AlQalam
Alq J Med App Sci
https://journal.utripoli.edu.ly/index.php/Alqalam/index eISSN 2707-7179

Original article

Isolated Cortical Vein Thrombosis: A Case Report

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ARTICLE INFO

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Received: *09-02-2024*
Accepted: *25-03-2024*
Published: *28-03-2024*

Keywords. *Thrombosis, Cortical Vein, Cerebral, Death.*

ABSTRACT

Isolated cortical vein thrombosis (ICVT) is a rare entity of cerebral venous thrombosis (CVT) that represents a third leading cause of death during pregnancy and puerperium. IVCT manifests with wide spectrum of symptoms including headache, seizure, focal neurological deficits and changed mental status, makes the diagnosis challenging. Pregnancy, puerperium, lumbar puncture, trauma and infection, genetic predisposing and acquired prothrombotic causes, all are well recognized risk factors for ICVT. Although no established universal consensus for management, IVCT generally treated by anticoagulant. In this paper, a case of IVCT reported in 25 years old postpartum mother, delivered by cesarean using epidural anesthesia. The case presents by persisting non-orthostatic headache in first few days post-delivery that treated as post dural puncture headache but not improved. On day 7 post-delivery developed multiple episodes of focal convulsion progressed to generalized tonic-clonic convulsion associated by left hemi-paresis. MRI gradient echo sequences showed liner hypo-signal lesion with configuration of cortical vein over right high parietal area. The MRV showed multiple small filling defects along the right cortical vein in favor IVCT. The patient treated successfully by enoxaparin (according her body weight) followed by warfarin for 3 months with complete resolution of the neurological deficit.

Cite this article. *Ibrahim O, Alhaen E, Walid Mohmmad. Isolated Cortical Vein Thrombosis: A Case Report. Alq J Med App Sci. 2024;7(2):222-226. https://doi.org/10.54361/ajmas.2472004*

INTRODUCTION

cerebral venous thrombosis is a part of thromboembolic disease, represents the third leading reason for death during pregnancy and puerperium, at a rate of 0.79 female per 100,000 maternities [1]. Isolated cortical vein thrombosis (ICVT) is a rare subtype of cerebral venous thrombosis (CVT), it represents 6% of CVT [2,3]. Due to the uncommonness of ICVT, its protean clinical and radiological manifestation make it a diagnostic dilemma. In addition, there is no established universal consensus for management, and limited knowledge about outcomes [4,5]. ICVT manifests as a wide spectrum of nonspecific symptoms like headache, new onset seizures, altered consciousness and neurological deficits, make the diagnosis of ICVT challenging [6,7]. The most usual age of presentation is in young female or male patients with risk factors, for female the risk factors is pregnancy, infection or use of oral contraceptive drugs and dural puncture, risk factors for male acquired or genetic hypercoagulable state [3,6,8].

MRI of brain and MRV is the modality of choice as it can allow visualization of thrombosed cortical vein as hyperintense on T1 and hypointense on (T2*) T2-weighted gradient echo image, moreover, secondary changes due to venous congestion that seen as swollen gyri [9]. isolated cortical venous thrombosis is very rare and has been mostly documented in a small series or as case reports [1,5]. Because of paucity of information in literature regarding ICVT,

its sometimes missed or misdiagnosed, in this case report, the clinical presentation, diagnostic approach, management protocol and outcome is described, in order to raise awareness of this rare pathology.

Case presentation

Previously healthy 25-year-old women delivered by cesarean section (SC) mood by using epidural spinal anesthesia. On day two post-operative, developed severe headache and treated as a case of postdural puncture headache by simple analgesia, and fluids and discharged. On day 7 post-operative the patient developed multiple episodes of seizures (initially were focal, then progressed to generalized tonic-clonic convulsion), and weakness of both left upper and lower limb with fever and cough.

Initial CT scan revealed no appreciable abnormality. MRI showed abnormal area of cortical and sub-cortical high signal abnormality over right temporo-parito-occipital regions in T2 and flair sequences, facilitated diffusion on DWI sequence that denoted as edema mainly due to venous congestion, while gradient echo (GRE T*) showed liner hypo-signal lesion with configuration of cortical vein over right high parietal area (figure 1). The MRV showed multiple small filling defects along the right cortical vein in favor of isolated cortical vein thrombosis (figure 2). The major dural sinuses namely: superior sagittal, inferior sagittal, transverse and straight sinuses were all patent and showing no filling defects on MRV.

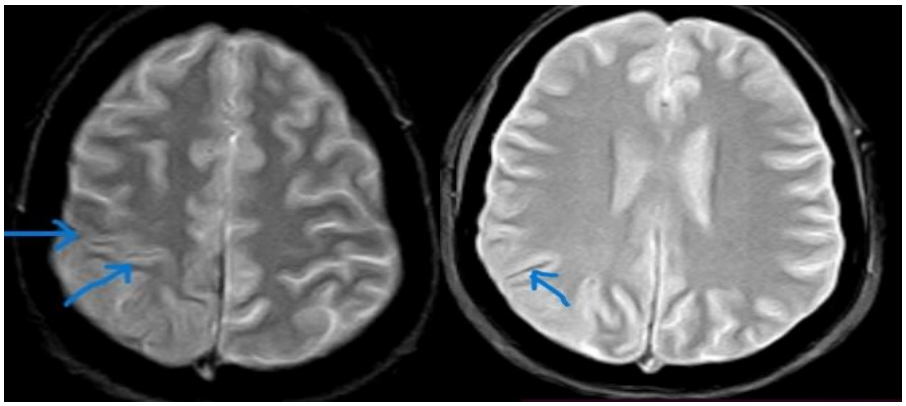

Figure 1. T2 gradient echo (GRE T*) showed linear hyposignal lesion with configuration of cortical vein over right high parietal area.

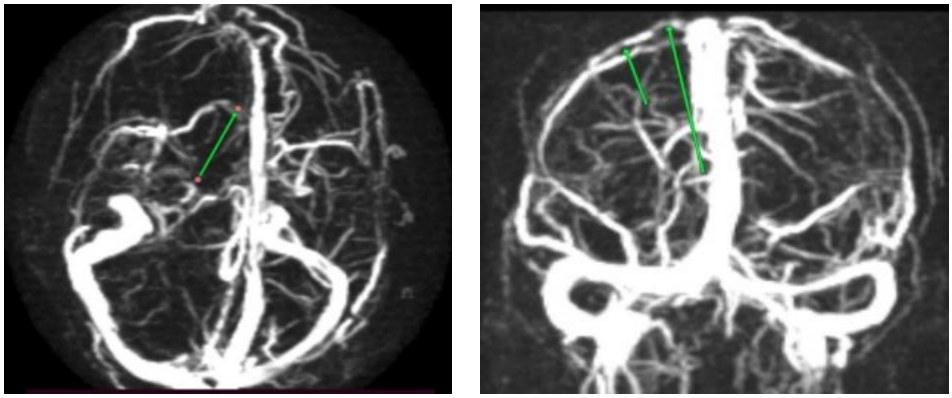

Figure 2. MRV demonstrated multiple filling defects along the right cortical vein.

Her lab results revealed leukocytosis, elevated inflammatory markers mainly due to chest infection. D-dimer was almost 10 folds higher than normal range. Antinuclear antibodies, anti-double strand antibodies, antiphospholipid antibodies all are negative. Lab results were within normal range for both renal and liver function.

The convulsion controlled by Levetiracetam (Keppra) 500mg tab twice/day. The ICVT treated by enoxaparin according her body weight bridging by warfarin in first few days then warfarin continued for 3 months, the dose of warfarin adjusted to maintain INR range between (2.5-3.5). The patient showed rapid recovery over few weeks with complete resolution of her neurological deficit. The chest infection treated by intravenous antibiotic with full recovery within few days.

DISCUSSION

Isolated cortical venous thrombosis is uncommon subtype of cerebral venous thrombosis and considered as unusual reason of stroke. In ICVT the thrombus forms inside the cortical vein without involvement of dural sinuses [6,8,10]. ICVT represents only 6.3% of all sinus and cerebral thrombosis and it is account for less than 1 % of the entire cerebral infarction [6].

A systematic review by Coutinho et al [4] involved 47 case series/ case reports showed that the mean age of the cases diagnosed with ICVT was 42 years, 68% of total cases were female. Our case was 25-year-old female. According to literature the recognized predisposing factors for ICVT are pregnancy, puerperium, lumbar puncture, trauma and infection as well as genetic predisposing factors and acquired prothrombotic causes [1,4,8].

The clinical manifestation has been reported as non-specific and including, headache, seizure, focal neurological deficits and changed mental state [4,11]. Epidural analgesia commonly utilized in labour and one of its recognized complications after epidural insertion is postdural puncture headache (PDPH), headache is a usual symptom of CVT also, thus early diagnosis is hard in the situation of peripartum dural puncture [1,2].

As cortical vein thrombosis and PDPH both presented by headache and happen early in the postpartum period, makes the diagnosis of IVCT more challenging and therefore the appropriate treatment delayed that worsen the already existing morbidity and mortality among IVCT cases [1,2,6]. Nevertheless, presence of seizure and neurological deficient prompt further imaging workup [1]. In our case the lady was delivered by cesarean section and epidural spinal anesthesia was used both are considered as risk factors for IVCT. Two days after the operation the patient developed severe headache and managed by simple analgesia and fluid as a case of PDPH then discharged. On day 7 post-cesarean the case developed multiple episodes of seizures which start as focal then progressed to generalized tonic-clonic convulsion and focal neurological deficits in the form of weakness of both left upper and lower limb. In addition to pneumonia that might be consider as another risk for IVCT.

The best diagnostic modality for ICVT is MRV [11]. Although diagnosis of ICVT is made by non-invasive imaging study, it remains challenging task due to anatomical variability of cortical veins and difficulty in visualization of small obstructed veins. In MRI the common findings are: hypo-intensity of affected cortical vein in gradient echo (T*). T2* weighted gradient echo sequence very sensitive to every paramagnetic product of Hb, makes it valuable tool in diagnosis of IVCT in early stage [9]. Furthermore, the cortical veins magnetic susceptibility effect MSE detected on T2* GE images might persist months and probably years after the diagnosis and management of ICVT [5,9,10,12]. While the intensity of venous thrombi signal on T1, T2 weighted images change chronologically as the time elapse. Moreover, in MRI other secondary changes due to venous congestion can be noticed as swollen gyri, venous infarction, and ipsilateral dural thickening. The MRV showing non-visualized thrombosed vessels or filling defects along the affected vessels [2,9,12].

In some cases, IVCT in particular hard to diagnose by using T1, T2 MR images and MRV only, due to anatomical variability in the number and size of cortical veins, that necessitate the usage of digital subtraction angiography, to demonstrated indirect signs as tortuous veins, collateral veins or delayed venous drainage [8,9, 12]. As a part of imaging workup in IVCT, non-contrast CT usually requested. it may show a cord sign, that is defined as linear hyper density of occluded vessels [3,13].

In this case, initial CT scan was normal. while MRI was enough to diagnose ICVT and the images showed abnormal area of cortical and sub-cortical high signal abnormality over right temporo-parito-occipital regions in T2 and flair sequence no diffusion restriction on DWI sequence these changes denoted as edema mainly due to venous congestion, liner hypo-intensity over the affected vein in gradient echo T*. MRV showed multiple filling defects along the right cortical vein in favor of isolated cortical vein thrombosis. The major dural sinuses were all patent and showing no filling defects on MRV.

The laboratory results of our patient revealed leukocytosis, elevated inflammatory markers mostly because of pneumonia. D -dimer was 10 times higher than normal level. Antinuclear antibodies, anti-double strand antibodies, antiphospholipid anti-bodies all are unremarkable. Liver function and renal function tests were both within normal. The treatment of CVT involve anticoagulation with heparin to end the process of thrombosis, most cases are managed with warfarin for six months after that, some case reports have documented successful utilization of endovascular thrombolysis in cases who worsen during standard anticoagulation therapy and antiepileptic treatment is used for control of seizures [1]. In exceptional cases decompressive craniotomy is used where impending herniation happens [4,8,10]. In this case the Convulsion treated by Levetiracetam (Keppra) 500 mg tab twice/ day, the ICVT treated by enoxaparin according to her body weight bridging by warfarin in first few days then continued for 3 months. The patient showed rapid improvement over few weeks with full recovery of her neurological deficits. Intravenous antibiotic is used to treat the chest infection with complete resolution within few days.

CONCLUSION

ICVT is uncommon high morbidity disease, due to the wide spectrum of clinical manifestations, the diagnosis is challenging. Therefore, raising awareness regarding clinical features and neuroimaging findings are crucial. MRI including GRET* and MRV should be performed urgently in any case of persisting severe headache and seizures in peripartum especially if there are predisposing factors, since the treatment is simple and can prevent catastrophic neurological complication.

Conflict of interest. Nil

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https://journal.utripoli.edu.ly/index.php/Alqalam/index eISSN 2707-7179

تقرير عن حالة تخثر الوريد القشري المعزول

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المستخلص

تخثر الوريد القشري المعزول نوع نادر من التخثر الوريدي المخي الذي يمثل السبب الثالث للموت خلال الحمل والنفاس. هناك مجموعة واسعة من الأعراض التي تأتي مع تخثر الوريد القشري المعزول مثل الصداع و نوبات تشنج والعجز العصبي البؤري وتغير الحالة العقلية وهذا يجعل التشخيص صعبا. الحمل و النفاس والبزل القطني والرضوح والعدوى والعوامل الوراثية وأسباب تجلط الدم المكتسبة كلها عوامل خطر معروفة جيدا لتخثر الوريد القشري المعزول ,على الرغم من عدم وجود إجماع على طريقة العلاج , تخثر الوريد القشري المعزول عادة يعالج بمضادات التخثر. في هذه الورقة سجلت حالة لتخثر الوريد القشري المعزول لسيدة تبلغ من العمر 25 سنة بعد الولادة والولادة كانت بالعملية القيصرية باستخدام ا لتخدير فوق الجافية , الأعراض التي كانت تعاني منها الحالة صداع غير انتصابي مستمر في الأيام الأولى بعد الولادة والذي تم علاجه على انه صداع ما بعد ثقب الجافية ولكن لم يتحسن . في اليوم السابع بعد الولادة تطورت الأعراض إلى نوبات متعددة من التشنج البؤري ثم إلى تشنج منشط رمعي معمم مصاحب له خزل شقي في الجهة اليسرى. أظهرت تسلسلات صدى التدرج في التصوير بالرنين المغناطيسي آفة نقص إشارة البطانة مع تكوين الوريد القشري فوق المنطقة الجدارية العليا اليمنى كما اظهر تصوير الأوردة بالرنين المغناطيسي عيوبا صغيرة متعددة في الحشوة على طول الوريد القشري الأيمن يتماشى مع تشخيص التخثر الوريد القشري المعزول . تم علاج المريضة بنجاح بواسطة الاينوكسبارين (حسب وزن جسمها) ثم بالورفارين لمدة 3 أشهر مع تعافي تام من العجز العصبي.

الكلمات الدالة. تجلط الدم، الوريد القشري، الدماغي، الموت