66.7%), papilledema (30%), and seizure (26.7%) were the most frequent symptoms on admission. Table 1 shows the frequency of CVT predisposing factors in both groups. Pregnancy was observed in all patients in the case group and was significantly associated with CVT ($P=0.03$) (Table 1). According to the findings presented in Table 2, patients with CVT exhibited significantly lower mean thickness measurements for the outer layer of the temporal macula, the superior inner macular layer, the inner macular layer, and the inner-temporal macular layer compared to the control group. When analyzing the RNFL across different layers between the case and control groups,

Table 1. Frequency of CVT predisposing factors in both groups

Variables	Total No. (%)	Case group No. (%)	Control group No. (%)	<i>P</i> value*
Hypertension	7 (11.7)	5 (16.7)	2 (6.7)	0.22
Diabetes	5 (8.3)	3 (10.0)	2 (6.7)	0.64
Trauma	3 (5.0)	2 (6.7)	1 (3.3)	0.55
Pregnancy	4 (6.7)	4 (13.3)	0 (0)	0.03
Postpartum period	6 (10.0)	5 (16.7)	1 (3.3)	0.08
Consumption OCP	13 (21.7)	7 (23.3)	6 (20.0)	0.75
History of DVT	1 (7.1)	1 (3.3)	0 (0)	0.31
Coagulation disease	1 (1.7)	1 (3.3)	0 (0)	0.31
Antiphospholipid antibody	2 (3.3)	2 (6.7)	0 (0)	0.15
History of stroke	6 (10.0)	3 (10.0)	3 (10.0)	0.99
Infection	7 (11.7)	24 (13.3)	3 (10.0)	0.68

* Chi-square test was used.

only the inferior temporal layer in the case group showed a significantly increased thickness compared to controls ($P=0.04$) (Table 3). Furthermore, when comparing OCT and RNFL measurements between patients with CVT who had papilledema and those who did not, it was found that the superior temporal layer was significantly thicker in patients with papilledema ($P=0.02$) (Table 4). Additionally, a multivariate analysis using a logistic regression model indicated that being in the postpartum period raised the

likelihood of developing CVT by 18.61 times (odds ratio: 18.61, 95% confidence interval: 10.18–29.21, $P=0.03$).

Discussion

This study evaluated CVT risk factors and their relationship with OCT. The most important result of the present study was that based on OCT findings, the thickness of different macular layers and CFT decreased in patients with CVT. The differences observed in the

Table 2. Comparison of OCT findings between case and control groups

OCT findings	Case group mean $\pm$ SD	Control group mean $\pm$ SD	P-value*
Superior outer macular (SOM) thickness	248.46 $\pm$ 6.18	254.07 $\pm$ 17.21	0.07
Inferior outer macular (IOM) thickness	237.34 $\pm$ 11.80	239.87 $\pm$ 11.87	0.34
Temporal outer macular (TOM) thickness	249.82 $\pm$ 4.48	263.61 $\pm$ 28.76	0.009
Nasal outer macular (NOM) thickness	232.93 $\pm$ 12.19	238.87 $\pm$ 12.10	0.70
Superior inner macular (SIM) thickness	316.19 $\pm$ 14.00	93.14 $\pm$ 10.324	0.04
Inferior inner macular (IIM) thickness	321.31 $\pm$ 13.09	336.58 $\pm$ 36.07	0.03
Temporal inner macular (TIM) thickness	225.42 $\pm$ 40.01	254.05 $\pm$ 38.65	0.007
Nasal inner macular (NIM) thickness	328.04 $\pm$ 45.02	342.95 $\pm$ 26.00	0.12
Central foveal thickness (CFT)	237.86 $\pm$ 10.23	242.07 $\pm$ 11.16	0.09

* T-test was used.

Table 3. Comparison of RNFL findings between case and control groups

RNFL findings	Case group mean $\pm$ SD	Control group mean $\pm$ SD	P value*
Superior temporal (ST) layer thickness	163.03 $\pm$ 40.38	151.28 $\pm$ 53.37	0.13
Superior nasal (SN) layer thickness	122.30 $\pm$ 87.03	119.27 $\pm$ 28.34	0.96
Temporal lateral (TL) layer thickness	83.5 $\pm$ 90.07	81.4 $\pm$ 33.73	0.71
Nasal lateral (NL) layer thickness	77.2 $\pm$ 94.79	76.3 $\pm$ 73.22	0.34
Inferior temporal (IT) layer thickness	157.33 $\pm$ 40.76	140.13 $\pm$ 54.05	0.04
Inferior nasal (IN) layer thickness	123.13 $\pm$ 94.34	121.27 $\pm$ 10.59	0.47

* T-test was used.

Table 4. Comparison of OCT and RNFL findings among patients with cerebral venous thrombosis with and without papilledema

OCT and RNFL findings	Without papilledema mean $\pm$ SD	With papilledema mean $\pm$ SD	P value*
Superior outer macular (SOM) thickness	249.7 $\pm$ 67.09	247.6 $\pm$ 90.87	0.50
Inferior outer macular (IOM) thickness	239.9 $\pm$ 63.15	236.11 $\pm$ 16.18	0.45
Temporal outer macular (TOM) thickness	250.5 $\pm$ 91.02	247.3 $\pm$ 51.78	0.11
Nasal outer macular (NOM) thickness	234.12 $\pm$ 22.21	232.31 $\pm$ 83.94	0.27
Superior inner macular (SIM) thickness	318.6 $\pm$ 69.69	309.22 $\pm$ 47.68	0.09
Inferior inner macular (IIM) thickness	326.21 $\pm$ 12.41	318.6 $\pm$ 69.69	0.16
Temporal inner macular (TIM) thickness	227.42 $\pm$ 70.46	220.34 $\pm$ 10.28	0.64
Nasal inner macular (NIM) thickness	329.47 $\pm$ 48.74	323.04 $\pm$ 38.27	0.74
Central foveal thickness (CFT)	239.01 $\pm$ 15.82	238.01 $\pm$ 86.86	0.77
Superior temporal (ST) layer thickness	159.23 $\pm$ 91.01	165.92 $\pm$ 30.36	0.02
Superior nasal (SN) layer thickness	116.21 $\pm$ 50.52	125.33 $\pm$ 74.45	0.64
Temporal lateral (TL) layer thickness	82.4 $\pm$ 53.55	84.6 $\pm$ 63.62	0.37
Nasal lateral (NL) layer thickness	77.1 $\pm$ 98.89	76.4 $\pm$ 55.65	0.26
Inferior temporal (IT) layer thickness	140.03 $\pm$ 74.77	140.33 $\pm$ 41.75	0.99
Inferior nasal (IN) layer thickness	120.72 $\pm$ 50.19	123.92 $\pm$ 62.95	0.77

* T-test was used.

outer temporal layer, inner superior and inferior layer, and the inner temporal macula were statistically significant (Table 1). Another important study result was increased RNFL layer thickness in patients with CVT. Although this difference was reported to be significant only in the inferior temporal layer, the increase in thickness can be clinically significant (Table 2). In a study conducted by Koban et al. using OCT, a decrease in macular thickness and RNFL was seen in patients with CVT (18), but in the present study, a decrease in macular thickness and an increase in RNFL thickness were observed. Due to discrepancies in results, further studies in this area are recommended to reach a conclusive finding. Regarding a similar study regarding RNFL thickness in patients with CVT, the research conducted by Zhou et al is relevant. Like the results of our study, they found that the OCT performed in this patient showed a significant increase in RNFL thickness (21), but since the above study was a case report, further studies are needed. Examining different macular and RNFL layers among patients with and without CVT is one of the strengths of the present study. However, the small sample size may be explained by the lack of significant difference between macular thickness and RNFL between patients with and without CVT. Increasing sample size might reveal a significant difference between macular thickness and RNFL. It should be noted that examining the relationship between OCT findings and CVT was one of the researchers' aims. We did not find any published article on this subject. In our study, the RNFL thickness of the superior temporal layer was significantly reduced in CVT patients with papilledema compared to patients without papilledema. Although a decrease in RNFL thickness was observed in all layers in patients with papilledema, this difference was insignificant. Evaluation of macular thickness and RNFL in patients with CVT with and without papilledema was another strength of the present study. However, the clinical value of these findings is unknown. We did not find any published article to compare our results with, but OCT has been used to detect mild papilledema (22). Bassi and Mohana, in a cross-sectional study, showed that increased RNFL thickness in all four quadrants was noted in 43.3% of patients in papilledema, and no patients with papilledema had normal RNFL thickness in all four quadrants (23). The prevalence of papilledema in our study was 30% in patients with CVT, but the most common symptom was headache (66.7%). In research conducted by Alqahtani et al, headache was the most common symptom in these patients. However, papilledema in our study and vomiting in their study were the second most common symptoms (24). The cause of the difference in the frequency of symptoms could be the difference in the number of patients. In research conducted by Foroughipoor et al, the most common clinical symptoms were headache, papilledema, blurred vision, sensory and motor

abnormality, impaired consciousness, diplopia, behavioral disorders, speech disorders, focal seizures, and generalized seizures, respectively (25), which was in line with the present study's findings. In the present study, 13.3% of the patients with CVT were pregnant. However, multivariate analysis showed that women in the postpartum period were 18.61 times more likely to develop CVT. The results of a study conducted by Khomand et al also showed the high prevalence of cerebral venous sinus thrombosis in women compared to men due to some sex-related risk factors such as OCP consumption, pregnancy, and being in the puerperium period (26). Vembu et al found that the most common risk factor for CVT in women was using OCP (11). The main CVT risk factors in Alang's study were the consumption of OCP(50%), hyper-coagulation conditions (45.7%), and, after that, dehydration, trauma, and diabetes (27). Some findings of these studies are inconsistent with the results of the present study. The differences between the results of our study and the others might be attributed to different population studies. The superior sagittal sinus is the most common site of CVT in this study. In their study, Khomand et al reported superior sagittal sinus thrombosis in 83.3% of patients (26), which is consistent with the results of the present study. In another study by Shabiri et al, vein thrombosis was reported in the superior sagittal sinus in 71.4% of patients (28) which is in line with the results of the present study. This study had some limitations, the most important of which is the small sample size. Also, patients with severely decreased levels of consciousness could not undergo OCT.

Conclusion

In conclusion, the results of this study showed that macular thickness decreases in patients with CVT, but the thickness of different RNFL layers increases in patients with CVT. The clinical value of these findings is unknown.

Acknowledgments

The authors thank the Neurology Research Center of Kerman University for supporting this project.

Authors' Contribution

Conceptualization: Shakiba Ahmadi, Farhad Iranmanesh.

Data curation: Hossein Ali Ebrahimi Meimand.

Funding acquisition: Hossein Ali Ebrahimi Meimand.

Investigation: Mahdijeh Khazaneha.

Methodology: Mahdijeh Khazaneha.

Project administration: Farhad Iranmanesh.

Supervision: Farhad Iranmanesh.

Validation: Shakiba Ahmadi.

Writing-original draft: Shakiba Ahmadi, Farhad Iranmanesh, Mahdijeh Khazanehha, Hossein Ali Ebrahimi Meimand.

Competing Interests

The authors declared no conflict of interest.

Ethical Approval

This study was approved by the Ethics Committee of Kerman

University of Medical Sciences (ethical code: IR.KMU.AH.REC.1400.123).

Funding

This study was supported by the Kerman University of Medical Sciences.

References

1. Bousser MG, Ferro JM. Cerebral venous thrombosis: an update. *Lancet Neurol*. 2007;6(2):162-70. doi: [10.1016/S1474-4422\(07\)70029-7](#).
2. Ferro JM, Canhão P, Stam J, Bousser MG, Barinagarrementeria F. Prognosis of cerebral vein and dural sinus thrombosis: results of the International Study on Cerebral Vein and Dural Sinus Thrombosis (ISCVT). *Stroke*. 2004;35(3):664-70. doi: [10.1161/01.Str.0000117571.76197.26](#).
3. Singh RJ, Saini J, Varadharajan S, Kulkarni GB, Veerendrakumar M. Headache in cerebral venous sinus thrombosis revisited: exploring the role of vascular congestion and cortical vein thrombosis. *Cephalalgia*. 2018;38(3):503-10. doi: [10.1177/0333102417698707](#).
4. Stam J. Thrombosis of the cerebral veins and sinuses. *N Engl J Med*. 2005;352(17):1791-8. doi: [10.1056/NEJMra042354](#).
5. Aaron S, Alexander M, Maya T, Mathew V, Goel M, Nair SC, et al. Underlying prothrombotic states in pregnancy associated cerebral venous thrombosis. *Neurol India*. 2010;58(4):555-9. doi: [10.4103/0028-3886.68676](#).
6. Zétola VH, Nývák EM, Camargo CH, Carraro H Jr, Coral P, Muzzio JA, et al. [Stroke in young adults: analysis of 164 patients]. *Arq Neuropsiquiatr*. 2001;59(3-B):740-5. doi: [10.1590/s0004-282x2001000500017](#). [Portuguese].
7. van Kammen MS, Zuurbier SM, Coutinho JM. Understanding cerebral venous thrombosis: rare but sometimes fatal. *Stroke*. 2019;1-10.
8. Atalu A, Fattahzadeh-Ardalani G, Sharghi A, Ezzativand H. Risk factors and clinical manifestations of cerebral venous thrombosis (CVT) in patients admitted to Ardabil city hospitals during 2012-2017. *JSM Neurol Disord Stroke*. 2019;5(4):1-4.
9. Buccino G, Scoditti U, Patteri I, Bertolino C, Mancina D. Neurological and cognitive long-term outcome in patients with cerebral venous sinus thrombosis. *Acta Neurol Scand*. 2003;107(5):330-5. doi: [10.1034/j.1600-0404.2003.00031.x](#).
10. Koennecke HC. Cerebral venous thrombosis in adults. *Vasa*. 2019;48(6):473-82. doi: [10.1024/0301-1526/a000788](#).
11. Vembu P, John JK, Mohammed MI, Al-Shubaili AF. Cerebral venous thrombosis in Kuwait. Clinical presentation, risk factors, and management. *Neurosciences (Riyadh)*. 2011;16(2):129-36.
12. Duncan IC, Fourie PA. Imaging of cerebral isolated cortical vein thrombosis. *AJR Am J Roentgenol*. 2005;184(4):1317-9. doi: [10.2214/ajr.184.4.01841317](#).
13. Coutinho JM, Ferro JM, Canhão P, Barinagarrementeria F, Cantú C, Bousser MG, et al. Cerebral venous and sinus thrombosis in women. *Stroke*. 2009;40(7):2356-61. doi: [10.1161/strokeaha.108.543884](#).
14. Sajjad Z. MRI and MRV in cerebral venous thrombosis. *J Pak Med Assoc*. 2006;56(11):523-6.
15. Hiltunen S, Putaala J, Haapaniemi E, Tatlisumak T. Long-term outcome after cerebral venous thrombosis: analysis of functional and vocational outcome, residual symptoms, and adverse events in 161 patients. *J Neurol*. 2016;263(3):477-84. doi: [10.1007/s00415-015-7996-9](#).
16. Azin H, Ashjazadeh N. Cerebral venous sinus thrombosis--clinical features, predisposing and prognostic factors. *Acta Neurol Taiwan*. 2008;17(2):82-7.
17. Stolz E, Rahimi A, Gerriets T, Kraus J, Kaps M. Cerebral venous thrombosis: an all or nothing disease? Prognostic factors and long-term outcome. *Clin Neurol Neurosurg*. 2005;107(2):99-107. doi: [10.1016/j.clineuro.2004.06.002](#).
18. Koban Y, Ozlece H, Karayol S, Huseyinoglu N. Decreased retinal nerve fiber layer thickness in patients with cerebral venous thrombosis. *BMC Ophthalmol*. 2019;19(1):57. doi: [10.1186/s12886-019-1046-9](#).
19. Narayan D, Kaul S, Ravishankar K, Suryaprabha T, Bandaru VC, Mridula KR, et al. Risk factors, clinical profile, and long-term outcome of 428 patients of cerebral sinus venous thrombosis: insights from Nizam's Institute Venous Stroke Registry, Hyderabad (India). *Neurol India*. 2012;60(2):154-9. doi: [10.4103/0028-3886.96388](#).
20. Aghsaei Fard M, Fakhree S, Abdi P, Hassanpoor N, Subramanian PS. Quantification of peripapillary total retinal volume in pseudopapilledema and mild papilledema using spectral-domain optical coherence tomography. *Am J Ophthalmol*. 2014;158(1):136-43. doi: [10.1016/j.ajo.2014.03.008](#).
21. Zhou D, Ding JY, Ya JY, Pan LQ, Yan F, Yang Q, et al. Understanding jugular venous outflow disturbance. *CNS Neurosci Ther*. 2018;24(6):473-82. doi: [10.1111/cns.12859](#).
22. Vartin CV, Nguyen AM, Balmitgere T, Bernard M, Tilikete C, Vighetto A. Detection of mild papilloedema using spectral domain optical coherence tomography. *Br J Ophthalmol*. 2012;96(3):375-9. doi: [10.1136/bjo.2010.199562](#).
23. Bassi ST, Mohana KP. Optical coherence tomography in papilledema and pseudopapilledema with and without optic nerve head drusen. *Indian J Ophthalmol*. 2014;62(12):1146-51. doi: [10.4103/0301-4738.149136](#).
24. Alqahtani MS, Alhazzani AA, Alnaami I, Alqahtani SA, Alahmari TM, Alqarni AM, et al. Clinical and epidemiological profile of cerebral venous thrombosis. *Neurosciences (Riyadh)*. 2020;25(5):380-5. doi: [10.17712/nsj.2020.5.20200028](#).
25. Foroughipoor M, Moghaddam Ahmadi A, Fadaei S, Ravankhah Moghaddam K, Sharifi Razavi A. Causes of cerebral venous thrombosis and its clinical manifestation in patients admitted in emergency unit and neurology ward of Ghaem hospital. *J Mazandaran Univ Med Sci*. 2013;22(98):86-90. [Persian].
26. Khomand P, Ahsan B, Gharibi F, Khadami AH. An evaluation of the frequency of cerebral venous sinus thrombosis and its associated factors in Sanandaj, 2010-2011, Iran. *Qom Univ Med Sci J*. 2014;8(3):49-54. [Persian].
27. Alang O, Asadollahi M. Evaluation of Risk Factors for Venous Sinus Thrombosis in Patients Admitted to Loghman Hospital. Tehran: Shahid Beheshti University of Medical Sciences, School of Medicine; 2017. [Persian].
28. Shobairi E, Razazian N, Rezaie M, Shaykh Esmali MR. Incidence rate of cerebral venous thrombosis and its related factors in Kermanshah city in 2009-2010. *Sci J Kurdistan Univ Med Sci*. 2010;15(2):64-9. [Persian].