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Superior Semicircular Canal Dehiscence in Idiopathic Intracranial Hypertension: Preliminary Report

İdiyopatik İntrakraniyal Hipertansiyon Hastalarında Süperior Semisirküler Kanal Dehisansı: Ön Rapor

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ABSTRACT Objective: Idiopathic intracranial hypertension (IIH), is a challenging condition with raised intracranial pressure without any identifiable cause. It's incidence increases due to it's close association with obesity. This pathology has strong relation with tegmental dehiscence and spontaneous cerebrospinal fluid (CSF) leaks. There are reports of tegmental dehiscence, spontaneous CSF leaks ocurring with SSCD. Superior semicircular canal dehiscence is also associated with obesity in the literature. The aim of this study is to determine the incidence of SSCD in IIH patients, to evaluate the audiovestibular findings in IIH patients with SSCD, and to discuss the possible pathogenetic mechanisms causing this co-occurence. **Material and Methods:** Twenty five consecutive patients diagnosed with IIH in the neurology department between 2016-2018 were evaluated. Ten patients fulfilling the necessary criteria and accepting to participate in this study, were enrolled in the study group. Audiometry, tympanometry, vestibular evoked myogenic potentials (VEMP) tests and high resolution computed tomography (HRCT) imagings were performed. The control group was constituded of 20 age and sex matched patients attended to our clinic with various other complaints, and to whom HRCT was conducted between 2016 and 2018. **Results:** Among the study group 1 (10%) patient with IIH had SSCD. Two (20%) patients had thinning in the bony canal. None of the patients in the control group had neither radiographic SSCD, nor bony thinning. Patient with SSCD had pathological signs in VEMP. **Conclusion:** According to our results, though not statistically significant, the incidence of SSCD seems higher in IIH. The incidence of bony thinning also seems more frequent in IIH. However further studies with wide patient series are essential.

Keywords: Idiopathic intracranial hypertension; superior semicircular canal dehiscence; vertigo; hearing loss

ÖZET Amaç: İdiyopatik intrakraniyal hipertansiyon (IIH), tanımlanabilir herhangi bir nedeni olmayan kafa içi basıncı yüksekliği durumudur. Obezite nedeni ile dünya çapında görülme sıklığı artmaktadır. Bu patolojinin tegmental dehisans ve spontan beyin-omurilik sıvısı (BOS) kaçaklarıyla güçlü bir ilişkisi vardır. Superior semisirküler kanal dehisansı (SSKD) da literatürde obeziteyle ilişkilidir. Bu çalışmanın amacı, IIH hastalarında SSKD insidansını belirlemek, SSKD'li IIH hastalarında odyovestibüler bulguları değerlendirmek ve bu birlikteliğe neden olan olası patogenetik mekanizmaları tartışmaktır. **Gereç ve Yöntemler:** 2016-2018 yılları arasında nöroloji bölümünde IIH tanısı konulmuş 25 ardışık hasta değerlendirildi. Gerekli kriterleri yerine getiren ve bu çalışmaya katılmayı kabul eden 10 hasta çalışma grubuna alındı. Odyometri, timpanometri, vestibüler uyarılmış miyojenik potansiyeller (VEMP) testleri ve yüksek çözünürlüklü bilgisayarlı tomografi (HRCT) görüntülemeleri yapıldı. Kontrol grubu, kliniğimize çeşitli şikâyetlerle başvuran ve 2016-2018 yılları arasında HRCT uygulanan, yaş ve cinsiyet uyumlu, 20 hastadan oluşturuldu. **Bulgular:** Çalışma grubunda IIH olan 1 (%10) hastada SSKD tespit edildi. İki (%20) hastada kemik kanalında incelme mevcuttu. Kontrol grubundaki hiçbir hastada radyografik SSKD veya kemik kanalda incelme saptanmadı. SSKD'li hastada VEMP'de patolojik bulgular tespit edildi. **Sonuç:** Sonuçlarımız istatistiksel olarak anlamlı olmamakla birlikte, SSKD insidansı IIH'de daha yüksek görünmektedir. Geniş hasta serileriyle yapılacak ileriye dönük çalışmalar esastır.

Anahtar Kelimeler: İdiyopatik intrakraniyal hipertansiyon; superior semisirküler kanal dehisansı; vertigo; işitme kaybı

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Superior semicircular canal dehiscence is a condition which may cause vestibular and/or cochlear symptoms, resulting from the direct contact of the inner ear and neighbouring structures.[1] Otic capsule has two mobile windows for the sound transmission, however in this pathology a 'third mobile window' is created.[1] Superior semicircular canal dehiscence syndrome (SSCDS) was first defined by Minor et al. in 1998.[2,3] The 'third window' causes an acoustic energy loss and increased stimulation of the vestibular system. Patients with SSCDS have increased sensitization of the vestibular system to bone-transmitted sound and intracranial pressure (ICP) alterations.[1,3] Valsalva maneuvers both against pinched nostrils (increased middle ear pressure) or a closed glottis (increased intracranial pressure) may cause motion in the superior semicircular canal ampulla (ampullofugal, ampullopetal respectively).[1]

The confirmation of the bony dehiscence is made by high resolution computed tomography scans (HRCT).[4] However prevalence of radiologic SSCD is higher compared to clinical studies. It is emphasized in the literature that images with 1.0 mm thickness have low specificity in the identification of SSCD.[4] Additionally the incidence of thinning of the bone overlying the superior canal in the opposite ears of patients with radiologically determined SSCD is higher compared to patients without SSCD.[5,6] These findings may suggest a developmental or congenital abnormality in this syndrome but Nadgir et al. demonstrated in their radiological study that, SSCD significantly increases with age and and they suggested an increasing trend of thinning of the overlying bone.[7] They also found no relation of thinning in one side and canal dehiscence in the opposite.[7] Though the etiology regarding this pathology remains unclear so far; it seems likely that a congenital or developmental bony dehiscence may stand silent until an acquired trauma or persistent increased intracranial pressure occurs.[7] Whyte Orozco et al. reported in their cadaveric study that the bony layers which lack of the external and middle layers, make the canal vulnerable to following events leading to fracture and dehiscence.[8]

Idiopathic intracranial hypertension (IIH) is a challenging disorder, with raised ICP in the absence of no identifiable cause.[9,10] It has an increasing incidence due to the increase in obese patients. The reported annual incidence of this pathology in young overweight females is as high as 20 per 100.000 individuals.[9] Currently IIH, is diagnosed according to the following criteria: 1) symptoms and signs of increased intracranial pressure; 2) normal neurologic exam except 6th cranial nerve abnormalities; 3) increased cerebrospinal fluid (CSF) opening pressure (≥250 mm H_2O) but normal CSF constituency; 4) no evidence of hydrocephalus, mass, structural, or vascular lesion on imaging; 5) no other cause of increased ICP identified.[9,10] Up to date we know very little regarding the pathogenesis of this disease.

The aim of this study is to determine the incidence of SSCD in IIH patients and to evaluate the audiovestibular functions of these IIH patients both with and without SSCD. To our knowledge, this is the first prospective study investigating SSCD in IIH patients.

MATERIAL AND METHODS

Twenty five consecutive patients diagnosed with IIH in the neurology department between 2016 and 2018 were evaluated. The diagnosis of IIH was made according to the clinical criteria defined for IIH.[9,10] Ten patients (20 ears) fullfilling the necessary criteria and accepting to participate in this study were included in the study group.

Audiometry, tympanometry, vestibular evoked myogenic potentials (VEMP) tests and high resolution computed tomography (HRCT) imagings were performed to the cohorts.

The criteria for establishing the study group patients were as follows:

1. ≥18 years of age

2. Patients who were diagnosed with IIH,

3. IIH patients who do not have any other neurological and/or otological pathologies that may cause any audiovestibular symptoms,

4. IIH patients who are not in the gestational period,

5. IIH patients who could easily accomodate audiometry, tympanometry, VEMP tests and HRCT scanning.

The control group consisted of 20 (40 ears) age and sex matched individuals who attended to our clinic with various other complaints, and to whom HRCT was conducted in years between 2016 and 2018.

The study group patients were informed and asked to sign the clinical research informed consent form. This study was approved by Başkent University Institutional Review Board and Ethics Comittee (Project no: KA16/333) and supported by Başkent University Research Fund.

Routine otorhinolaryngologic examination was performed in the study group. Audiometry test was conducted with the International Acoustics Company (IAC) standards with 'Clinic Audiometer, AC40'(Interacoustics Co., Assens, Denmark) and 'Telephonics TDH 39P' earphone (Telephonics Co., Farmingdal, New York, U.S.A). Air and bone conducting tresholds were determined between 250-8000 Hz., and 250-2000 Hz. respectively. Acoustic impedance analysis was detected using Grason Stadler/Tympstar (Grason-Stadler Inc.,Eden Prairie, Minnesota, USA).

Both cervical (c VEMP) and ocular (o VEMP) VEMPs were performed on both sides for all patients with 'EP 25' (Interacoustics Co., Assens, Denmark) device. During c VEMP, the patients were asked to sit straight with the neck rotated towards the opposite side of the ear that was stimulated. The active electrode was placed in the middle one-third of the sternocleidomastoid muscle (SCM), the reference electrode was placed above the sternoclavicular joint and the ground electrode was placed in the middle forehead. Ipsilateral SCM responses to monoaural stimulus (tone burst stimulus in 500 Hz.) was recorded.The electromyographic signals were amplified, and filtered below 10 Hz, and 3000 Hz. The stimulation period was 5 mseconds and stimulus given for 50 mseconds with a velocity of 5 Hz. via a 'Telephonics TDH-49P'earphone (Telephonics Co., Farmingdal, New York, U.S.A). Calculation of the average of 128 stimuli was recorded. During the analysis, the first positive (p13) and the following negative (n23) wave latencies and interpeak amplitudes were calculated.

For o VEMP, all the patients were placed in a sitting position and they were asked to look superomedially at a small target placed 1 m from the eyes. Bipolar electrodes were placed: one just beneath the eye over the inferior oblique muscle (IOM) and the reference ~1 cm below, whereas the ground was placed on the forehead. VEMP tests were repeated 3 times on each subject to ensure reliability and reproducibility of responses. The initial negative-positive biphasic waveforms (N1 and P1) and the amplitude of the first positive-negative peak (p1-n1) were recorded. VEMPs were defined as present or absent. Abnormal VEMP was defined as a pattern of 'no response'. All VEMPs were recorded at 105 dBnHL.

Asymmetry ratios were calculated in both tests as: 100 $(A_L-A_S)/(A_L+A_S)$, in which A_L refers the larger p1-n1 amplitude, A_S refers the smaller p1-n1 amplitude.[11]

The HRCT scans were performed using a Siemens Hi-Speed CT scanner (Siemens, Erlangen, Germany) with the scanning parameters of 130 kV, 94 mA. All the patients in the study underwent scanning in the supine position with the head reclined and neck flexed. The scan plane was parallel to the orbitomeatal line and skull base. Images were acquired in the sequential mode with a 1 mm slice thickness and zero interslice gap. The images were reformatted from the raw data using a 512x512 matrix. Measurements were performed in a workstation (GE Advantage, Windows version 4.2; GE Medical Systems, Wilmington, MA,USA). For the optimal evaluation of superior semicircular canal dehiscence, coronal reformatted imagings were obtained, The dehiscence was defined as a defect or a discontinuity in the bony capsule of a superior canal observed in at least two planes.[4] If a dehiscence was noted, then the widest part of the dehiscence was measured. If the bone thickness was ≤0.1 mm, then it was considered as a 'very thin' bony layer.[12] A radiologist who was blind to the study analysed the images.

The quantitative variables were expressed as the 'mean ± standard deviation (mean ± SD) and median

(maximum-minimum), while categorical variables were shown with n (%).

RESULTS

In the study group, 7 (70%) patients were female, 3 (30%) patients were male. The mean age was 40.6±11.1. The control group consisted of 14 (70%) female, 6 (30%) male patients with mean age 41.1±11.3.

Of the study group, 1 (10%) patient (1 of 20 ears) had SSCD (left ear) (Figure 1, Figure 2). Two (20%) patients (2 of 20 ears) had very thin bony layer covering the superior semicircular canal (1 patient in the right ear, 1 patient in the left ear) (Figure 3, Figure 4). The patient with SSCD had no thinning of the bony canal in the opposite ear.

None of the control group patients (none of the 40 ears) had neither SSCD nor thinning of the bone overlying the superior semicircular canals.

Nine patients with IIH, had normal pure tone averages, while 1 patient (without SSCD) had mild low frequency (250 Hz) sensorineural hearing loss. All the study patients (100%) had middle ear impedance and compliance levels within normal levels and positive acoustic reflexes.

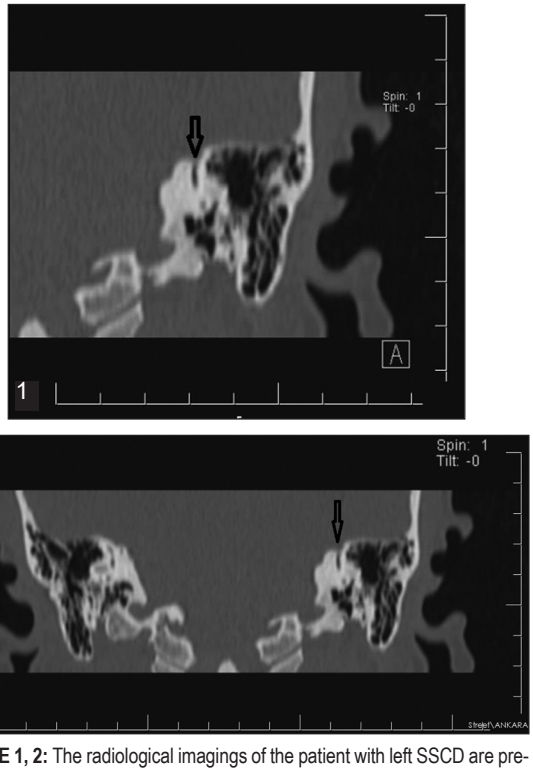

FIGURE 1, 2: The radiological imagings of the patient with left SSCD are presented.

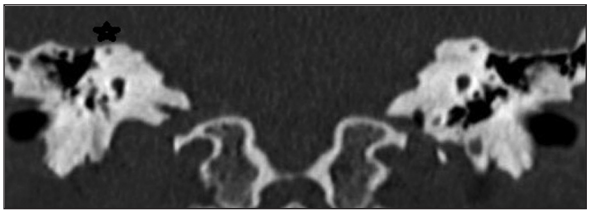

FIGURE 3: The radiological finding of the right ear of the patient with thinned superior semicircular canal is presented.

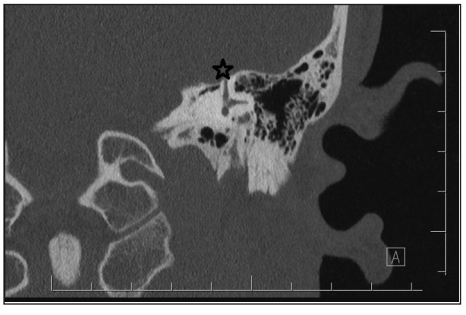

FIGURE 4: The radiological finding of the left ear of the patient with thinned superior semicircular canal is presented.

Eight of the IIH patients (16 ears) undergone VEMP tests and the results are shown in Table 1, 2. For o VEMP, the mean N1 latency was 12.6 ±4,5 (6-21.33) msec. The mean P1 latency was 14.7±1.7 (12-17.46) msec. The mean N1-P1 amplitude was 4.3±3 (0.2-15.11) µV. For c VEMP, the mean N1 latency was 23.5±1.9 (21.3±28) msec, mean P1 latency was 15.8±1.3(14-18,7) msec. Mean N1-P1 amplitude was 86.4±72.4 (13.4-300.2) µv. Accordingly, patient with SSCD had increased interpeak amplitudes of the affected (left) ear in both VEMPs (Table 1, Table 2).

DISCUSSION

In patients with SSCD, the superior semicircular canal and middle cranial fossa communicate abnormally. Thus we proposed that increased ICP may provoke the thinning of the bony layer covering the superior semicircular canal leading to the development of SSCD.

In 2017, a study that retrospectively evaluated the radiographic incidence of SSCD in IIH in 24 patients found no SSCD in their IIH patients.[12] How-

TABLE 1: o VEMP results of the study group.

Patient (#)	Age	Sex	Right P1 (msec)	Right N1 (msec)	Left P1 (msec)	Left N1 (msec) Amplitude	Right N1-p1 (μV)	Left N1-P1 Amplitude (μV)	Asymmetry ratio %
1	19	M	12.00	6.00	12.00	8.67	4.117	4.480	3.4
2	45	F	15.33	10.33	16.67	12.67	3.471	3.949	6.7
3	29	F	14.34	10.96	12.50	10.38	4.011	3.870	2.6
4	32	M	16.11	11.38	17.46	11.29	3.315	3.121	3.1
5	43	F	16.46	11.54	15.25	9.38	4.371	4.780	4.34
6	55	F	15.33	21.33	14.33	21.33	4.072	3.214	11.1
7	44	F	12.67	16.33	16.00	19.33	2.931	3.986	14.3
8*	39	F	14.25	10.96	14.04	10.3	0.19	15.11	96.7

*: The patient with SSCD. Patient #1 had thin bony layer of the left superior semicircular canal, #6 had thin bony layer of the right superior semicircular canal.

TABLE 2: c VEMP results of the study group.

Patient (#)	Age	Sex	Right P1 (msec)	Right N1 (msec)	Left P1 (msec)	Left N1 (msec) Amplitude	Right N1-p1 (μV)	Left N1-P1 Amplitude (μV)	Asymmetry ratio %
1	19	M	17.33	26.00	15.67	27.67	132.1	106.7	10.6
2	45	F	15.67	24.00	16.33	24.67	64.24	180.0	47.4
3	29	F	14.7	22.3	17.00	24.00	29.40	38.35	13.2
4	32	M	14.3	22.3	15.3	23.3	67.70	71.80	2.93
5	43	F	18.7	26.0	16.7	24.00	97.371	89.311	4.3
6	55	F	15.33	21.33	14.33	21.33	23.30	39.55	26.4
7	44	F	16.67	23.33	14.33	22.33	30.00	13.41	38.2
8*	39	F	14.0	21.3	14.7	22.3	99.64	300.23	50

*: The patient with SSCD. Patient #1 had thin bony layer of the left superior semicircular canal, #6 had thin bony layer of the right superior semicircular canal.

ever in the same study, they reported an incidence of 8.2% for SSCD in patients without IIH. They suggested that there was no association between IIH and SSCD.The incidence of SSCD in our cohort was 9%. Varios other studies reported a radiological incidence between 0,8-12% for SSCD.[7,13-16] Our findings correlate with the literature.

LeVay et al. found that patients with spontaneous CSF leakes are more likely to be obese.[17] All the patients' but one had their tegmen defects repaired and no longer experienced CSF otorrhea. Thus they recommended a detailed screening for increased intracranial hypertension in those patients. Tegmen defects are strongly associated with IIH in the literature as previously mentioned. However, though not statistically significant, we found an increased incidence of SSCD in IIH compared to the control group. We observed thinning of the bony canal wall in two patients with IIH. We did not observe such a sign in the control group. Notably Davey et al. pointed out that an individual will lose nearly 0.005 mm of bone overlying the superior semicircular canal per year.[18] Thus, it is likely that patients with IIH have an increased susceptibility to develop SSCD and consequently SSCD. Therefore our results may be of remarkable clinical significance.

Belden et al reported high sensitivity for both HRCT images with 1mm and 0.5 mm thin sections, however they suggested that the spesificity decreases when 1 mm sections are used. In our institute, HRCT is performed with 1mm slice thickness.[4]

In SSCDS, conductive hearing loss in two or more frequencies with an air-bone gap of 5-10 dB is observed. However, cases with sensorineural hearing loss are also noted. Tympanometry commonly shows preserved acoustic reflexes in this pathology.[16] The patient with SSCD in our cohort had normal hearing tresholds, with positive acoustic reflexes. According to the study by Crovetto et al. SSCD is more frequently diagnosed radiograhically, compared to temporal bone histopathologic evaluations.[14] Thus not all the SSCD patients with radiologic evidence has clinical audiovestibular symptoms and signs. The IIH patients were not evaluated at the time of their initial diagnosis for IIH. Thus they were under various therapies for the normalization of the CSF pressure. Therefore it was likely that the audiovestibular signs might have been regressed.

VEMP, a relatively new vestibular testing procedure has attracted increased interest in recent years. There seems to be a consensus that cVEMP predominantly reflects saccular activation via the inferior vestibular nerve, whereas oVEMP reflects utricular activation via the superior vestibular nerve.[19]

After the diagnosis of SSCD via HRCT, clinical confirmation using especially oVEMP is suggested in the literature.[19-21] In SSCDS, o VEMP with air conducted stimuli shows increased amplitudes and decreased tresholds.[1] Zuniga et al. showed N1 amplitude of greater than 9.3 µV and a peak-to-peak amplitude (N1-P1) of greater than 17.1 µV 100% sensitivity and spesificity for SSCD.[21] They demonstrated that c VEMP has sensitivity and specificity between 80-100%.[12,21] This was believed to be because o VEMP shows mainly the utricular function, while c VEMP reflects saccular function.[12,22] Milojcic et al. compared the audiometric signs and c VEMP results of SSCD patients affected ears and non-affected ears, and, found that patients with SSCD had significantly lower tresholds.[23] Çoban et al. evaluated audiovestibular signs in IIH patients and observed various pathological signs(prolonged latency, increased or decreased amplitudes, no response) in c VEMP tests of eight patients; while only 1 healthy volunteer in the control group had abnormal VEMP responses.[24] They suggested that increased intracranial hypertension may cause increased endolymphatic pressure as seen in hydrops, affecting the saccular maculae and causing pathological VEMP responses. However some of the pathological signs in VEMP could have been related to SSCD.

We performed both c VEMP, and o VEMP in our participants. In our study, the mean N1 latency and mean amplitude values were increased; whereas the mean P1 latency was similar when compared to the normative data of our own clinic.[25,26] Also, the mean N1 and P1 latencies of c VEMPs seemed consistent with various normative data published in the literature.[27-30] The mean amplitude values were consistent with the literature.[27-30] However, the patient with SSCD had increased interpeak amplitudes of the affected ear in both o and c VEMPs (the asymmetry ratios were 97%, 50% respectively). This indicates a peripheral pathology in the left ear.

The major limitation of this study was the low number of patients. Therefore, it was not possible to achieve statistical significance. However despite the rising incidence, IIH is still not commonly observed. Another limitation was the delayed time period between the initial diagnosis of IIH and the audiovestibular evaluation. Prospective controlled studies with larger sample sizes are required.

CONCLUSION

In this study, SSCD was observed only in IIH group. Also thinning of the bony canal was detected in IIH group; a result which we did not observe in the control group. Thus, it is worth noting that the results may be of clinical significance, which requires to be ascertained by future studies. To our knowledge this is the first study that prospectively investigated the correlation between SSCD and IIH.

Ethical Approval

All procedures performed in studies involving human participants were in accordance with the ethical standards of Başkent University research committee (Project no: KA16/333) and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Source of Finance

During this study, no financial or spiritual support was received neither from any pharmaceutical company that has a direct connection with the research subject, nor from a company that provides or produces medical instruments and materials which may negatively affect the evaluation process of this study.

Conflict of Interest

No conflicts of interest between the authors and / or family members of the scientific and medical committee members or members of the potential conflicts of interest, counseling, expertise, working conditions, share holding and similar situations in any firm.

Authorship Contributions

Idea/Concept: *Kübra Çoban;* ***Design:*** *Kübra Çoban, Erdinç Aydın;* ***Control/Supervision:*** *Kübra Çoban, Seda Kibaroğlu, Erdinç Aydın, Feride Kural Rahatlı;* ***Data Collection and/or Processing:*** *Kübra Çoban, Gülfem Beyazpınar, Seda Kibaroğlu, Gülfem Beyazpınar;* ***Analysis and/or Interpretation:*** *Kübra Çoban, Gülfem Beyazpınar, Feride Kural Rahatlı, Seda Kıbaroğlu;* ***Literature Review:*** *Kübra Çoban;* ***Writing the Article:*** *Kübra Çoban;* ***Critical Review:*** *Erdinç Aydın, Kübra Çoban;* ***References and Fundings:*** *Erdinç Aydın, Gülfem Beyazpınar, Seda Kibaroğlu, Feride Kural Rahatlı;* ***Materials:*** *Gülfem Beyazpınar, Seda Kibaroğlu, Feride Kural Rahatlı.*

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