

# A Case of Arachnoid Cyst Presenting with Cognitive Impairment and Hypomania Symptoms



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## SUMMARY

Arachnoid cysts are benign congenital malformations of the arachnoid which account for approximately 1.4% of the intracranial lesions. Although it is usually asymptomatic, it may be accompanied by headache, hydrocephalus and seizure. Psychiatric disorders associated with arachnoid cysts are rare. In this article, we present a giant arachnoid cyst with hypomania symptoms and marked cognitive impairment. A 44-year-old female patient was admitted to our outpatient clinic with a 4-year history of headache, nervousness and attention problems. Magnetic resonance imaging revealed a giant arachnoid cyst with a size of 5.5x10.5x12.5 cm was found in the left hemisphere of the patient. Considering the patient's irritability, increase in the amount of speech, flight of ideas, sleep disturbance and attention disorders, the diagnosis of hypomania was made. The neuropsychological tests showed that the speed of information processing, mental flexibility and attention functions decreased, and executive functions were impaired. The patient was consulted to the neurosurgery department. But no surgical treatment was offered. Drug therapy for hypomanic symptoms and cognitive impairment was planned, but could not be started since the patient did not attend to the follow-up exams. Albeit the lack of follow-up constitutes a limitation for our report, we believe that the size of the cyst, significant impairment of cognitive functions and the presence of hypomania symptoms might contribute significantly to the literature. Other cases with arachnoid cyst displaying cognitive impairment were summarized in our article.

**Keywords:** Bipolar disorder, arachnoid cyst, memory disorders, cognitive disorders, hypomania, affective symptoms

## INTRODUCTION

Arachnoid cysts (AC) are benign congenital malformations of arachnoid mater and have been reported prevalence of 1.4% in adults (Al-Holou et al. 2013). AC is formed by the accumulation of cerebrospinal fluid between the arachnoid membrane layers surrounding the neural tube. It is usually caused by developmental defects or trauma. (Helland and Wester 2007).

ACs are located in the middle cranial fossa, mostly on the left, and in the retrocerebellar region with respective incidences of 34% and 33%. Only 5% of the cases are reported to be symptomatic; the most common symptoms associated with AC being headache, hydrocephalus, ataxia, seizures and dizziness (Al-Holou et al. 2013). There are very

few case reports in the literature on psychiatric disorders comorbid with AC; and those reported are mostly psychotic disorders and depression (Kuloglu et al. 2008, Boomkens et al. 2010, Miskey and Gross 2016, Mormont et al. 2017). In this article, we present a case with giant arachnoid cysts accompanied with marked cognitive impairment and hypomania symptoms.

## CASE

The 44-year old female patient, graduated from university, divorced and living with her two children, consulted our outpatient clinic with complaints of headache, irritability and distraction. Her headaches had started four years previously, following an intermittent course, not localized

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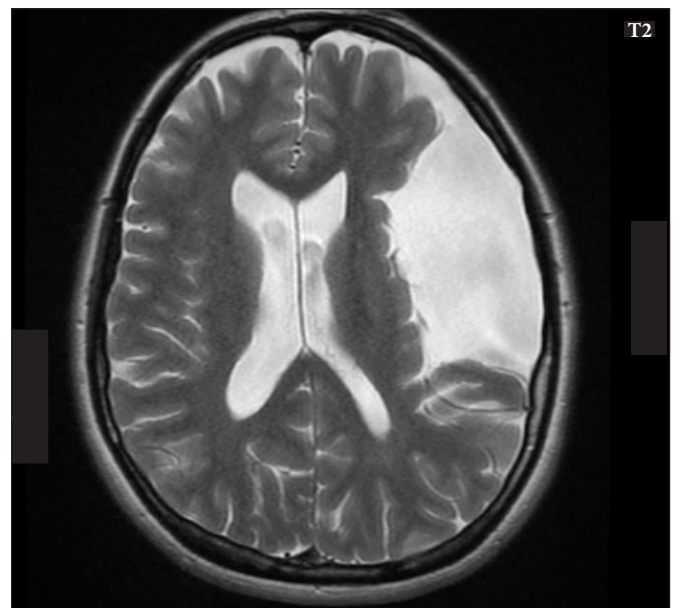
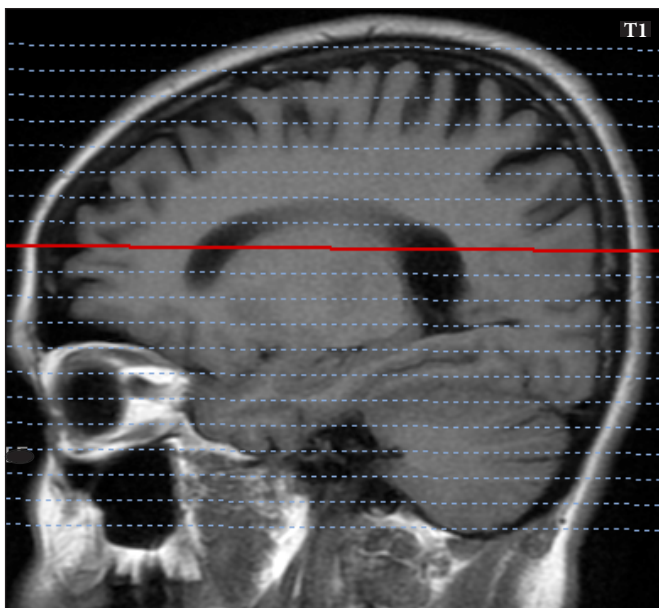
to a region, and had increased in severity during previous one year. Her nervousness, attention and focus problems had increased within the previous month. She complained of starting to forget names, having difficulty reading books and not being able to focus on what was spoken. She had difficulty in continuing the work she had started; her mind was very confused by having new ideas all the time. She

had difficulty falling asleep, her sleep duration decreased and her speech increased. She stated that her friends' circle decreased, and her social relationships deteriorated due to these complaints. The patient's mood was irritable but there were no signs of mania such as risky behaviour, increased goal-directed movements, increased energy, increased sexual desire, or excessive spending.

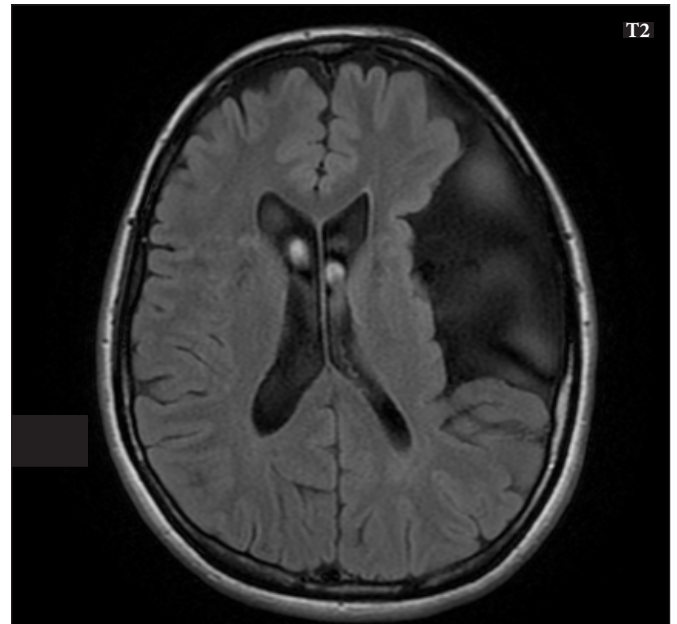
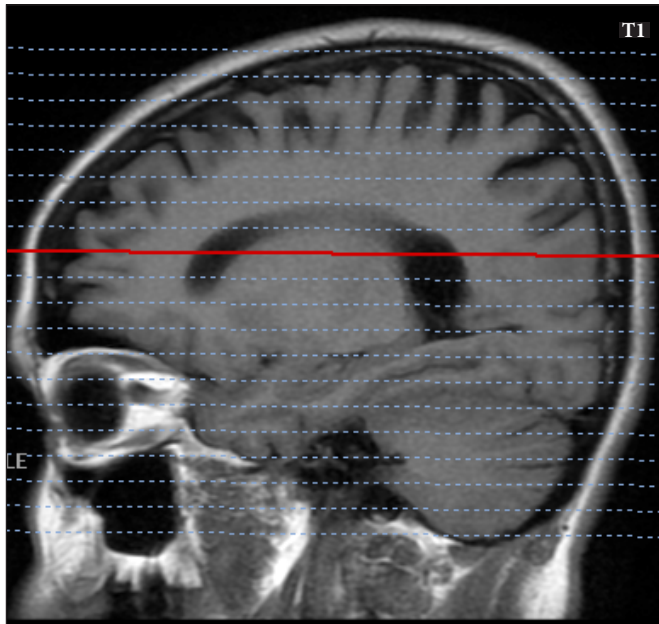
The patient's history included a period of intense pessimism after the death of her father ten years previously when she developed symptoms of decreased self-care, drowsiness, loss of appetite and avolition. Also, symptoms such as guilt and regret had almost reached the delusional dimension. It was learned that this depressive period lasted about one year, that the patient did not receive any medical support during this period and that his depression improved without medication in spring. The patient did not have a history of alcohol / substance abuse or any cranial trauma. She had been smoking approximately one pack of cigarettes a day for the past 17 years. It was learned that her family history did not include any psychiatric disorders. The patient had never consulted neurology clinics for long term headache. When magnetic resonance imaging (MRI) was performed on account of the increased headache and the psychiatric symptoms, a giant arachnoid cyst with dimensions of 5.5x10.5x12.5 cm was detected in the left cerebral hemisphere, starting from the inferior to the vertex level. There were hypoplastic changes in the temporal, parietal and frontal lobes adjacent to the AC. The cortical sulci and fissures were natural in other sections. The 4th ventricle was in the midline and the lateral ventricles



**Figure 1.** Giant arachnoid cyst located in the left hemisphere and frontal, temporal and parietal lobe hypoplasia adjacent to the cyst (T2 weighted coronal section)



**Figure 2.** Giant arachnoid cyst shown as co-appearance in T1-flair-weighted sagittal section and T2-weighted axial section.



**Figure 3.** Giant arachnoid cyst shown as co-appearance in T1 flair-weighted sagittal section and T2 flair axial section.

were in normal position and size. The intensity of the brain stem and cerebellum was homogeneous (Images 1, 2 and 3).

In her psychiatric examination, her appearance was consistent with her age and socioeconomic status. The patient was fully oriented. Her psychomotor activity was partially increased. Instant memory and remote memory were normal and near memory was partially reduced. Her spontaneous and voluntary attention had diminished. The amount and speed of speech were significantly increased. Her mood was irritable and her affect was labile. No perception defect was detected. Her speed of thought had increased. Her thought contents did not include delusions. Her judgment and ability to evaluate the truth were normal. Impulse control was partially reduced.

After these evaluations, the patient underwent the Rorschach test, the Minnesota Multidimensional Personality Inventory (MMPI) and a battery of neuropsychological tests. In the Rorschach test, the patient's reality test and ego strength were normal. It was found that she tried to cope with stressful situations through divergence. Affective symptoms were prominent in the test protocol. It was predicted that the MMPI profile of the patient could cause anger bursts and somatic complaints in case of verbal aggression and frustration. In the Stroop test, the information processing speed and attention functions of the patient were found to be below the standard level. In the Rey Auditory Verbal Learning test, the patient showed normal performance in the short-term and long-term memory functions (remembering

and recognition functions). This suggested that the patient had no problems with recording, learning and recalling information. In the Wisconsin card matching test, the patient demonstrated disorders such as not being able to change her mental strategy, tendency to show perseverative response, not paying attention to the selective aspect of the stimulus, not being able to distinguish critical features and classifying common features. When all test findings were evaluated together, the affective features were found to be dominant, the short-term memory and long-term memory functions were normal, but the ability to concentrate and maintain attention and executive functions were impaired.

Routine blood tests, including a hemogram, liver and kidney function tests, vitamin B12, folic acid, blood sugar levels, carried out to detect any organic pathology underlying the psychiatric symptoms and cognitive disorders were within the normal ranges.

The neurosurgery department was consulted for the treatment of the cyst. Since the cyst was quite large, follow-up with symptomatic drug treatment was planned before surgical treatment. We planned to start medication for the patient's hypomanic symptoms and cognitive impairment. However, we could not start treatment because the patient did not come to her next appointment. Despite attempts to contact the patient by phone or mail we did not receive any response.

**Table 1.** Cases with Arachnoid Cysts Accompanied by Cognitive Impairment

Author/Year	Year/ Gender	Admission symptom	Size	Localization	Psychiatric evaluation	Neuropsychological test results	Surgical treatment	Drug treatment	Status of symptoms after surgery / drug treatment
Geniş and Coşar / 2019	44/K	Headache, irritability and attention problems	55x105x125 mm	Left cerebral hemisphere	Hypomania and Cognitive Impairment	- Ability to change response, decreased processing speed and attention functions - Normal encoding (record), learning and recalling of information - deterioration in mental flexibility - Impairment of executive functions	No	No	-
Shettar et al. / 2018	56/K	Right side body and face weakness, memory disorders and depressive symptoms	-	Left frontal lobe	Cognitive Impairment and Depression	- Temporal, frontal and parietal lobe neurocognitive dysfunction - Reduction in phonemic and semantic verbal fluency - Distortion of mental flexibility and cognitive inhibition - Weakness in the area of selective attention - Lack of free recall in episodic memory	Yes	Sertaline 100 mg/day	-Improvement of all neurocognitive functions and depression
Mormont et al. / 2017	75/K	Parkinson's symptoms and cognitive impairment	95x65x58 mm	Right frontal lobe	Secondary Parkinsonism and Cognitive Impairment	-	Yes	Levodopa 600 mg/day	- Partial improvement in symptoms of Parkinsonism. - Significant improvement in verbal fluency
Miskey et al. / 2016	65/E	Memory impairment	164x77 mm	Left frontotemporal	PTSD and Depression	- Reduced visual learning and memory.	No	-	-
Zwageman et al. / 2016	49/K	Headache, double vision, facial numbness, memory impairment	30x25x17 mm	Left orbital	Cognitive Impairment and Depression	- Impairment of expressive language, decreased attention and cognitive flexibility, psychomotor slowdown	Yes	-	- Ongoing mild impairment of memory and verbal fluency - Significant improvement in other cognitive functions and depressive symptoms
Sugimoto et al. / 2016	72/E	Dizziness, gait disturbance and cognitive impairment	-	Fourth ventricle	Severe Dementia	HDSR=6/30	Yes	-	- Improvement in walking and cognitive capacity - Improvement of truncal ataxia -HDSR=21/30
B Gjerde et al. / 2013	46.4/E (12) 37.8/K (10)	Only headache in 13 patients Cognitive impairment only in 1 patient Dizziness in only one patient Epilepsy in only 1 patient Headache and cognitive impairment in 4 patients Headache and epilepsy in 1 patient Headache and dizziness in 1 patient	-	Left frontal lobe in 2 patients Left frontal lobe in 1 patient Left temporal lobe in 7 patients Left temporal lobe in 12 patients	Cognitive Impairment	Color-word interference test; STC Color naming = 33.6 + 6.9 Word reading = 25.3 + 4.7 Inhibition = 69.1 + 23.4 Inhibition / switching = 81.6 + 25.3 Verbal Fluency test; NWP Letter of Fluency = 36.7 + 10.6 Category fluency = 37.1 + 8.0 Category switching = 13.2 + 2.8 Total switching accuracy = 12.5 + 2.9 Tower test; TAS Total achievement score = 16.7 + 2.6	Yes	-	Color-word interference test; STC Color naming = 29.4 + 4.5 Word reading = 25.8 + 12.9 Inhibition = 56.4 + 14.5 Inhibition / switching = 68.9 + 14.7 Verbal fluency test; NWP Letter fluency = 55.0 + 10.3 Category fluency = 48.8 + 8.6 Category switching = 18.5 + 3.2 Total switching accuracy = 17.4 + 3.1 Tower test; TAS Total achievement score = 20.7 + 3.5
Boonkens et al. / 2010	81/K	Dizziness, short memory problems, difficulty finding words, mild depressive symptoms	62x43 mm	Left frontotemporal	Depression	- Short memory problems, decreased attention and executive function abnormalities	Yes	-	- Dizziness, short-term memory problems and improvement in depressive symptoms
Kuloglu et al. / 2008	22/K	Delusion, hallucination, memory and attention deficiencies, negative symptoms	50x50x40 mm	Left temporal lobe	Schizophrenia-like psychosis	- Attention and memory problems - BPRS; 69	No	Olanzapine 20 mg / day	- Complete recovery of hallucinations, psychomotor agitation, aggression and decrease in ambivalent behavior - No changes in delusions, attention and memory functions - BPRS:41
Koril et al. / 2007	70/K	Headache, dizziness, memory and behavior disorders	61x65x69 mm	Left frontal lobe	Cognitive Impairment	-VEM score = 45 points -VIM score = 55 points -GM score = 100 points -DR score = 85 points	Yes	-	-VEM score = 75 points -VIM score = 85 points -GM score = 155 points -DR score = 98 points -Improvement in behavior disorders
Lebowitz et al. / 2006	33/E	Frontal headache	-	Left front temporo-parietal	None*	- Reduction in fine motor skills and hand power - Partial disorder in word production - Impaired Spatial Thinking - Impaired Judgment of Spatial Orientation	No	-	-
Soukup et al. / 1998	20/E	Headache	150x35x25 mm	Left temporal fossa	Cognitive Impairment	-Abnormalities in expressive language including confrontation naming and verbal fluency -PIQ = 90	Yes	-	-Significant improvement in visual-perceptual abilities, structural skills, verbal learning / memory, conceptual shifting and psychomotor speed -PIQ = 117

\*Reported depression in her 20s. VEM: Verbal memory; VIM: Visual memory; GM: General memory; DR: Delayed recall; BPRS: Brief Psychiatric Rating Scale; PIQ: Performance Intelligence Quotient; HDSR: Hausgrwa Dementia Scale-Revised; STC: Seconds to complete the trial; NWP: Words produced in 60 seconds; TAS: Total achievement score



## DISCUSSION

With the widespread use of intracranial imaging, more ACs are detected but the prevalence and prognosis in adults are still poorly understood. This may be due AC being asymptomatic in significant number of cases.

ACs are rarely comorbid with psychiatric disorders and are usually diagnosed incidentally. The frequent reporting of psychotic disorders comorbid with psychiatric disorders (Kuloglu et al. 2008, Das et al. 2017, Khan and Ahmed 2017) may be due to increasing use of imaging methods in psychotic disorders and the frequent location of ACs in the left temporal region (Al-Holou et al. 2013). There is much evidence to suggest a clear association between temporal lobe structural abnormalities and psychotic symptoms (Shenton et al. 1992). Therefore, ACs may be more common with psychotic disorders. Our case had a giant AC in the left cerebral hemisphere. Although there was no psychotic symptom in her last examination, she had experienced major depression in the past with symptoms of borderline psychotic disorder. When consulting us she was having a hypomanic episode.

Apart from psychotic disorders, psychiatric disorders accompanying AC such as depression (Boomkens et al. 2010), catatonia (Kastenholz et al. 2014), panic disorder (de-Melo-Neto et al. 2009) and obsessive-compulsive disorder (Hegde et al. 2014) etc. have also been reported in the literature. Bipolar affective disorder (BAD) is very rarely associated with AC, there being only four reported cases (Hanretta et al. 2007, Bakım et al. 2012, Harmancı and Gökbakan 2013, Khan and Ahmed 2017). Arachnoid cysts were located in the right anterior temporal lobe in two of the four cases, in the left frontal lobe in one case and in the occipital lobe in the other case. In the case of arachnoid cyst located in the occipital lobe, it was reported that the cyst pressed against the right hemisphere. In harmony with these cases, it is seen in the literature that mania symptoms are related to right hemisphere lesions, especially with thalamic, temporal lobe and the frontal lobe involvement (Schmid-Ghika and Bogousslavsky 2000). In our case, the giant AC almost covered the left cerebral hemisphere. There was marked hypoplasia in the frontal, temporal and parietal lobes adjacent to the lesion. However, the right hemisphere was normal. It is not possible to relate the occurrence of psychiatric symptoms to lesion localization alone. Because the interaction of neural networks in the brain region where the lesion compresses is also very important in the formation of symptoms.

One of the important differences of our case from other BAB cases accompanied by AC is the observation of marked cognitive disorders. In cases followed by Khan and Ahmed (2017) and Bakım et al. (2012), attention, focus and memory functions were evaluated as normal. In the cases followed by Harmancı and Gökbakan (2013) and Hanretta et al. (2007), information regarding the evaluation of cognitive symptoms was not given. Cognitive impairment may be overlooked without a detailed neuropsychological assessment. In a study on ten patients with AC located in different parts of the brain, the cognitive disorders experienced by nine of these patients could not be clearly identified until neuropsychological tests were performed (Lang et al. 1985). In the literature, cognitive disorders associated with AC vary widely. Reduction in memory functions (Miskey and Gross 2016), short-term and long-term memory loss (Boomkens et al. 2010), decrease in verbal fluency (Mormont et al. 2017) and naming impairment (Soukup et al. 1998) are some of these. There are also cases in which a significant portion of cognitive functions are preserved despite very large AC dimensions (Lebowitz et al. 2006). Although short-term and long-term memory functions were normal in our case, we found that the focusing, sustaining attention and executive functions were impaired. One of the main causes of cognitive impairment in our patient may be the AC or the hypomania. It has been reported that deficits related to verbal memory and executive functions are present even in euthymic patients who are not having episodes (Demirel et al. 2012). It has been reported that other neurocognitive deficits also increase, especially the distractibility, which is considered as a diagnostic criterion during the attack period. In the case discussed here, although the symptoms of cognitive impairment that had increased in the previous one month could have been more related to the hypomanic episode, significant disruptive effects of a giant AC on cognitive functions cannot be overlooked. Cases with cognitive impairment comorbid with AC reported in the literature are shown in Table 1 (Kotil et al. 2007, Kuloglu et al. 2008, Boomkens et al. 2010, Gjerde et al. 2013, Miskey and Gross 2016, Sugimoto et al. 2016, Zwagerman et al. 2016, Mormont et al. 2017, Shettar et al. 2018). In most of the reported cases, the AC was located in the left part of the brain. Although this suggests that there may be a relationship between cyst position and cognitive impairment, this is not clear. A cross-sectional study using functional MRI has suggested that the right hemisphere is more important on attention function (Rosen et al. 1999). In a prospective study, however, it was reported that patients with AC had

intelligence and memory test scores similar to those in the normal population, and the results were not related to cyst localization (Kunz et al. 1988).

Based on case reports, it is seen that the risk of cognitive impairment increases in individuals with AC (Table 1). Most of the disorders in cognitive function seem to be reversible after surgical treatment (Soukup et al. 1998, Kotil et al. 2007, Boomkens et al. 2010, Gjerde et al. 2013, Sugimoto et al. 2016, Zwagerman et al. 2016, Mormont et al. 2017, Shettar et al. 2018). Surgical treatment of ACs can improve not only the clinical symptoms and complaints, but also the cognitive impairment. However, it should be remembered that the outcomes are based on case reports and surgical treatments may cause significant complications. The fact that we could not follow our case is a significant limitation. However, the size of the cyst in the patient with a diagnosis of hypomania and the detailed neuropsychological evaluation is expected to make a significant contribution to the literature.

## CONCLUSION

The causality relationship between ACs and psychiatric disorders is not always clear and paucity of case reports increases the uncertainty. ACs can cause serious psychiatric disorders and cognitive deficits. A multidisciplinary approach should be adopted at all stages of treatment. Future studies evaluating cognitive functions prospectively in individuals with ACs will further clarify this relationship.

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