

# Anterior Communicating Artery Aneurysm With Refractory Central Vomiting: A Case Report

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## Abstract

**Introduction:** Anterior communicating artery aneurysm produces symptoms by compression of adjacent neural structures. It can cause nausea and vomiting because of direct compression or destruction of the hypothalamus.

**Case Presentation:** We described a 59-year-old man with refractory nausea and vomiting. He was evaluated completely for this problem yet no gastrointestinal pathology was found. Gastroenterologic examination was normal and he was referred to our center for neurological evaluation. We found anterior communicating artery aneurysm in his brain magnetic resonance imaging (MRI). This finding was confirmed by digital subtraction angiogram (DSA). It was decided to coil the aneurysm. After aneurysm embolization, the patient's symptoms were surprisingly improved completely.

**Conclusions:** We hereby report refractory vomiting as a rare presentation of un-ruptured anterior communicating artery aneurysm. This may be explained by a direct compressive effect of the aneurysm on adjacent structures.

**Keywords:** Refractory Vomiting, Anterior Communicating Artery Aneurysm, Aneurysm Coiling

## 1. Introduction

Endocrine disturbance and vomiting may be provoked by compression of hypothalamus and hypothalamic-pituitary complex by the compression effect; this compression may be due to tumors or rarely aneurysms. This is particularly true for anterior communicating artery (AcomA) because arteries derived from this vessel supply a portion of the hypothalamus (1). It is also known that vomiting can be elicited by stimulation of the supraoptic area of the hypothalamus. We herein report a rare case of anterior communicating aneurysm with refractory vomiting.

## 2. Case Presentation

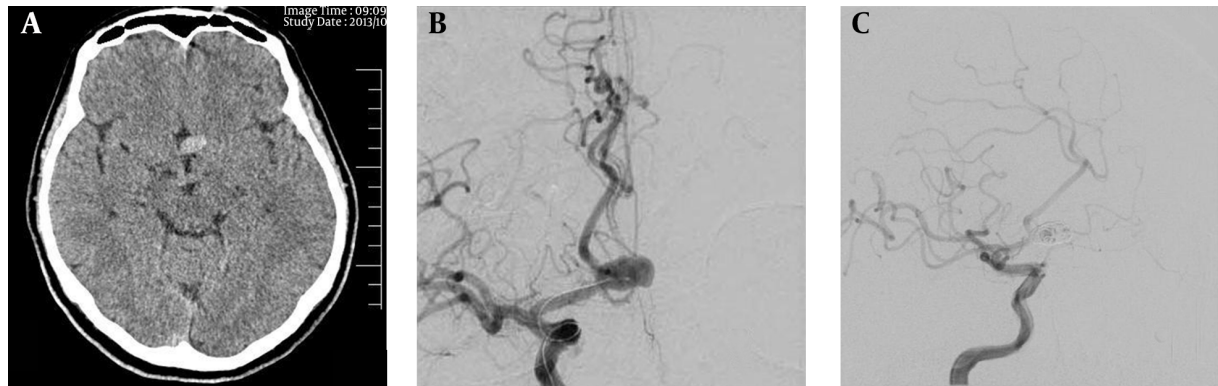
A 59-year-old man was admitted to the neurology ward of Firoozgar hospital affiliated to Iran University of Medical Sciences, in November 2013 with a six-month-history of refractory nausea and vomiting. He did not report any other accompanying symptoms during this period. He was a retired man and his past medical and drug history were not significant. He was a smoker but denied use of alcohol or illicit drugs.

His physical and neurological exams were normal. The blood investigations revealed normal hematologi-

cal, biochemical and hormonal profile. Endoscopy and colonoscopy had been performed before referral to this center; reported to be normal. Abdominopelvic computed tomography (CT) scan with oral and intravenous contrast showed no evidence of abnormality. Unenhanced brain CT scan showed a (12 × 7 mm) hyperdense suprasellar lesion. Brain MRI suggests that the lesion could be an AcomA aneurysm. Digital subtraction angiography (DSA) confirmed the findings of imaging and revealed saccular aneurysm of AcomA. It was decided to treat the aneurysm by the endovascular approach. The aneurysm was completely obliterated by coiling. The operation was successful and interestingly, his symptoms were resolved immediately after the embolization. During the follow up, after six months, he did not experience nausea and vomiting any more.

## 3. Discussion

Twenty-five to thirty-eight percent of all cases of cerebral aneurysms occur in AcomA, making it the most common site among intracranial arteries (2). In the setting of un-ruptured aneurysm, the symptoms arise from either the mass effect of a large aneurysm over the surrounding tissues, or possibly from minimal leakage of blood irritating the meninges (3). Nevertheless, occurrence of large



**Figure 1.** A, Brain computerized tomography scan showed a hyperdense suprasellar lesion (arrow); B, digital subtraction angiography (DSA) revealed a saccular aneurysm of the anterior communicating artery (A1 - A2 junction); C, anterior communicating aneurysm after coil embolization (arrow).

AcomA aneurysms is extremely rare because they are generally susceptible to rupture while they are relatively small (4). Thus, an AcomA aneurysm is expected to cause symptoms due to hemorrhage (mainly subarachnoid hemorrhage) rather than compression effects. According to the literature, an un-ruptured AcomA may present, through its pressure effect, in different ways, with visual disturbance (4-6) being the most common reported presentation, followed by central vomiting, hypopituitarism (7), and cognitive impairment (5). Our patient presented chronic refractory nausea and vomiting without any other accompanying symptom such as headache, which to the best of our knowledge, is a rare presentation of an un-ruptured AcomA and we could not find any other similar report in the literature. Neurons, in the para-ventricular nucleus of the hypothalamus, project to the preganglionic neurons of the sympathetic and parasympathetic divisions in the brainstem and spinal cord. These cells exert hypothalamus control over the visceral motor system and modulate autonomic reflexes such as respiration and vomiting. Compression of these cells by the aneurysm could be responsible for symptoms of intractable nausea and vomiting in our patient (8). In this setting, we expect the occurrence of symptoms after aneurysm treatment.

### 3.1. Conclusion

The aneurysm of anterior communicating artery may present itself through its pressure effect over the adjacent cerebral tissues, and not through hemorrhage. In such a setting, the patient may experience chronic refractory vomiting, which is expected to response to the treatment.

### Footnotes

**Authors' Contribution:** Masood Mehrpour and Leila Hashami developed the original idea and the protocol, abstracted and analyzed the data, wrote the manuscript and were the guarantors. Elham Ghaebi and Fatemeh Taherian contributed to the development of the protocol, abstracted the data and prepared the manuscript.

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