

Upper Extremity Deep Vein Thrombosis in a Patient with Hemorrhagic Stroke - A Case Report

Hemorajik İnmeli Bir Hastada Üst Ekstremitte Derin Ven Trombozu: Olgu Sunumu

Evren YAŞAR, Rıdvan ALACA, İsmail SAFAZ, Salim GÖKTEPE

Gülhane Askeri Tıp Akademisi Fiziksel Tıp ve Rehabilitasyon Anabilim Dalı, Türk Silahlı Kuvvetleri Rehabilitasyon Merkezi, Ankara, Türkiye

Summary

In this article, we present a 55-year-old woman with hemorrhagic stroke who had upper extremity deep venous thrombosis (DVT) at the hemiplegic side and a review of the literature concerning the DVT treatment after hemorrhagic stroke. Her right upper extremity was flaccid. The passive movements of the right shoulder were painful and there was mild edema localized in her right hand. Duplex ultrasound scanning was performed to investigate any thrombosis causing edema in the right upper extremity and a thrombus in right vena basilica was determined. We administered nadroparine calcium 19,000 IUAXa daily for 10 days subcutaneously and observed that deep venous system of right upper extremity was cleared after two weeks. The use of anticoagulants in the prevention or treatment of DVT in patients with hemorrhagic stroke is potentially hazardous and there is lack of information about this issue in the literature. In our opinion, low-molecular-weight heparins may be a relatively good choice to treat upper extremity DVT and to prevent highly fatal pulmonary embolism in patients with hemorrhagic stroke. *Türk J Phys Med Rehab 2009;55:173-5.*
Key Words: Hemorrhagic stroke, upper extremity edema, deep venous thrombosis

Özet

Bu makalede, hemiplejik tarafında üst ekstremitte derin ven trombozu (DVT) olan hemorajik inmeli, 55 yaşında bir kadın hasta ile hemorajik inmede DVT tedavisi hakkındaki literatürlerin bir derlemesini sunuyoruz. Sağ üst ekstremitesi flakstı. Pasif sağ omuz hareketleri ağrılı idi ve sağ elinde lokalize hafif ödem mevcuttu. Duplex ultrasonografik tarama yapılarak sağ üst ekstremitesinde ödeme yol açan herhangi tromboz varlığı araştırıldı ve sağ vena basilikada bir trombus saptandı. Subkutan olarak 10 gün süreyle, günlük 19.000 IUAXa "nadroparin kalsiyum" uygulandı ve 2 hafta sonra sağ üst ekstremitte derin venöz sisteminin temizlendiği görüldü. Hemorajik inmeli hastalarda DVT'nin önlenmesi veya tedavisinde antikoagülanların kullanımı potansiyel olarak risklidir ve mevcut literatürde bu konuda bilgi eksikliği vardır. Bizim görüşümüze göre, hemorajik inmeli hastalarda düşük molekül ağırlıklı heparinler üst ekstremitte DVT'sinin tedavisinde ve oldukça ölümcül olan pulmoner embolinin önlenmesinde göreceli olarak iyi bir seçenek olabilir. *Türk Fiz Tıp Rehab Derg 2009;55:173-5.*

Anahtar Kelimeler: Hemorajik inme, üst ekstremitte ödemi, derin ven trombozu

Introduction

Deep venous thrombosis (DVT) is a major complication after stroke and threatens life. The incidence of DVT in stroke patients was reported as 40-50% (1). However, upper extremity DVT accounts for 1-4% of all cases of DVT (2). Pulmonary embolism (PE) is a major life-threatening consequence of upper extremity DVT. On the other hand, it is well known that patients with hemorrhagic stroke have higher rates of DVT and PE than

patients with ischemic stroke. It may be related to the frequent use of antithrombotic prophylaxis in patients with ischemic stroke (3). However, the use of anticoagulants to prevent or treat DVT in patients with hemorrhagic stroke is potentially hazardous and there is a lack of information about this issue in the literature.

Here, we present a case with hemorrhagic stroke who had upper extremity DVT at the hemiplegic side and we review the literature on DVT treatment after hemorrhagic stroke.

Address for Correspondence/Yazışma Adresi: Dr. Evren Yaşar, Türk Silahlı Kuvvetleri Rehabilitasyon Merkezi Bilkent, Ankara, Türkiye

Phone: +90 312 291 14 07 E-mail: evrenyasar@yahoo.com **Received/Geliş Tarihi:** November/Kasım 2007 **Accepted/Kabul Tarihi:** December/Aralık 2008

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Case

A 55-year-old woman was admitted to our stroke rehabilitation unit with complaints of right-sided weakness and aphasia. Two months before the admission, she had had a hemorrhagic stroke due to uncontrolled hypertension. Her cerebral computed tomography (CT) demonstrated intracerebral hemorrhage at left basal ganglia. There were no co-morbid metabolic and cardiovascular conditions, except hypertension. Immobilization was the single factor which was known and might cause venous thromboembolism in this patient. Before admission, she had not participated in any rehabilitation programme and had taken only anti-hypertensive drugs. On admission, she was diagnosed as having global aphasia and right central facial paralysis. Her right upper extremity was flaccid (Brunnstrom stage 1), however, she had minimal right leg movement (Brunnstrom stage 2). Although her sitting balance was good, she was not able to stand up or walk independently. The passive movements of the right shoulder were painful and there was mild edema localized in her right hand.

We used a shoulder sling to provide a proper right upper extremity positioning while seating. Despite positioning of her upper extremity above heart level, her shoulder pain persisted. Supraclavicular fullness, cyanosis of extremity, and jugular or superficial vein distention were not detected in physical examination. We performed provocative maneuvers, such as Adson's test, to exclude thoracic outlet syndrome (TOS). Physical and radiological examinations excluded the diagnosis of shoulder subluxation or complex regional pain syndrome (CRPS). There were no allodynia, hyperesthesia, hyperpathia or sudomotor changes as hyperhidrosis in physical examination and no demineralization or osteoporosis in radiological examination. Routine blood tests revealed normal values including alkaline phosphatase. Arterial pulse examination was also normal in the lower and upper extremities. On the other hand, we determined increased right leg circumference.

Doppler ultrasound scanning was performed to investigate any thrombosis causing edema in the right upper and lower extremities. B-mode imaging was normal in right lower extremity deep veins and no intraluminal thrombus was observed. The pulsed-wave Doppler confirmed normal venous flow in the right lower extremity. On the other hand, a hyperechoic 16x6 mm thrombus not causing vein cutoff appeared in right vena basilica next to the antecubital region. Normal respiratory phasicity was observed on the pulsed-wave Doppler examination of the upper extremity (Figure 1).

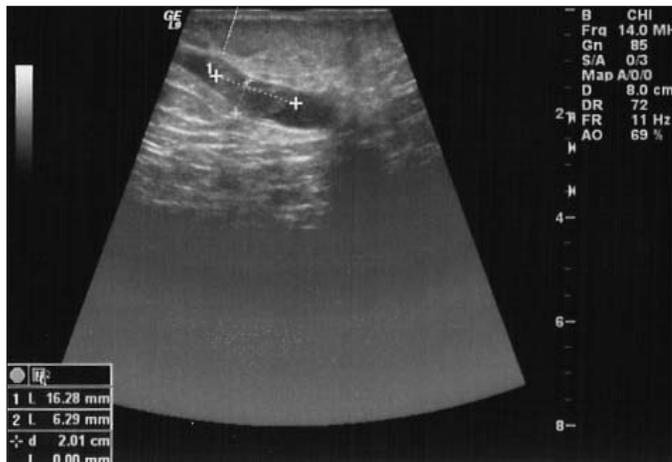


Figure 1. A hyperechoic 16 x 6 mm thrombus not causing vein cutoff appeared in right vena basilica next to the antecubital region.

Eventually, we performed laboratory testing for Protein S, protein C, antithrombin III, factor V Leiden mutation, antiphospholipid antibodies, and homocysteine and we observed that they were all within normal ranges (Table 1).

We discontinued her passive range of motion exercises and initiated nadroparine calcium 19,000 IUxa (0.1 ml/10 kg) daily for 10 days subcutaneously. We repeated the duplex scanning two weeks later. The deep venous system of the right upper extremity was cleared in colour Doppler ultrasound. At the same time, her complaints like pain and edema were reduced. Then, we started upper extremity exercises and took off her sling.

Discussion

As paralyzed arm swelling and pain are common in stroke patients, symptoms associated with DVT may be misinterpreted as being related to the stroke. In addition, there are several possible causes of persistent swelling and pain at upper extremity such as CRPS, TOS or heterotopic ossification. In our case, we excluded other causes and determined upper extremity DVT using Doppler ultrasonography. Upper extremity DVT is a relatively uncommon but important cause of morbidity. Thirty six percent of upper extremity DVT cases may progress to PE (4).

In this case, we examined any situation such as central venous catheter history, anatomic abnormalities (e.g. thoracic outlet syndrome), hypercoagulable states, malignancy, trauma, venous stasis states or medications (e.g. oral contraceptives), which are mentioned as related factors to the majority of upper extremity DVT in literature (5) and found out venous stasis state which is possibly related to flaccid extremity.

The patients with hemorrhagic stroke are at substantial risk of DVT and fatal PE in the absence of prophylaxis and of recurrent intracerebral hemorrhage if anticoagulants are used (6). Some authors notified almost four-fold greater prevalence of DVT in the hemorrhagic group compared with the thromboembolic group (2). On the other hand, Counsell et al (7) determined in a study that 5% of the patients with intracerebral hemorrhage died of PE within the first 30 days.

Although some authors have claimed that anticoagulation can safely be started 10 to 14 days after neurosurgery (8), the risk of bleeding is greatest during the first month of treatment (9). In patients with intracerebral hemorrhage, baseline risk of rebleeding after anticoagulation was determined as 0.5-1% (10,11). Hence, the treatment of clinical venous thromboembolism with anticoagulants after intracerebral hemorrhage should be planned after evaluation of the risk-to-benefit ratio (6).

Table 1. Laboratory results of the factors that may increase thromboembolism.

	Laboratory results
Protein S	118.56% (15-160%)
Protein C	112.3% (15-140%)
Factor V leiden mutation	50%
Lupus anticoagulants	Poor positive
Anti-cardiolipin Ig M	<2.0 IU/mL
Anti-cardiolipin Ig G	<2.0 IU/mL
Homocysteine	11.96 mmol/L (<12)

Our patient had hemorrhagic stroke due to hypertension 2 months before admission. She was in subacute phase. Cerebral CT pointed out that hematoma was almost completely resorbed and there was no cerebral malformation that may cause bleeding. We performed a medical treatment of DVT with low-molecular-weight heparin (LMWH) and we kept her upper extremity in rest for 10 days. Then, we repeated the ultrasonographic examination to monitor the thrombus and observed that it was resolved. Rehabilitation programme was resumed. Complaints related to paralyzed upper extremity were improved, moreover no complication was encountered.

In conclusion, DVT should be considered in a case with swelling and pain localized at paralyzed upper extremity. Colour Doppler ultrasonography, as a non-invasive method, is the gold standard for diagnosis. In our opinion, LMWH may be a relatively good choice to treat upper extremity DVT and to prevent highly fatal PE in hemorrhagic stroke. However, the decision for treatment modality and its duration should be taken after careful evaluation of the risk factors that may cause hypercoagulation or bleeding.

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