Introduction

Arachnoid cysts (ACs) are non-tumorous intra-arachnoidal space-occupying lesions that are filled with clear, colorless liquid that resembles cerebrospinal fluid. Pathologically, they contain a thin transparent wall that is separated from the inner dural layer and underlying pia-arachnoid (1). Arachnoid cysts consist of liquid formations surrounded by an arachnoideal sheet (2). The prevalence of ACs has been found 0.5–1.7 % in hospital-based and clinical studies. It has been found that men have ACs two to three times higher than women (3,4). The etiology of ACs has been an arguable topic. It can be congenital cysts which were called true ACs, a most common type, or secondary ACs (5). Infections, brain trauma or increased intracranial pressure are an etiologic factor of the ACs (6). Congenital ACs are developmental anomalies which occurred from maldevelopment of the primitive arachnoid membrane in embryonal life. In acquired cysts, the intracystic fluid may contain hemosiderin or inflammatory cells depending on the etiology (7).

Arachnoid cysts are often asymptomatic and discovered incidentally. The clinical features of ACs are variable and usually nonspecific. The neurological symptoms and signs of ACs are dependent on localization, size and the impact of cerebrospinal fluid (CSF) circulation. They can be a headache, vomiting, symptoms of hydrocephalus, focal neurological deficits, cerebellar signs and seizure (5,8). If ACs are small they are usually asymptomatic, but bigger supratentorial, suprasellar and posterior fossa cysts may impact CSF flow which can cause hydrocephalus. Large middle cranial fossa cysts may be associated with headaches and seizures (5). Most of the arachnoid cysts (90%) were found in supratentorial locations and the others are in the posterior fossa (5,6).

Studies about the association of epilepsy and ACs did not provide accurate data (6). It can be seen in epilepsy but there is no relation to the specific seizure type or focus in electroencephalogram (EEG). We investigate the effect of ACs on EEG in order to determine the necessity of EEG in patients with arachnoid cysts and without a history of epilepsy.

ABSTRACT

Aims: Studies about the association of epilepsy and arachnoid cysts (ACs) did not provide accurate data. It can be seen in epilepsy but there is no relation to the specific seizure type or focus in electroencephalogram (EEG). We investigate the effect of ACs on EEG in order to determine the necessity of EEG in patients with arachnoid cysts and without a history of epilepsy.

Methods: This is a retrospective, case-control study. Participants of this study are patients with incidental ACs without history of seizures as ACs group, patients with epilepsy as epilepsy group and patients without history of seizures as control group. EEG findings were classified as normal, nonspecific abnormal and specific epileptiform activity.

Results: The study included 104 patients in ACs group, 102 patients in epilepsy group and 105 patients in control group. We identified a significantly higher proportion of slow waves (17.6%) and epileptiform EEG discharges (27.5%) in epilepsy patients compared to ACs group (slow waves: 5.8%, epileptiform EEG discharges: 1.9%, p<0.001). There was no difference on EEG activity between the ACs group and control group (slow waves: 3.8%, epileptiform EEG discharges: 1%, p=0.667). Also, there was no relation between the localization of ACs and EEG abnormality.

Conclusions: To the best of our knowledge this is the first study which evaluate the effect of incidental ACs on EEG activity. We found that ACs do not have effect on EEG compared to control groups and also there is no correlation between the localization of ACs and EEG abnormality.
However, to the best of our knowledge, there is no EEG study in patients with incidental ACs and without a history of seizures. And we do not know the effect of ACs on EEG in patients without the history of seizures. In this study, we investigated the effect of ACs on EEG in patients with ACs and without seizure history and compared these findings with the EEG of epilepsy patients and the EEG of patients who have spells and history inconsistent with epilepsy. The primary aim of this study was to evaluate the necessity of EEG in patients with ACs and without a history of epilepsy.

**Methods**

**Study Population**

This is a retrospective, case-control study. Participants of this study are patients who were admitted to the neurosurgery outpatient clinic for incidental ACs without a history of seizures and were consulted to the neurology clinic with EEG records. These patients were accepted as group ACs. MRI scans of these patients were evaluated. The patients who have concomitant intracranial space occupying mass or history of seizures were excluded.

The patients who have epilepsy and EEG was performed for control were accepted as epilepsy group. Also if they have ACs they are also excluded from the study. Finally, the patients who were performed EEG and do not have epilepsy or ACs with normal neurological examinations were accepted as a control group.

**EEG Protocols**

We used a 32-channel acquisition system with international 10–20 electrode placement. The routine EEG record was performed for 30 minutes and the standard activation process, eye-closing, eye-opening, included 3-min long hyperventilation and intermittent photic stimulation in a selected stimulation range of 1-60 Hz were applied. All EEG recordings were evaluated by two senior neurologists (ÖK, GK). The EEG interpretation was performed with knowledge of the location of the arachnoid cyst. EEG results were classified as normal, nonspecific abnormal such as theta wave and specific interictal discharges such as sharp or spike wave. The EEG of all groups were evaluated in this term.

**Brain MRI Acquisition**

MRI was performed in all patients. Structural images were acquired using 1.5 T Symphony Siemens, 1.5 T Intera Philips, and 3 T Achieva Philips MRI Scanners. Protocols for conventional T1-weighted, T2- weighted and FLAIR sequences with sagittal, axial and coronal orientation were performed. Section thickness was 5 mm.

**Results**

A total of 311 patients included the study, the number of patients in group ACs, epilepsy and control was 104, 102, 105 respectively. The median age of patients was 29.5 (18-59), 27 (19-60), 26 (19-55) years in group ACs, epilepsy and control respectively. There were 30 females and 74 males in group ACs where 48 females and 54 males in the epilepsy group and 60 females and 45 males in the control group. There was a significant difference in age and gender between the groups. The age of patients in the ACs group was higher than the epilepsy group and control group. Also, there was a male dominance in group ACs compared to epilepsy and control groups (Table 1).

We identified a significantly higher proportion of slow waves (number = 18, percentage = 17.6%) and epileptiform EEG discharges (number = 28, percentage = 27.5%) in epilepsy patients compared to ACs group (slow wave number = 6, percentage = 5.8%; epileptiform EEG discharges number = 2, percentage = 1.9%) (p<0.001). There was no difference on EEG activity between the ACs group and control group (slow wave number = 4, percentage = 3.8%; epileptiform EEG discharges number = 1, percentage = 1%) (p=0.667). Table 1 showed the comparison of all the groups.

In the group ACs, placement of ACs was supratentorial in 89 patients (85.5%) and infratentorial in 15 (14.5%) patients. When placement of ACs and EEG recordings was evaluated, in the supratentorial group 82 patients had normal EEG, 5 patients had a slow wave on EEG and 2 patients had epileptiform discharges, in the infratentorial group 14 patients had normal EEG and 1 patient had a slow wave on EEG. There was no difference between the groups (p>0.05).

Table 2 showed the relation between localization of ACs and

<table>
<thead>
<tr>
<th>Table 1. Age, gender and EEG results of groups</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
<tr>
<td>Age mean (min-max)</td>
</tr>
<tr>
<td>Gender (F/M)</td>
</tr>
<tr>
<td>Normal, N (%)</td>
</tr>
<tr>
<td>Slow wave, N (%)</td>
</tr>
<tr>
<td>Epileptiform discharges N (%)</td>
</tr>
</tbody>
</table>

ACs: Arachnoid cysts, EEG: Electroencephalogram, F: Female, M: Male, N: Number, P value less than 0.05 was accepted as statistically significant.
EEG abnormality. Two patients had left frontal ACs and one of them had left temporal slow wave while the other had left frontal slow wave. Two patients had left temporal ACs one of them had left temporal slow wave and the other one had right temporal slow wave. Right temporal localized ACs were found in two patients who had epileptiform EEG discharge right temporal and right temporo-parietal localization. Left occipital AC was found in one patient who had left occipital slow wave. The patient whose localization of AC was cerebellar had left temporal slow wave. There was no relation between them (r=0.6,05, p=0.112).

Discussion

The main finding of this study is there is no difference on EEG findings between ACs patients and control group which suggest that ACs do not have an effect on EEG. In healthy adults occurrence of epileptiform EEG abnormalities was reported in 0.11 to 2.5% of aircrew applicants (11). Also Oh et al. found EEG abnormalities in 9 (1.2 %) of 740 pilot applicants which were only 3 (0.4%) applicants had epileptiform discharges (12). We found epileptiform activity in ACs group 1.9% which is not different from healthy adults. This finding showed that the rate of epileptiform discharges is the same between individuals with ACs and healthy adults.

There was no relation between the localization of ACs and localization of abnormality on EEG. This finding showed that ACs have no effect localization of abnormal activity on EEG. Consistent with our finding Yalcin et al. found EEG abnormality consistent with arachnoid cyst location in only one patient out of 20 patients. They screened 612 epilepsy outpatient clinic patients and found 20 patients with ACs. Generalized seizures were seen in 11 patients and 4 patients had generalized spike waves, 5 patients had diffuse slowing. They did not find a relation between ACs and a specific seizure type or EEG focus (9). Arroyo et al. found that ACs are an incidental finding in epileptiform patients and do not show the location of the epileptogenic zone. They retrospectively evaluated patients in their epilepsy clinic with interictal or ictal EEGs who had cerebral imaging. Seventeen of 867 patients had ACs. Temporal lobe cysts were detected in 12 patients which only 3 of them had temporal lobe epilepsy Frontal lobe cysts were seen in 4 patients, only one of them had frontal lobe epilepsy ipsilaterally. They saw only 4 (23.5 %) patients that the seizure focus was adjacent to the ACs (6).

<table>
<thead>
<tr>
<th>Patients No</th>
<th>ACs Localization</th>
<th>EEG abnormality</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Left occipital</td>
<td>Left occipital slow wave</td>
</tr>
<tr>
<td>2</td>
<td>Left frontal</td>
<td>Left frontal slow wave</td>
</tr>
<tr>
<td>3</td>
<td>Cerebellar</td>
<td>Left temporal slow wave</td>
</tr>
<tr>
<td>4</td>
<td>Right temporal</td>
<td>Right temporoparietal sharp wave</td>
</tr>
<tr>
<td>5</td>
<td>Left temporal</td>
<td>Right temporal slow wave</td>
</tr>
<tr>
<td>6</td>
<td>Left temporal</td>
<td>Left temporal slow wave</td>
</tr>
<tr>
<td>7</td>
<td>Left frontal</td>
<td>Left temporal slow wave</td>
</tr>
<tr>
<td>8</td>
<td>Right temporal</td>
<td>Right temporal sharp wave</td>
</tr>
</tbody>
</table>

ACs: Arachnoid cysts. EEG: Electroencephalogram. All EEG data of the other 92 patients with ACs were normal.

Table 2. Localization of ACs and EEG abnormality

We found that ACs were seen in males more than females. Rabiei et al. found that 2.7 % of the women and 1.5 % of the men had ACs which was not a significant difference (16). Whereas Al-Holou et al. found greater prevalence in 356 (1.8%) of 20.327 men and compared to 305 (1.1%) of 28.090 women. Also, most previous studies reporting with a two to three times higher occurrence of ACs among men than among women, which has been found in hospital-based and clinical studies consistent with our results (4,8,17,18).

In ACs group EEG abnormalities consistent with localization had strong correlation coefficient but not statistically significant. Also, EEG abnormalities in ACs group had a higher number and percentage compared to the control group but this is also not statistically significant. The limitation of our study is that it was retrospective. In this study variable MRI machines of the different magnetic field may affect the evaluating of ACs.

In a recent large population-based study, authors did not find a difference in epilepsy between with and without AC which all the cysts were asymptomatic (16). Arachnoid cysts rarely present with neurological signs or symptoms, many studies revealed that a majority of arachnoid cysts are asymptomatic and are found incidentally in most cases (4,8,17,19). Even if neurological symptoms are present, it is hard to properly correlate symptoms with ACs (4,8,14,20). Supporting this, we found that in asymptomatic patients with ACs that there is no difference on EEG compared to control group.

To the best of our knowledge, this is the first study in the literature which evaluate the effect of ACs on EEG activity. We found that ACs do not have an effect on EEG compared to control groups and also there is no correlation between the localization of ACs and EEG abnormality which leaves a strong question mark on the necessity and cost-effectiveness of EEG recording in patients with ACs. In future, prospective randomized-controlled, multicenter studies with a larger number can clear this uncertainty. We suggest that a more-targeted EEG
recording approach is applied to the patients with ACs with epilepsy risk factors or a seizure history as revealed by thorough medical history.

**Acknowledgment**

Author contributions: Concept; G.K. and Ö.K., design; G.K., supervision; Ö.K., resource; G.K. and Ö.K., materials; G.K. and Ö.K., data collection &/or processing; G.K. and Ö.K., analysis &/or interpretation; G.K. and Ö.K., literature search; G.K., writing; G.K., critical review; Ö.K. No financial support was received from any source for this work.

**Conflict of Interest**

The authors declared they do not have anything to disclose regarding conflict of interest with respect to this manuscript.

**References:**


