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Özgün Araştırma / Original Article

Extracranial and intracranial artery dissections: Experiences from a tertiary referral center

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Abstract

Background: Craniocervical artery dissection is an important cause of ischemic stroke especially in young and middle aged adults. In this study, we evaluated extracrainal and intracranial artery dissections in terms of etiologies, risk factors, stroke severity and functional outcomes.

Methods: A total of 29 patients who were diagnosed with ischemic stroke due to extracranial or intracranial artery dissections were enrolled to this study. The ischemic stroke diagnosis was confirmed with diffusion weighted magnetic resonance imaging in all patients. Computed tomography angiography, magnetic resonance angiography and digital subtraction angiography were used to demonstrate the dissection. Demographic findings, risk factors and presence of trauma were evaluated. National Institute of Health Score Scale (NIHSS) was used for stroke severity assessment at disease onset. Functional outcomes were measured with Modified Rankin Scale (mRS) at the 3rd month.

Results: Fifteen patients (51.72%) had carotid artery dissections while 12 patients (41.37%) had vertebral artery dissection (VAD), 1 (3.4%) had basilar artery dissection (BAD) and both VAD and BAD were seen in 1 patient (3.4%). Coagulopathy was detected in 12 patients (41.37%). Connective tissue disease was suspected in 3 patients (10.3%). In 6 patients, dissections occured after trauma. Nineteen patients (65.51%) presented with somatosensory deficits. The 3rd month mRS scores were in the range of 0-2 and no significant correlation was found in terms of risk factors, etiology and trauma history.

Conclusion: Arterial dissection should be kept in mind for the clinical presentation of ischemic stroke in young adults. We think that better understanding of the risk factors, etiologies and clinical presentation of the dissections and early diagnosis-proper treatments might yield improved clinical outcomes.

Keywords: Stroke, dissection, etiology, prognosis.

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Ekstrakraniyal ve intrakraniyal arter diseksiyonları: Bir üçüncü basamak merkezinden deneyimler

Öz

Amaç: Kraniyoservikal arter diseksiyonu özellikle genç ve orta yaşlı erişkinlerde iskemik inmenin önemli bir nedenidir. Bu çalışmada ekstrakrainal ve intrakranial arter diseksiyonlarını etiyolojiler, risk faktörleri, inme şiddeti ve fonksiyonel sonuçlar açısından değerlendirdik.

Yöntemler: Bu çalışmaya, ekstrakraniyal veya intrakranial arter diseksiyonu nedeniyle iskemik inme teşhisi konan toplam 29 hasta alındı. İskemik inme tanısı tüm hastalarda difüzyon ağırlıklı manyetik rezonans görüntüleme ile doğrulandı. Diseksiyonu göstermek için bilgisayarlı tomografi anjiografi, manyetik rezonans anjiyografi ve dijital subtraksiyon anjiyografi kullanıldı. Demografik bulgular, risk faktörleri ve travma varlığı değerlendirildi. Hastalığın başlangıcında inme şiddet değerlendirmesi için National Institute of Health Score Scale (NIHSS) kullanıldı. Fonksiyonel sonuçlar 3. ayda Modifiye Rankin Skalası (mRS) ile ölçüldü.

Bulgular: On beş hastada (%51,72) karotis arter diseksiyonu, 12 hastada (%41,37) vertebral arter diseksiyonu (VAD), 1'inde (%3,4) baziler arter diseksiyonu (BAD), 1'inde VAD ve BAD görüldü. (%3,4). Koagülopati 12 hastada (%41,37) saptandı. 3 hastada (%10,3) bağ dokusu hastalığı şüphesi vardı. 6 hastada travma sonrası disseksiyonlar meydana geldi. On dokuz hasta (%65,51) somatosensoryal defisitlerle başvurdu. 3 aylık mRS skorları 0-2 aralığındaydı ve risk faktörleri, etiyoloji ve travma öyküsü açısından anlamlı bir ilişki bulunmadı.

Sonuç: Genç erişkinlerde iskemik inmenin ayırıcı tanısında arteriyel diseksiyon akılda tutulmalıdır. Risk faktörleri, etyoloji ve klinik bulguların daha iyi anlaşılması ve erken tanı- uygun tedavilerin klinik sonuçları iyileştirebileceği görüşündeyiz.

Anahtar kelimeler: İnme, diseksiyon, etyoloji, prognoz.

INTRODUCTION

Craniocervical artery dissection was reported to be an important reason for the stroke in young adults with a prevalence of 20%[1]. In a study performed with 1008 stroke patients aged between 15 to 49 years, the dissection rate was 15%[2]. The dissection can be seen via computed tomography angiography (CTA) or magnetic resonance angiography (MRA) but the golden standard is conventional angiography. The classical finding is the double lumen image which is not frequently obvious. The most common finding is the irregular or conical narrowing of the lumen which is seen in 60% of the cases. The consequent ischemic findings in the relevant structures might be due to narrowing or occlusion of the lumen or secondary thrombotic events[3].

The dissections can be spontaneous or post-traumatic. It can be seen after major traumas, simple physical activities or coughing[4]. The clinical presentation might be with local symptoms like Horner's syndrome or ischemic conditions like transient ischemic attacks or stroke. The vascular wall vulnerability is important in the pathophysiology of especially spontaneous cases. The association between the connective tissue disorders and dissections supports this knowledge[5]. In a study conducted with 65 cases, the skin biopsy was confirmed the connective tissue disease in 55% of cases[6]. Recently, the role of mild to moderate hyperhomocysteinemia has attracted attention in the carotid artery dissections (CAD)[7]. Moreover, the genetic contribution to the pathophysiology of the CAD seems to be high[8].

Another important issue related to this topic was that the target was young adult population

which brings out social and economic problems. This is especially important because the early diagnosis and treatment can decrease both mortality and morbidity. Therefore, recognizing risk factors and the clinical characteristics of disease are so valuable[9]. There are a few studies evaluating both intracranial and extracranial dissections according to the clinical features and possible stroke mechanisms[10,11]. Chen et al reported that intracranial dissections were important causes of ischemic stroke, and displayed unique radiologic characteristics and specific stroke mechanisms compared with extracranial dissections[10].

In this study, we evaluated extracrainal and intracranial artery dissections in terms of etiologies, risk factors, stroke severity and functional outcomes.

METHODS

This retrospective study was performed with 29 ischemic stroke patients diagnosed with extracranial or intracranial artery dissections between 2011 and 2015. It was approved by our institutional ethics committee. Acute stroke diagnosis was confirmed with diffusion weighted magnetic resonance imaging (DW-MRI) in all patients. For the demonstration of dissection, CTA, MRA and digital subtraction angiography (DSA) were used. The presence of crescent sign in the T1 weighted MR images was used as a supportive finding in suspicious cases. The presence and localization of occlusions and pseudoaneurysms were recorded. The diagnosis was confirmed by Cervical Artery Dissections and Ischemic Stroke Patients (CADISP) criteria in all cases[12].

Presence of trauma, other classical risk factors, vasculitis, connective tissue diseases and coagulopathies were noted. Electrocardiography (ECG), transthoracic echocardiography (TTE) and in some cases, transesophageal echocardiography (TEE) were performed for the etiologic investigations. In the acute conditions, cardiac monitorization and/or rhythm holter monitoring was performed for rhythm abnormalities.

In the evaluation process, National Institute of Health Stroke Scale (NIHSS) scores were used at the admission and Modified Rankin Scale (mRS) was used at the 3rd month control[13,14]. Additional cranial nerve paresis or sensorial symptoms were noted. In the follow-up period, the progression or recurrence of the dissections were recorded. The association of etiological factors with the 3rd month mRS scores was also investigated.

The study was approved by our institutional ethics committee (Desicion no: 4/36, date:21.02.17).

Statistical Analysis

Statistical analysis was performed using the SPSS 11.0 statistical software package (SPSS Inc., Chicago, IL, USA). Descriptive statistics are reported as frequencies and percentages for categorical variables and as mean and range for continuous variables. Comparison of the parameters between groups was performed using $\chi 2$ tests for categorical variables and t-test for continuous variables. $P<0.05$ was accepted for statistical significance.

RESULTS

The mean age of patients was 44.8±11.4 and 16 patients (55.7%) were male. Anterior circulation infarction was detected in 14 cases and posterior circulation in 14 cases. In 1 patient there was no diffusion restriction and the problem was intramural hematoma causing secondary local symptoms. The demographic characteristics of the patients were stated in Table 1.

Carotid artery dissection was found in 15 patients (51.7%) while in 5 cases, dissections were bilateral. Vertebral artery dissection (VAD) was detected in 12 (41.4%), and basilar artery dissection in 1 patient (3.4%). In 1 patient (3.4%), vertebral artery dissections

Table I: Patients' evaluated data

Patient No	Age (years)	Gender	Risk Factors	Dissected artery	Headache-Horner	CN Paresis	Sensory-Motor	Cerebellar Signs	NIHSS	mRS
1	65	Male	HT+DM+HL	LICA	None	+	None	-	2	0
2	43	Male	HT+Smoking	LVA	None	-	Sensory+motor	-	5	1
3	56	Male	Smoking	LVA	Blurred vision	-	None	+	2	0
4	41	Female	HT	RICA	None	+	Motor	-	3	0
5	61	Female	HT	BA+BVA	None		None	+	2	0
6	35	Male	None	LVA	Horner	+	Motor	+	4	1
7	45	Female	None	RVA	None	+	Sensory	+	5	0
8	37	Male	Smoking	RICA	Headache	+	Sensory	-	2	0
9	31	Female	None	RVA	Headache	-	None	+	2	0
10	45	Male	HT	BA	None	-	None	+	2	0
11	57	Male	None	BICA	None	+	Sensory+motor	-	9	1
12	45	Male	Smoking	RVA	None	-	None	+	2	0
13	27	Female	None	RICA	Headache	-	Motor	-	1	0
14	46	Male	None	LVA	None	-	Motor	-	4	1
15	33	Male	HL	RICA	None	+	Motor	-	3	1
16	33	Female	None	BICA	None	+	Motor	-	11	0
17	38	Female	Smoking	RICA	None	-	Motor	-	4	0
18	58	Female	None	LICA	Headache	+	Motor	-	6	0
19	36	Female	HT	RVA	Headache	+	None	+	4	0
20	39	Male	None	RVA	None	-	None	-	0	0
21	73	Male	HT+DM+HL	RVA	None	+	Sensory+motor	-	8	1
22	61	Male	HT+DM+	LVA	Horner	+	Sensory	+	3	1
23	33	Male	None	LICA	None	+	Motor	-	7	1
24	52	Female	None	RICA	None	+	Sensory+motor	-	10	2
25	46	Female	None	BICA	None	-	Sensory+motor	-	8	1
26	46	Female	None	BICA	Headache	-	None	+	2	0
27	44	Female	None	BICA	None	+	Motor	-	8	1
28	43	Male	None	RVA	None	-	None	+	2	1
29	32	Male	None	LICA	None	+	Motor	-	2	1

Abbreviations: CN, Cranial Nerve; NIHSS, National Institute of Health Stroke Scale; mRS, Modified Rankin Scale; HT, Hypertension; DM, Diabetes Mellitus; HL, Hyperlipidemia; RICA, Right Internal Carotid Artery; LICA, Left Internal Carotid Artery; BICA, Bilateral ICA; RVA, Right Vertebral Artery; LVA, Left Vertebral Artery; BVA, Bilateral Vertebral Artery; BA, Basilar artery; NIHSS, National Institutes of Health Stroke Scale; mRS, modified Rankin Scale.

were bilateral. Five CAD cases and 3 VAD cases were intracranial. Total artery occlusion was seen in 5 of the internal carotid artery (ICA) dissections and 1 of the vertebral artery dissections.

6 patients (20.7%) had trauma history. The traumas were due to swimming in 1 case and falling in other cases. Hypertension was detected in 8 cases (27.6%), diabetes in 3 (10.3%) and hyperlipidemia in 3 patients (10.3%). Smoking history was found in 5 patients (17.2%).

In the laboratory tests, the lipoprotein levels were high in 2 patients. In 12 patients (41.4%), coagulation parameters were abnormal. Ten of these patients (34.5%) had homocysteine metabolism abnormalities, 1 had lupus anticoagulant positivity and 1 had decreased levels of Protein S. In patients with homocysteine metabolism abnormalities, 7 patients had methylenetetrahydrofolatereductase (MTHFR) gene mutation, and 4 patients had elevated homocysteine levels. 1 patient had both elevated homocysteine levels and MTHFR gene mutation.

After the rheumatological evaluations, the diagnosis of vasculitis was confirmed in 4 (13.8%) patients. 3 of them had positive markers and 1 had diagnostic findings in imagings. Connective tissue disease was suspected in 3 of 29 (10.3%) patients. There were bilateral ICA dissections in 2 of them. Of these patients, 1 had marfan syndrome, 1 had osteogenesis imperfecta and 1 had fibromuscular dysplasia.

TTE, TEE and holter examinations were performed to investigate the cardiac risk factors. Only in 1 patient, ventricle wall motility abnormality was detected and the rest of the patients were completely normal.

Nineteen patients (65.5%) had admitted to the emergency with sensorimotor deficits. Local findings were present in rest of the patients. 6 (20.7%) of them had headache, 2 (6.9%) patients had ipsilateral Horner's syndrome and 1 (3.4%) patient had blurred vision with completely normal neurologic evaluation. Sixteen patients had cranial nerve palsies. Seven patients had a syndrome of lower cranial nerve palsies (with invariable involvement of cranial nerve XII with or without additional involvement of cranial nerves XI, X, and IX), five had palsy of cranial nerve V, and three had a syndrome of ocular motor palsies. Palsy of cranial nerve VII occurred in one patient. Clinical presentations, admission NIHSS scores and 3rd month mRS scores were listed in Table 1. The mean NIHSS score was 4.24±2.92 for all patients. Older age and presence of more than 1 risk factor were significantly correlated with higher NIHSS scores.

While the thrombolytic therapy was given to 3 patients, 23 patients were treated with anticoagulant therapy and 3 patients with antiaggregant therapy. The 3rd month mRS scores were between 0 and 2 in all of the patients. In 2 patients there was no recanalization after 3 months. Age, gender and risk factors had no significant correlation with the 3rd month mRS scores (p>0.05) (Table II).

Table II: Correlation between mRS scores and age, gender and presence of risk factors

	mRS=0(n)	**mRS=1(n)**	**mRS=2(n)**	**p**
Age (n=28)	16	12	1	0.214
Gender Female (n=12) Male (n=16)	 9 7	 5 7	 1 0	 <0.01
Hypertension	4	0	0	0.04
Hyperlipidemia	0	1	0	0.507
Smoking	4	0	0	0.043
>1 risk factor	1	3	0	<0.001

Abbreviations: n, number; mRS, Modified Rankin Scale

Power analizi:

t tests - Means: Difference between two independent means (two groups)			
Analysis:	Post hoc: Compute achieved power		
Input:	Tail(s)	=	Two
	Effect size d	=	1.115
	α err prob	=	0.05
	Sample size group 1	=	15
	Sample size group 2	=	14
Output:	Noncentrality parameter δ	=	3.0004439
	Critical t	=	2.0518305
	Df	=	27
	Power (1-β err prob)	=	0.8243973

İki bağımsız grupta değerlendirilen parametreler üzerinden G Power programı 3.1 versiyonu ile yapılan post hoc Power analizi sonuçları yukarıdaki gibidir. Buna göre Etki büyüklüğü large olarak hesaplanmış 1. Grup 14 ikinci grup 15 kişi üzerinden yapılan iki yönlü 0.05 hata payına sahip analizin power(güç) değeri 0.82 olarak hesaplanmıştır.

DISCUSSION

Carotid and vertebral artery dissections are major causes of ischemic stroke in young to middle aged individuals in the fourth and fifth decades[15,16]. Similar to the previous studies, 72.4% of our patients were below 50 years.

The symptoms might be local or ischemic findings. Local symptoms are headache in 80-90% cases and Horner syndrome, pulsatile tinnitus and crania nerve paresis in other cases. The most frequent ischemic presentations are ocular and cerebral ischemia. Mostly encountered ischemic symptoms are hemiparesis, aphasia and amaurosis fugax[17]. Nineteen patients had sensorimotor deficits due to ischemic injury of the brain in our study. Other patients showed local symptoms, most of which were headache.

In a study performed to investigate clinical presentations, etiologies and prognosis of dissections with 22 patients, 18 patients had cerebral infarction but none had hemorrhage. These results indicate that the dissections of cerebrovascular arteries result with ischemic stroke rather than hemorrhage[17]. Similarly, 28 patients had ischemic infarction and 1 had intracranial hemorrhage in our study. The patient had parenchymal hemorrhage and intracranial CAD.

Previous studies showed that head and neck pain was the most common presentation in higher than 50% of patients while this ratio was 20.7% in our study[18,19]. The most frequent symptoms in our study were sensorimotor deficits which were seen in 65.5% of patients. A similar finding was presented in a different study with a ratio of 45.5%. In that study the percentage of patients having both sensorimotor deficit and headache was 9.09%[17].

Dissections might be traumatic or spontaneous. The frequency of traumatic cases showed variation in different studies while it was 20.7% in our study[4,8,16]. The abnormalities in arterial wall structure are blamed in dissection pathophysiology with consistently higher rate of connective tissue disease in these patients. The weaknesss in arterial walls, whether traumatic or not, increases the risk of dissections[5]. The diseases causing vessel wall pathology such as Marfan syndrome, cystic medial degeneration, fibromuscular dysplasia, Ehlers Danlos syndrome, osteogenesis imperfecta type 1 and other connective tissue diseases are blamed in dissection etiology[16,20]. In a previous study, fibromuscular dysplasia was found to be responsible from 15% of dissections while this rate was only 3.4% in our study[18]. The rates of Osteogenesis imperfecta and Marfan syndrome were equal to fibromuscular dysplasia in our study. Together with vasculitis cases, [24].1% of our cases had vessel wall pathology.

The incidence of CAD is 1.7 per 100 000 and bilateral dissections occur less frequently[21]. Bilateral ICA dissections were seen in 5 patients (17.2%) and all of them had connective tissue diseases in our study. This implies that the vessel wall pathology should be investigated in especially bilateral cases.

Hypercoagulation had a role in approximately half our patients (41.7%). The exact rate of pure homocysteine metabolism abnormality was 34.48%. Recently, mild to moderate hyperhomocysteinemia related with CAD draws attention7 and needs to evaluate in further studies. The relation between MTHFR gene mutation and CAD has been investigated in different studies[7,8], but the results were conflicting. Konrad et al stated that both hyperhomocysteinemia and MTFHR gene mutation might be regarded as risk factors for CAD[7]. Similar results were published by Luo et al[22]. However, some studies had found no significant relationship between CAD and MTFHR gen mutation[23,24]. We have confronted a significant rate of homocysteine metabolism abnormality in our study and we think it might be an important issue that should be investigated in larger studies.

Aneurysms due to traumatic carotid artery dissections are rare, but they are still more commonly seen from traumatic vertebral artery dissections[25]. Only 1 patient who had aneurysm due to traumatic bilateral ICA dissection in our study.

The outcomes were good in CAD in most of previous studies. The recanalization mostly occured between 7 and 30 days. The recurrences were seen at most in the first months and the average 10 year recurrence risk was 11%. The recurrence was more common in young patients and the prognosis is better in this group. When all dissections were considered, 88% of cases had completed recovery with early diagnosis and treatment and the mortality was seen in only 10% of the cases[5,16,17]. There was no mortality in our cases and the rate of total recovery was 89.7% which was consistent with the literature. When the prognosis was evaluated, recanalization was seen in most of the patients in CTA images, which was consistent with the literature[5]. This recanalization process was usually between 1 to 3 months. The treatment with either anticoagulation or antiplatelet drugs was evaluated for each patient especially according to the recanalization and continued for a minimum duration of 6 months.

According to 3rd month mRS scores, all of our patients had good outcomes. We found no correlation between etiological factors and mRS scores. This result might be due to the relatively low number of the patients. The other possible reason of the better outcomes compared to the literature might be related to the early diagnosis and proper treatment. Otherwise, the absence of NIHSS scores at 3 months and admission mRS scores were other limitations of our study. We think that better understanding of the risk factors, etiologies and clinical presentation of the dissections and early diagnosis-proper treatments might yield improved clinical outcomes.

CONCLUSION

The clinical presentation in dissections may have variations. In the settings of stroke with somatosensory deficit in young adult population, dissections had to be considered. Cervical trauma must also be questioned. In bilateral dissections, clinicians should investigate connective tissue diseases and vessel wall pathologies.

This study has showed that vessel wall pathologies with hypercoagulation were important in dissection etiology. We think that the relationship between homocysteine metabolism abnormality and dissection might be remarkable and studies with larger sample sizes are needed to evaluate this issue. Although dissections might result with

mortality and morbidity, the prognosis is usually good and depends on proper early diagnosis and treatment.

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