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Mechanisms, Clinical Features and Risk Factors for Stroke in the Posterior Cerebral Artery Infarcts

Posterior Serebral Arter Enfarktlarında İnme Mekanizmaları, Klinik Özellikler ve Risk Faktörleri

Şenay Aydın[1], Zehra Işıl Satılmış Borucu[2], Nevin Pazarcı[3], Dilek Necioğlu Örken[3], Hulki Forta[3]

[1]Yedikule Chest Diseases and Thoracic Surgery Training and Research Hospital, Clinic of Neurology, İstanbul, Turkey
[2]Gaziosmanpaşa Taksim Training and Research Hospital, Clinic of Neurology, İstanbul, Turkey
[3]Şişli Hamidiye Etfal Training and Research Hospital, Clinic of Neurology, İstanbul, Turkey

Summary

Objective: The objective of our study was to investigate the association between the infarcts in the cortical and deep posterior cerebral artery (PCA) perfusion area and the mechanisms of stroke, their clinical features and risk factors.

Materials and Methods: Fifty seven patients who suffered strokes first time and have infarcts in the PCA perfusion area and admitted to our Neurology Clinics between January 2002 and August 2007 were included in this study. Subjects were divided in two groups as cortical branch infarct group and deep (thalamus) + cortical branch group. Patients were evaluated in four etiologic clusters; 1. Occlusion in the posterior cerebral artery or its branches. 2. Occlusion proximal to the posterior cerebral artery. 3. Cardio-embolic reasons. 4. Cryptogenic embolism group. All patients had cranial CT, MRI, neck Doppler USG, MRA or DSA, trans-esophageal or trans-thoracic echocardiography, hematologic and vasculytic investigations. Risk factors in all patients were recorded.

Results: Thirty five (61%) patients had cortical branch infarct, 22 patients (39%) had infarcts in the cortical and deep PCA perfusion area. The cardio-embolism (n=27, 47%) was the most frequently observed etiologic factor in both groups. This is followed by intrinsic PCA disease (n=12, 21%), proximal artery disease (n=10, 17%), cryptogenic embolism (n=8, 15%). Headache was observed in 52% of the patients at the beginning of stroke. Seventy-eight of the patients had visual disturbances, 54% had motor symptoms, 24% had sensorial symptoms.

Conclusion: It was concluded that cardiogenic embolism and intrinsic PCA disease are more frequent etiologic factors in infarcts observed in the cortical and deep PCA perfusion area. There was no statistical difference in etiology and risk factors between the patients who had PCA cortical branch infarct group and deep (thalamus) + cortical branch group. (Turkish Journal of Neurology 2015; 21:49-54)

Key Words: Posterior cerebral artery, acute cerebral infarction, etiology

Özet

Amaç: Çalışmamızda posterior serebral arterin (PSA) kortikal ve derin sulama alanı enfarktlarında risk faktörleri, klinik özellikler ve inme mekanizmaları arasındaki ilişkiyi ortaya koymayı amaçladık.

Gereç ve Yöntem: Çalışmaya Şişli Etfal Eğitim ve Araştırma Hastanesi Nöroloji Kliniği'ne Ocak 2002-Ağustos 2007 yılları arasında ilk kez inme geçiren saf PSA alanında enfarktı olan 57 hasta alındı. Olgular kortikal dal enfarktı olanlar ile kortikal dal+derin dal (talamus) enfarktı olanlar olmak üzere iki gruba ayrıldı. Bu iki grupta yapılmış olan tetkikler ile hastalar dört etyolojik grupta incelendi. PSA ve dallarındaki tıkanıklık, PSA proksimalindeki tıkanıklar, kardioembolik nedenler, kriptojenik grup olarak sınıflandırıldı. Tüm hastalara kranyal bilgisayarlı tomografi (BT) ve manyetik rezonans görüntüleme (MRG), boyun Doppler ultrasonografi (DUSG), manyetik rezonans anjiyografi (MRA) ya da dijital substraksiyon anjiyografi (DSA), transösofagial ya da transtorasik ekokardiyografi (TEE, TTE), hematolojik ve vaskülitik tetkik yapıldı.

Address for Correspondence/Yazışma Adresi: Şenay Aydın MD, Yedikule Chest Diseases and Thoracic Surgery Training and Research Hospital, Clinic of Neurology, İstanbul, Turkey
Phone: +90 506 631 81 35 E-mail: aydin.senay@hotmail.com
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Bulgular: Otuz beş hasta (%61) PSA kortikal dal enfaktüsü geçirirken, 22 hastada (%39) kortikal ve derin PSA besleme alanında enfarkt saptandı. Etyolojiye bakıldığında her iki grupta da kardioembolizmin (n=27, %47) daha sık olduğu saptandı. Bunu sırayla entrensek PSA hastalığı (n=12, %21), proksimal arter hastalığı (n=10, %17), kriptojenik emboli (n=8, %15) izledi. İnme başlangıcında hastaların %52 baş ağrısı gösterdi. Hastaların %78'inde görme alanı bozuklukları, %54'ünde motor semptomlar, %24'ünde duysal semptomlar mevcuttu.

Sonuç: PSA kortikal ve derin sulama alanındaki enfarktlarda kardiyojenik emboli ve entrensek PSA hastalığının daha sık olduğu düşünüldü. PSA kortikal dal enfarktı olanlar ile derin ve kortikal dal enfarktı olanlar arasında etyoloji ve risk faktörleri açısından anlamlı istatistiksel değişiklik saptanmadı. (Türk Nöroloji Dergisi 2015; 21:49-54)

Anahtar Kelimeler: Posterior serebral arter, akut serebral enfarktüs, etyoloji

Introduction

In the past 30 years, the risk factors for ischemic cerebrovascular diseases have largely been identified thanks to international multicentric studies. Advanced age, sex, race, arterial hypertension (HT), diabetes mellitus (DM), cardiovascular diseases, hyperlipidemia (HL), cigarettes, carotid and/or vertebral artery (VA) stenosis are considered among the risk factors (1,2). The etiology of the ischemic stroke is detrimental in the secondary prophylaxis which determines the prognosis and prevents recurrence. Despite using all of the available diagnostic tools, however, the etiology of 20% of the ischemic stroke cases cannot be determined (2,3).

Determining the subgroups in stroke is important for treatment, prognosis, clinical and secondary prophylactic aspects. According to the Bamford Clinical Classification established by Bamford et al. using clinical findings, the maximum deficit findings can be used in the acute stage to reach a classification without using neuroimaging methods (4). Adams et al. used clinical findings, neuroimaging findings, Doppler ultrasonography (DUSG), conventional angiography (DSA), transthoracic echocardiography (TTE)/transesophageal echocardiography (TEE), other laboratory findings and the criteria of "Trial of Org 10172 in Acute Stroke Treatment" (TOAST) to make etiological classification in first time ischemic stroke patients (5). TOAST classification system requires many examinations in addition to the clinical history and findings, and precise laboratory findings. This method reduces the sensitivity but boosts specificity and prevents assigning cases to wrong subgroups (1,2).

Posterior cerebral artery (PCA) irrigation area infarctions are common. The fact that they cause blockages in the other arteries, their clinical properties and manifestations are well known. There has not been a lot of studies investigating the relationship between PCA infarctions distributed over different irrigation areas, stroke mechanisms and risk factors. In this study, we aimed to determine the clinical presentations, stroke mechanisms, risk factors and possible etiological differences between the patient groups who either have isolated occipital infarctions or occipital and thalamic infarctions within PCA's irrigation area.

Materials and Methods

Our study included first-time stroke patients with PCA infarctions affecting one or many cortical areas. All patients were admitted to Şişli Etfal Training and Research Hospital, Neurology Clinic between the dates January 2002-August 2007. Informed consent was obtained from all patients included in our study. Ethical approval was given by Şişli Etfal Training and Research Hospital Ethical Board. The data collection was done prospectively. Patients who had isolated thalamus or mesencephalon infarctions were excluded due to the possibility of penetrating artery disease. All patients went under cranial computerized tomography (CT), magnetic resonance imaging (MRI), neck DUSG, magnetic resonance angiography (MRA)/digital subtraction angiography (DSA). In order to determine the source of cardiac embolism, the patients who did not have cardiac examination findings went under TEE and the rest went under TTE. In patients who had early-age stroke, hematological and vascular studies were made in order to identified coagulation disorders. The risk factors such as past transient ischemic attacks, HT, DM, HL, heart disease and smoking habit were also investigated in all patients.

The patients were split into two groups according to the distribution of infarctions in the cranial imaging. The first group was "group 1/PCA", who had infarctions confined to only the cortical parts of the PCA's irrigation area. The second group was "group 2/PCA+" who had infarctions in both cortical and deep (only thalamus) parts of the irrigation area. Both groups were split into 4 subgroups according to etiology. These were intrinsic PCA disorder (IPCAD) included people who had main branch or cortical side branch-blocking PCA disorders but did not have proximal arterial or cardiac disease; Proximal arterial disease (PAD) group included people with at least one lesion on aortic arc, subclavian, vertebral and basilar arteries; Cardiac-induced Embolism (CiE) group included people who had an identified source of cardiac embolism but did not have a serious intrinsic vascular lesion that was blocking posterior circulation. Cardiac stroke sources include atrial fibrillation, drastically reduced left ventricle function (or regional wall akinesia), intracardiac clots, right-to-left shunts, wall aneurysms and other cardiac causes. The cryptogenic embolism (CrE) group included people whose etiological causes could not be identified, or those who had multiple etiologies (cardiac, aortic or vascular).

The findings were analyzed using SPSS (Statistical Package for Social Sciences) Windows 10.0. For the statistical analysis, we used descriptive statistics (mean, standard deviation), Student's t-test and Mann-Whitney U test. For the rest of the quantitative comparisons, χ^2: Chi-square test was used. The results were evaluated within their 95% confidence interval and the statistical significance threshold was determined as $p<0.05$.

Results

The study included 28 female (49.1%) and 29 male (50.9%) patients. The mean age was 67.65±12.28 and the age interval was

between 35 and 90. Group 1/PCA included 35 (61.4%) and group 2/PCA+ included 22 (38.6%) cases. Forty nine patients had HT, 20 had DM, 22 had coronary artery disease (CAD), 20 had HL, and 22 were smokers. Twenty seven patients (47.3%) had CiE, 12 patients (21.1%) had IPCAD, 10 patients (17.5%) had PAD and 8 patients (14.1%) had CrE.

A large portion of the 27 patients who had cardiac embolism had multiple cardiac abnormalities. Four of the patients who had cardiac lesions had hypokinetic/akinetic myocardial wall motion abnormality, 15 had atrial fibrillation, 2 had patent foramen ovale, 3 had atrial septal aneurysm and 4 had intracardial thrombi. Sixteen of the 27 patients who were considered for cardiac embolism source were in the PCA group and 11 were in PSA+ group. There were no statistically significant differences between the groups. An analysis of the vascular lesions of the 10 patients from the PAD group showed that 5 had them on intracranial vertebral arteries (ICVA), 4 had them on basilar artery (BA) and one of them had them on both carotid artery and ICVA. Seven of the 12 IPCAD patients were in the PCA group and 5 were in the PCA+ group. The vascular imaging of these patients showed proximal PCA stenosis in 8 patients, proximal PCA occlusion in 1 patient, bilateral distal PCA occlusion in 2 patients and fetal PCA in 1 patient. In addition, two patients showed minimal pathological findings in the PCA irrigation area and one had distal BA stenosis.

Cryptogenic embolism group was evaluated in two subgroups. The first subgroup consisted of 7 patient whose source of embolism could not be determined. Their cardiac and vascular studies were normal. Four patients in the first subgroup had PCA, 3 patients had PCA+ infarctions. In the second subgroup included only one patient, whose primary etiology could not be determined because he/she had both cardiac and vascular etiologies.

There were no statistically significant differences between the groups (group 1/PCA, group 2/PCA+) in terms of mean age, sex, etiologies, risk factors, clinical presentation and prognosis ($p>0.05$) (Table 1).

The most common clinical finding was loss of vision, seen in 45 patients (80.4%). Motor weakness was seen in 31 patients (80.4%) and it was usually mild. Fourteen patients (24.5%) showed higher-order cortical dysfunctions (HOCD) such as aphasia, anosognosia and autotopagnosia. Agitation and confusion was seen more frequently compared to loss of consciousness (n=4, 7%). Cases with motor disorders were more often in Group 2 (81.8%), and vertebrobasilar system findings (VBSF) were more often in group 1 (35.3%). The difference between the groups were statistically significant ($p<0.05$) (Table 2).

Discussion

In the general population PCA irrigation area infarcts were seen at a rate of 8% among all cerebrovascular diseases, and at a rate of <10% among other cerebral infarcts (6). In addition, certain patient series reported PCA infarction frequency between 5-25% (7,8,9). Cortical PCA irrigation area infarcts located in calcarine, temporooccipital, parietooccipital and temporal artery irrigation areas constitute 2/3rd of all PCA infarcts while combined PCA infarcts (deep+cortical) were reported as 5-38.5% in some series (1,2,6,8,10). Posterial cerebral artery involvement was shown in 40% of the patients in studies using MRI (1,6,7,11). Kumral et al. reported that the thalamus involvement in combined PCA infarcts are 5 times more likely than mesencephalon involvement and expressed that this ratio can become important for the embolism as indicated by the previous studies (12). Three large-scale studies conducted with CT detected thalamus involvement in 42 patients (19%) our of 221 (13,14). In our study, 22 patients (38.9%) had thalamus infarction in addition to thalamus infarction. This high ratio associated with thalamic infarction can be explained by the use of MRI, which is a more sensitive imaging technique. In our series, PCA etiology was indicating embolism at a rate of 64.8%. This finding is in line with other series reporting the frequency of embolism in PCA as somewhere between 52% and 82% (6,7,8,9, 10,12,13,14,15,16,17,18,19).

Yamamoto et al. reported that embolisms in PCA's cortical and/or deep irrigation areas are most frequently of cardiac etiology (41%) and that cortical PCA infarctions are more often seen in cardiac embolism group (81%). The second most common etiological cause was proximal artery disease (32%) and 68% of the patients in this group had combined PCA infarctions. Involvement patterns consisting of other posterior circulation areas, such as brainstem and cerebellum, were more often seen in the PCA infarction developing as a result of artery-to-artery embolism whereas the PCA infarction seen in CiE and IPCAD groups were mostly isolated (9). In Kumral et al.'s 137-case series on superficial PCA irrigation area infarctions, posterior circulation infarctions constituted 11% of all ischemic strokes and 52% of the patients had cortical, 38% had combined and 10% had bilateral PCA infarctions. Broken down for etiology, 26% of the patients had IPCAD, 24% had PAD, 17% had CiE and 5% had CrE. In this study, there were no significant differences between the three groups in terms of etiology. Cardiac-induced embolism was more common in cortical PCA group followed by PAD but IPCAD and PAD were more commonly seen in the combined PSA group (18). In 11 studies consisting of 145 cases, 70% of the cases who had VA dissection had posterior circulation infarctions (17). Medullar and cerebellar infarctions in addition to PCA infarctions are often seen after VA dissection but cortical PCA infarctions through distal embolism are not common (12,17). Broken down for etiology, cardioembolism was most common in our series (47.3%) but there were no statistically significant differences between the two groups (group 1: 45.7%, group 2: 50%). When compared to earlier reports, the frequency of cardioembolism in our study was higher than Arboix (21.6%), Lee (20%) and Kumral (17%), less than Ntaios (52.9%) and about the same with Yamamoto (41%) (7,8,9,18,19,20). In addition, we found a superiority of PCA infarction in the CiE group, similar to Ntaios (20). While earlier studies reported IPCAD's frequency as 1.7% to 16%, the inclusion of MRI or MRI angiography in these studies increased this frequency (18.5%, 30%, 43.6%, 49.6%, 50.6%) (7,8,9,12,19,21). In our series, IPCAD group which was the second most common subgroup (21.1%) had 7 cases of cortical PCA and 5 cases of PCA+ infarction out of 12 patients. The lesion in the PAD subgroup was most commonly in intracranial VA (17.5%). The rate of patients with unidentified etiology was reported between 8-32% in recent large-scale series (9,11,12,14,18,19,21,22,23,24,25). In our series, this rate was 14.1%. The results of the earlier studies showing high cryptogenic embolism and low cardioembolism rates can possibly be explained by the existence of proximal arterial lesions such as

atherosclerotic plaques in aorta or cardiac etiologies that could not be detected with the technologies available at that time. When TEE is not conducted in an orderly manner, aortic lesions can be overlooked in patients with cryptogenic or cardiac embolism etiology (Table 3). There was no statistically significant differences between the two groups in terms of etiological classification.

Much like the previous studies, HT was the most important risk factor in our study (68%). Mean age in our sample was similar to previous studies. Male sex dominance seen in the samples of the previous studies was not observed in ours (16,17,22).

Visual field abnormalities were the most common clinical finding in PCA irrigation area infarctions. The visual symptoms developing in PCA infarctions can be explained by the weak collateral circulation in the inferior striate cortex. The importance of visual symptoms and findings were established in the previous studies (8,9,10,12,18,21,23). The majority of the visual field defects seen in 45 patients in our series was hemianopia. Much like other studies, even though there was no statistically significant difference between the two groups, this symptom was more common in the cortical PCA group. Bilateral visual field defects and cortical blindness was observed in 2 patients in our series. As reported by earlier series, this clinical finding is often seen as a result of CiE in cases with bilateral PSA infarctions (8,9,18,21,23,26). Hemiparesis is seen especially in PAD group among patients who had PCA irrigation area infarctions, and it usually occurs after the inclusion of cerebral peduncles in the

Table 1. Comparison of the two groups in terms of etiology, risk factors, prognosis and presentation

			Group 1		Group 2		p; t, χ^2 ,Fχ^2*
			n	%	n	%	
Etiology	CiE		16	45.7	11	50.0	χ^2=0.41, p=0.937
	PAD		7	20.0	3	13.6	
	IPCAD		7	20.0	5	22.7	
	CrE		5	14.3	3	13.6	
Risk Factors	HT		29	82.9	20	90.9	Fχ^2, p=0.466
	DM		12	34.3	8	36.4	χ^2=0.026, p=0.873
	CAD		12	34.3	10	45.5	χ^2=0.711, p=0.399
	HL		11	31.4	9	40.9	χ^2=0.533, p=0.465
	Smoking		13	37.1	9	40.9	χ^2=0.081, p=0.776
	Sex	Male	14	40.0	14	63.6	χ^2=3.020, p=0.082
		Female	21	60.0	8	36.4	
	Yaş		**Mean ± SD**		**Mean ± SD**		t=-1.635, p=0.108
			65.57±12.22		70.95±11.91		
Prognosis	Cured		16	45.7	7	33.3	χ^2=1.623, p=0.444
	Ongoing		18	51.4	14	66.7	
	Ex		1	2.9	-	-	
Presentation	Sudden		28	80.0	16	72.7	χ^2=3.29; p=0.192
	TIA		7	20.0	4	18.2	
	Progressive		-	-	2	9.1	

*t: Student t test, χ2: Chi-square test, Fχ2: Fisher's exact Chi-square test, CiE: Cardiac-induced embolism, PAD: Proximal arterial disease, TIA: Transient ischemic attack, IPCAD: Intrinsic posterior cerebral artery disease, CrE: Criptogenic embolism, HT: Hypertension, DM: Diabetes mellitus, CAD: Coronary artery disease, HL: Hyperlipidemia

Table 2. Clinical evaluation of the groups

	Group 1		Group 2		
	n	%	n	%	t, χ^2, Fχ^2*
Visual field defects	30	88.2	15	68.2	χ^2=3.403, p=0.065
Ataxia	4	11.4	6	7.3	χ^2=2.344, p=0.126
Motor impairment	13	38.2	18	81.8	χ^2=10.266, p=0.001‡
Sensory impairtment	9	26.5	5	22.7	χ^2=0.100, p=0.752
VBSF	12	35.3	2	9.1	χ^2=4.891, p=0.027†
Consciousness state	2	5.7	2	9.1	Fχ^2, p=0.635
HOCD	6	17.6	8	36.4	χ^2=2.496, p=0.114
Headache	17	50.0	13	59.1	χ^2=0.444, p=0.505

*t: Student t test, χ^2: Chi-square test, Fχ^2: Fisher's exact Chi-square test, VBSF: Vertebrobasilar system findings, HOCD: Higher-order cortical dysfunctions
†p<0.05 significance threshold, ‡p<0.01 significance threshold

infarction area (8,18,19,21,27,28). Lateral posterior choroidal artery or thalamogeniculate artery involvements are shown with MRI and MRA studies in PCA infarctions that show hemiparesis (27,29). Combined PCA infarctions involving lateral thalamus often show mild sensory and transient paresis (9,18). Motor findings were observed in 31 patients in our study and they were usually mild. In the literature, this rate changes between 8-34% (8,9,11,21,23,26,30). In our group, the rate of motor findings was higher compared to other studies (54.3%). This rate was found to be significantly high especially in the PCA+ group (81%, p<0.01). In the present study, motor findings were associated with thalamus involvement since patients with peduncle involvement were excluded from our sample. Earlier studies found sensory symptoms at rates of 15-31% (8,9,21,22,23,26). Yamamoto et al. reported that sensory symptoms in the thalamic infarctions are more frequent and that most of the infarctions involve sensory nuclei in the ventrolateral section of thalamus (9). Sensory loss was observed in 14 of our patients (24%). Even though 22 patients had thalamic infarction, sensory loss was seen in less than half of these patients (n=5). The lower rate in combined PCA infarction patients suggested that sensory findings may have been overlooked in the exam (Table 4).

In our series, 71.9% of our stroke patients had sudden onset and etiologically this was mostly true for CiE group (36.8%).

Table 3. Comparison of etiology in the literature

	Servan et al. (26) (n=76)	Moriyasu et al. (17) (n=72)	Steinke et al. (12) (n=74)	Pessin et al. (23) (n=35)	Yamamoto et al. (9) (n=79)	Cals et al. (21) (n=117)	Kumral et al. (18) (n=137)	ŞETRH NC§ (n=57)	Ntaios et al. (19) (n=185)
CiE	27	12	23	10	32	51	23	27	98
PAD	18	8	16	6	25	13	33	10	25
IPCAD	0	2	6	0	7	4	33	12	21
CrE	T¶	27	T¶	11	8	38	7	8	29
Coagulopathy	T¶	T¶	T¶	3	3	3	2	0	T¶
Vasoconstriction	3	T¶	T¶	5	4	4	T¶	0	T¶
Unknown	21	23	18	0	0	4	0	0	12

CiE: Cardiac-induced embolism, PAD: Proximal arterial disease, IPCAD: Intrinsic posterior cerebral artery disease, CrE: Cryptogenic embolism
ŞETRH NC§: Şişli Etfal Training and Research Hospital Neurology Clinic, T¶: Unknown

Table 4. Comparison of clinical features in the literature

	Servan et al. (26) (n=76)	Brandt et al. (8) (n=127)	Pessin et al. (23) (n=35)	Yamamoto et al. (9) (n=79)	Kumral et al. (18) (n=137)	Cals et al. (21) (n=117)	ŞETRH NC§ (n=57)
Visual field defects	64	118	35	66	127	112	45
Sensory	24	37	7	12	65	22	14
Motor	T¶	36	3	23	47	16	31
Cognitive	31	41	7	20	57	20	4
Ataxia	T¶	T¶	1	21	T¶	1	10

ŞETRH NC§: Şişli Etfal Training and Research Hospital Neurology Clinic, T¶: Unknown

In Yamamoto et al.'s study, the presentation and progression of stroke was associated with embolic etiology. However, the same study showed that sudden onset is most frequently seen in the PAD group (88%) and 8% of these patients showed progressive decline after the sudden stroke onset (9). Confirming previous studies, we also found transient ischemic attack presentation more frequently in the PAD group (40%) predominantly accompanied by visual symptoms (63%) (9,31). The mortality rate in our series (1.7%) was congruent with earlier reports (0-8.2%) (8,9,11,13,18,21,25).

In conclusion, cortical PSA infarctions were seen more frequently than combined PCA infarctions in our study. Etiologically, embolism was present most of the time. Cardioembolism was frequent in both groups. Cortical PCA infarctions were generally present in patients who had cardiac-induced embolism. The most common transient ischemic attack symptoms were visual. The most common clinical finding was visual field defects. In addition, motor findings were observed more frequently in the combined PCA group where thalamus was also involved. However, there were no statistically significant differences for etiology or risk factors between cortical and combined PCA infarctions in our study.

Ethical Committee Approval: The study was approved by Şişli Etfal training and Research Hospital Ethical Board

Informed Consent: Informed consent was obtained from all patients included in the study

Concept: Şenay Aydın, Zehra Işıl Satılmış Borucu, Nevin Pazarcı, Dilek Necioğlu Örken, Hulki Forta

Design: Şenay Aydın, Zehra Işıl Satılmış Borucu, Nevin Pazarcı, Dilek Necioğlu Örken, Hulki Forta

Data Collection or Processing: Şenay Aydın, Zehra Işıl Satılmış Borucu, Nevin Pazarcı

Analysis or Interpretation: Şenay Aydın, Dilek Necioğlu Örken, Hulki Forta

Literature Search: Şenay Aydın, Zehra Işıl Satılmış Borucu, Nevin Pazarcı

Written by: Şenay Aydın

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