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Probable Hair-Grooming Syncope in a Young Adult With an Incidental Extra-Axial Quadrigeminal Cistern Lesion Radiologically Consistent With an Epidermoid Cyst: A Case Report

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Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
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Patient: Male, 22-year-old
Final Diagnosis: Probable hair-grooming syncope with an incidental quadrigeminal cistern lesion radiologically consistent with an epidermoid cyst
Symptoms: Syncope • dizziness • pallor
Clinical Procedure: —
Specialty: Neurology • Neurosurgery
Objective: Rare disease
Background: Hair-grooming syncope is a rare form of situational reflex syncope, reported mainly in children and adolescents. Routine neuroimaging is not recommended in uncomplicated syncope with a normal neurological examination but can reveal incidental findings.
Case Report: A previously healthy 22-year-old man had 2 consecutive syncopal episodes, preceded by dizziness, during hair washing at a barbershop with forward neck flexion. Vital signs, neurological examination, electrocardiography, laboratory tests, and cranial computed tomography were normal; orthostatic blood pressure was not measured. Brain magnetic resonance imaging showed a 2.5-cm extra-axial lesion in the quadrigeminal cistern with marked diffusion restriction, low apparent diffusion coefficient values, incomplete suppression on fluid-attenuated inversion recovery images, no contrast enhancement, and no hydrocephalus or mass effect, radiologically consistent with an epidermoid cyst. The presentation was considered most consistent with probable situational reflex syncope, and the lesion likely incidental, although a causal contribution could not be definitively excluded. Surgical resection was recommended; its rationale was not documented. The patient declined surgery and left against medical advice; therefore, histopathological confirmation was unavailable. At 6-month telephone follow-up, he reported no recurrent syncope or new neurological symptoms but had not undergone neurosurgical, imaging, or cardiac follow-up.
Conclusions: Hair-grooming syncope should be considered when syncope is temporally related to grooming and the neurological examination is normal. Routine neuroimaging has a low yield in uncomplicated syncope; when an intracranial lesion is detected, its causal relevance should be judged by anatomical and clinical plausibility rather than its mere presence.

Keywords: Syncope, Vasovagal • Epidermoid Cyst • Diffusion Magnetic Resonance Imaging • Incidental Findings • Emergency Service, Hospital
Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/953337

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Introduction

Syncope is a common reason for emergency department (ED) visits and hospital admission, accounting for approximately 1-3% of ED presentations and up to 6% of hospital admissions [1]. Hair-grooming syncope is an uncommon form of situational reflex syncope triggered by activities such as hair combing, brushing, cutting, styling, or washing [2,3]. Published cases have predominantly involved children and adolescents, particularly girls, whereas occurrence in a healthy young adult man is unusual [2,3]. Although hair-grooming syncope is generally regarded as a benign neurally mediated phenomenon, its precise pathophysiological mechanism remains uncertain.

Current ACC/AHA/HRS and ESC guidelines recommend that syncope evaluation be guided primarily by a detailed clinical history, physical examination, orthostatic blood pressure measurement, and 12-lead electrocardiography. Routine cranial computed tomography (CT) or brain magnetic resonance imaging (MRI) is not recommended in patients with uncomplicated syncope and a normal neurological examination unless neurological disease or trauma is clinically suspected [4,5].

Intracranial epidermoid cysts are rare, benign congenital lesions that usually arise in extra-axial cerebrospinal fluid spaces. They most commonly occur in the cerebellopontine angle and parasellar region, although less common sites, including the quadrigeminal cistern, have also been described [6]. On MRI, classical epidermoid cysts typically demonstrate incomplete suppression on fluid-attenuated inversion recovery sequences and marked diffusion restriction with relatively low apparent diffusion coefficient values, often allowing a confident radiological diagnosis even in the absence of histopathological confirmation [6,7].

We report a case of a 22-year-old man with 2 consecutive syncopal episodes most consistent with probable hair-grooming syncope, in whom neuroimaging disclosed an apparently incidental extra-axial lesion radiologically consistent with an epidermoid cyst, centered within the quadrigeminal cistern and abutting the posterior aspect of the mesencephalon. This case highlights the diagnostic reasoning required to distinguish a coincidental neuroimaging abnormality from a clinically plausible explanation for syncope and to avoid attributing causality solely on the basis of temporal discovery.

Case Report

A 22-year-old previously healthy man presented to the ED approximately 30 minutes after experiencing 2 consecutive syncopal episodes while having his hair washed by a barber with his neck in forward flexion. Both episodes were preceded by dizziness and pallor. According to his accompanying friend, who witnessed the events, the patient transiently lost consciousness without convulsive movements, tongue biting, urinary incontinence, or prolonged postictal confusion. Each episode lasted approximately 1 minute and was followed by spontaneous and complete recovery. The interval between the 2 syncopal episodes was approximately 5-6 minutes, and both occurred during the same hair-washing session.

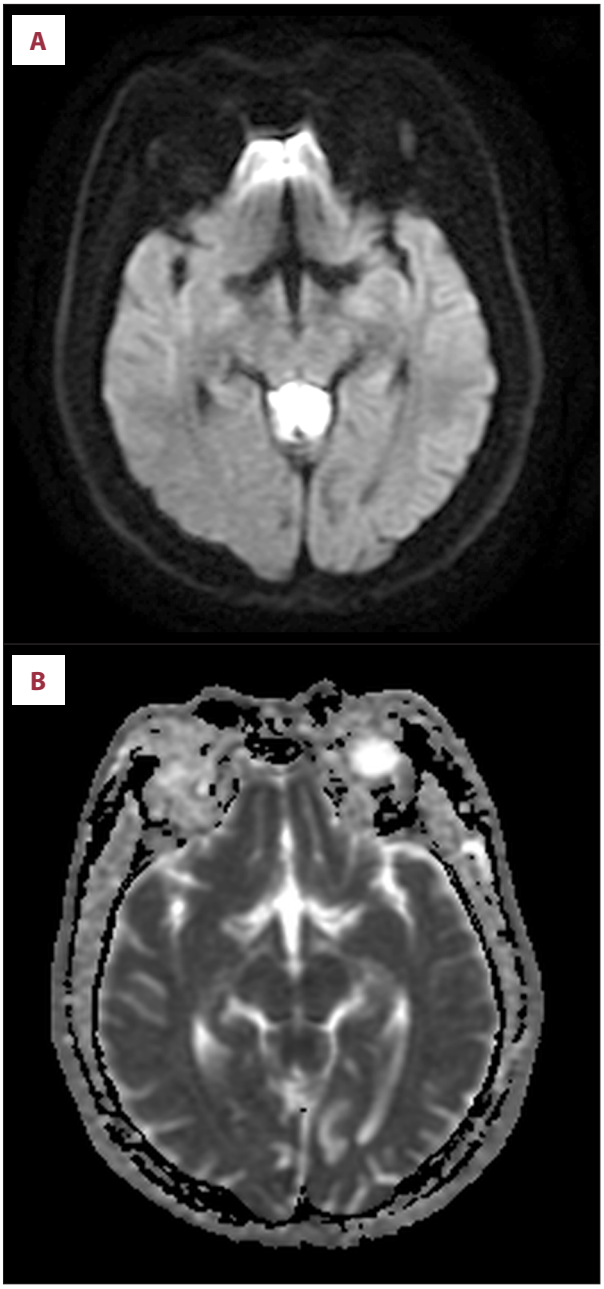
The patient reported no previous history of syncope. He denied chest pain, palpitations, dyspnea, headache, fever, recent infection, alcohol or illicit drug use, dehydration, prolonged fasting, or sleep deprivation. His medical history was unremarkable, and he was not taking any regular medications. There was no known family history of cardiovascular disease, epilepsy, sudden cardiac death, unexplained drowning, or early-onset arrhythmia.

On arrival, blood pressure was 110/75 mm Hg, heart rate was 93 beats/min, respiratory rate was 16 breaths/min, body temperature was 36.4 °C, oxygen saturation was 95% on room air, and capillary blood glucose was 93 mg/dL. Orthostatic blood pressure and heart rate measurements were not obtained during the initial ED evaluation; because orthostatic assessment is a recommended component of the initial syncope evaluation [4,5], the initial assessment was therefore incomplete. The patient was alert, fully oriented, and cooperative, with a Glasgow Coma Scale score of 15. Cardiovascular, respiratory, abdominal, and neurological examinations were unremarkable. Cranial nerve examination, motor strength, sensory examination, coordination, and cerebellar testing were normal, with no focal neurological deficits. A 12-lead electrocardiogram demonstrated normal sinus rhythm at 93 beats/min, with a PR interval of 130 ms, QRS duration of 66 ms, QT interval of 338 ms, and corrected QT interval (QTc, Bazett) of 420 ms. No evidence of pre-excitation, Brugada pattern, epsilon waves, conduction abnormalities, or significant ST-segment or T-wave changes was identified. Routine laboratory investigations, including complete blood count, serum biochemistry, electrolytes, renal and liver function tests, and high-sensitivity cardiac troponin, were all within normal limits. Because the clinical assessment, electrocardiographic findings, and cardiac biomarkers were normal, cardiology consultation was not considered necessary during the ED evaluation. No echocardiography, prolonged telemetry or Holter monitoring, or tilt-table testing was performed.

Non-contrast cranial CT demonstrated no evidence of acute intracranial hemorrhage or other acute intracranial abnormalities. Because the patient experienced 2 consecutive syncopal episodes and the initial ED evaluation did not identify an underlying cause, MRI was performed to exclude structural intracranial pathology.

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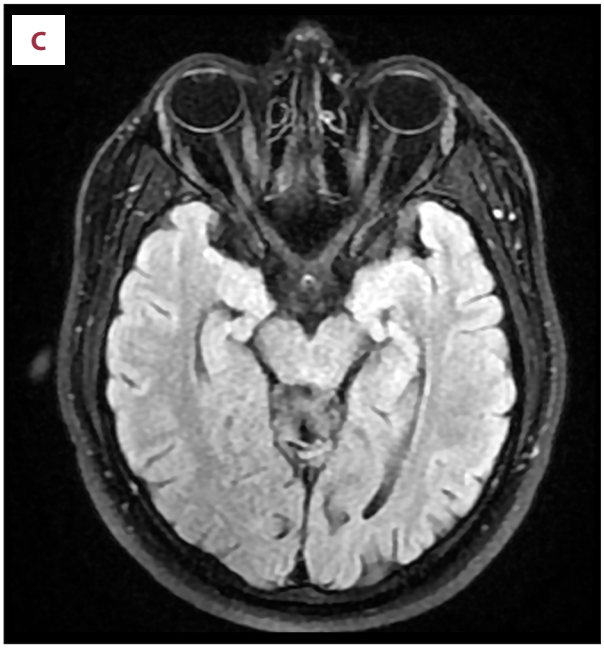


Figure 1. Axial brain magnetic resonance imaging at the level of the quadrigeminal cistern. (**A**) Diffusion-weighted imaging (b = 1000 s/mm^2) demonstrates marked hyperintensity of the extra-axial lesion located posterior to the mesencephalon. (**B**) Corresponding apparent diffusion coefficient map shows low signal within the lesion, similar to brain parenchyma and markedly lower than cerebrospinal fluid, confirming true diffusion restriction. (**C**) Axial fluid-attenuated inversion recovery image demonstrates heterogeneous, incomplete suppression of the lesion relative to cerebrospinal fluid. These features are consistent with, but not diagnostic of, an epidermoid cyst.

MRI was performed on a 1.5-T scanner using fluid-attenuated inversion recovery (FLAIR), three-dimensional (3D) T2-weighted imaging, diffusion-weighted imaging (DWI; b = 1000 s/mm^2), pre-contrast 3D T1-weighted BRAVO (brain volume), gadolinium-enhanced post-contrast 3D T1-weighted BRAVO, magnetic resonance angiography (MRA), magnetic resonance venography (MRV) sequences, and proton magnetic resonance spectroscopy (MRS).

Brain MRI demonstrated a well-circumscribed 2.5-cm extra-axial cystic lesion centered within the quadrigeminal cistern and abutting the posterior aspect of the mesencephalon. The lesion demonstrated incomplete suppression on FLAIR imaging, marked diffusion restriction on DWI with corresponding low apparent diffusion coefficient (ADC) values, similar to brain parenchyma and markedly lower than cerebrospinal fluid (**Figure 1**), and no pathological gadolinium enhancement (**Figure 2B-2D**). Sagittal 3D T2-weighted images confirmed its extra-axial location within the quadrigeminal cistern (**Figure 2A**). There was no evidence of hydrocephalus, aqueductal obstruction, significant mass effect, or vascular compression. MRA and MRV demonstrated no vascular abnormalities, with no displacement or compression of the posterior circulation arteries or the dural venous sinuses (**Figure 3**), and MRS showed nonspecific findings. Based on these imaging findings, the lesion was considered radiologically consistent with an epidermoid cyst; histopathological confirmation was not obtained. All MRI examinations, together with the figures and their legends, were subsequently reviewed by a neuroradiologist, who confirmed the lesion characterization, namely an extra-axial lesion within the quadrigeminal cistern radiologically consistent with an epidermoid cyst without hydrocephalus, aqueductal obstruction,

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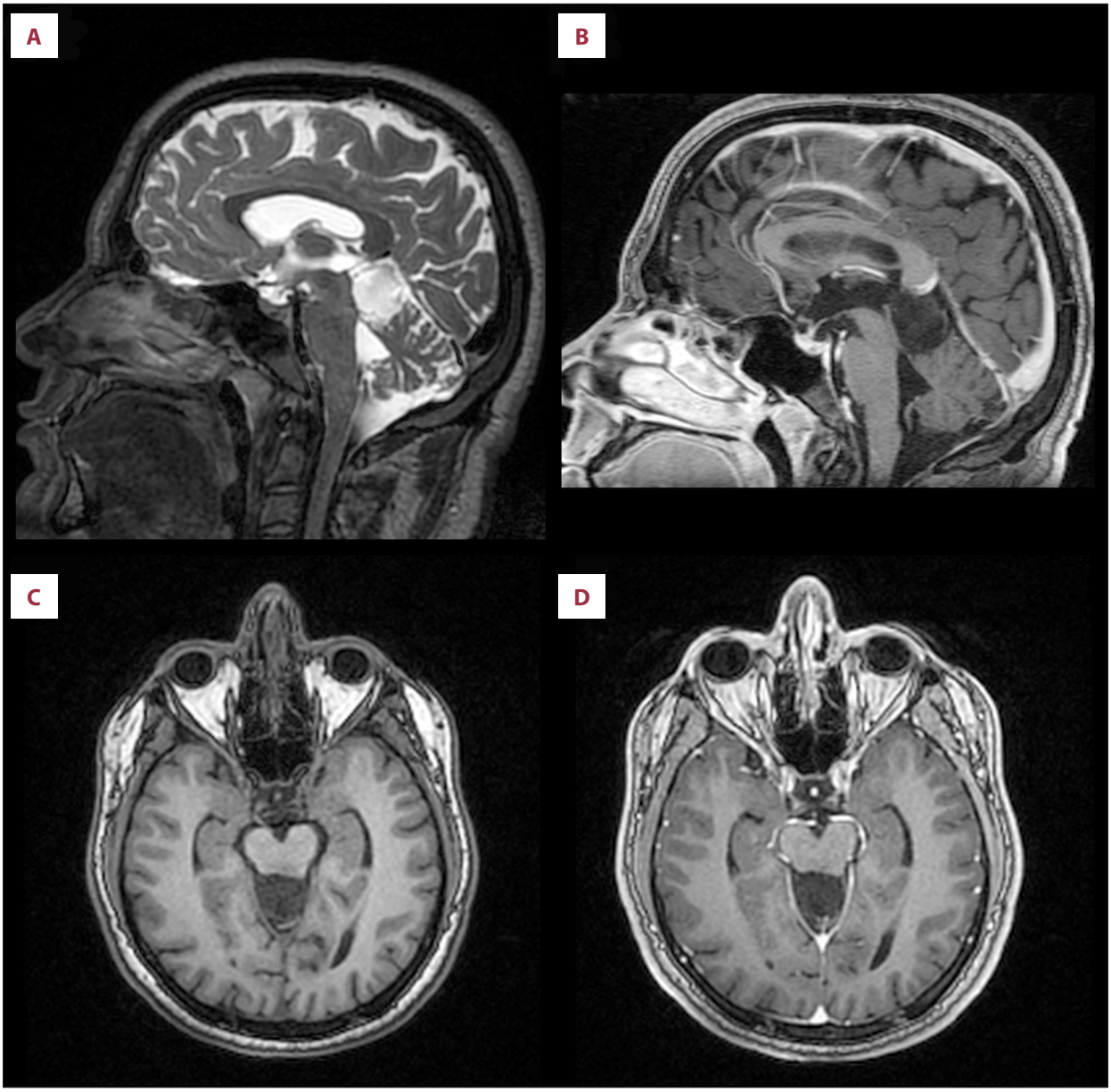


Figure 2. Sagittal and contrast-enhanced magnetic resonance imaging. (**A**) Midsagittal 3D T2-weighted image demonstrates a well-circumscribed hyperintense extra-axial lesion within the quadrigeminal cistern, posterior to the tectal plate, without hydrocephalus. (**B**) Midsagittal gadolinium-enhanced 3D T1-weighted image shows the hypointense lesion without pathological enhancement. (**C**) Axial pre-contrast and (**D**) corresponding post-contrast 3D T1-weighted (BRAVO) images at the same level demonstrate a hypointense lesion without pathological enhancement.

significant mass effect, or vascular compression, as well as the consistency between the figures and the manuscript text.

Although the lesion was anatomically adjacent to the posterior mesencephalon, neither the clinical findings nor the neuroimaging features supported a direct structural cause of the syncopal episodes. Specifically, there was no imaging evidence of brainstem compression, obstructive hydrocephalus, increased intracranial pressure, or vascular compromise. The lesion was therefore considered likely incidental, although a causal contribution could not be definitively excluded, and the clinical presentation was considered most consistent with probable situational reflex syncope associated with hair washing.

Neurosurgical consultation was obtained, and the patient was admitted to the neurosurgery service for further evaluation and management. The neurosurgery service recommended microsurgical resection. The specific clinical or radiological rationale

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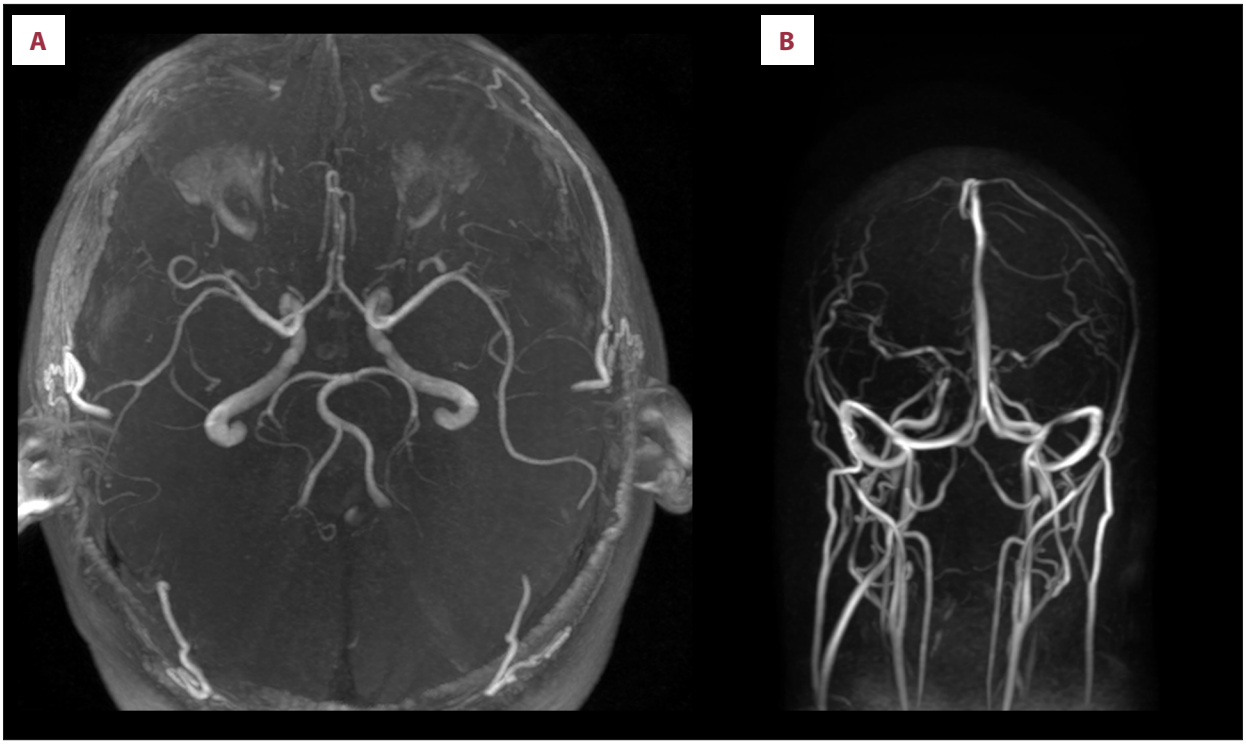


Figure 3. Magnetic resonance angiography and venography. (**A**) Axial maximum intensity projection image of three-dimensional time-of-flight magnetic resonance angiography and (**B**) coronal maximum intensity projection image of three-dimensional phase-contrast magnetic resonance venography. No displacement or compression of the arterial structures of the posterior circulation or of the dural venous sinuses is observed.

for this recommendation was not documented in the available medical record and is therefore not reconstructed here. Following detailed counseling regarding the potential benefits and risks of surgical and conservative management, the patient declined surgical intervention. The following day, he left the hospital against medical advice after signing the relevant documentation. Outpatient neurosurgical follow-up was recommended. Because surgery was not performed, histopathological confirmation of the lesion was unavailable.

At approximately 6-month telephone follow-up, the patient reported no recurrence of syncope and only occasional mild episodes of dizziness. He remained clinically well and continued his usual daily activities without any neurological symptoms or functional limitations. He had not attended the recommended outpatient neurosurgical follow-up and had not undergone repeat neuroimaging. He had also not sought cardiology evaluation or undergone any additional cardiac investigations during the follow-up period.

Discussion

The most distinctive feature of the present case is the occurrence of probable hair-grooming syncope in a 22-year-old man, as most previously reported cases have involved children or adolescents, with a marked female predominance [2,3,8]. This atypical demographic profile warrants caution in applying the diagnostic label solely on the basis of the triggering activity. The case was further complicated by the incidental detection of an extra-axial lesion radiologically consistent with an epidermoid cyst, centered within the quadrigeminal cistern. Although its proximity to the dorsal midbrain initially raised concern regarding a possible causal relationship, the absence of hydrocephalus, brainstem compression, vascular compromise, or other structural abnormalities argued against a direct mechanistic association.

Hair-grooming syncope is generally considered a subtype of neurally mediated (reflex) syncope; however, its precise pathophysiological mechanism has not been fully elucidated [2,3,8]. Several hypotheses have been proposed, including excessive stimulation of scalp sensory nerves during hair manipulation, activation of trigeminal or upper cervical afferent pathways, and subsequent autonomic reflex responses resulting in transient vagal activation and sympathetic withdrawal [9,10]. These physiological changes can produce peripheral vasodilation, bradycardia, or both, ultimately leading to transient cerebral hypoperfusion and loss of consciousness [9,10]. Nevertheless, no single mechanism has been consistently demonstrated, and the

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underlying pathophysiology is likely multifactorial. The reproducible association with specific grooming activities and the absence of structural neurological abnormalities in the vast majority of reported patients continue to support the concept of a benign situational reflex rather than an intrinsic neurological disorder [2,3,8].

Although reflex-mediated mechanisms provide the most plausible explanation for the syncopal episodes observed in our patient, the incidental intracranial lesion required careful diagnostic consideration. The lesion was located within the quadrigeminal cistern immediately posterior to the mesencephalon, raising an initial concern regarding a potential structural contribution to syncope. However, several radiological and clinical findings argued against such a relationship. The lesion was extra-axial, demonstrated no significant mass effect, caused neither aqueductal obstruction nor hydrocephalus, and showed no evidence of brainstem compression or vascular compromise. Furthermore, the patient had a normal neurological examination without focal deficits or other manifestations suggestive of symptomatic brainstem dysfunction. Collectively, these findings suggest that the lesion was most likely an incidental imaging finding rather than the underlying cause of the patient's syncopal episodes, although a causal contribution cannot be definitively excluded.

Current international syncope guidelines consistently emphasize that the initial evaluation should be guided primarily by a detailed clinical history, physical examination, orthostatic blood pressure measurements, and a standard 12-lead electrocardiogram [4,5]. In patients with uncomplicated syncope and a normal neurological examination, routine cranial CT or brain MRI is generally discouraged because of its low diagnostic yield and limited impact on clinical management [4,5]. In the present case, the clinical history was suggestive of situational reflex syncope, and neurological examination, electrocardiography, laboratory investigations, and cranial CT were all unremarkable; however, orthostatic measurements were not obtained, so the initial guideline-recommended assessment was incomplete. Therefore, according to current guideline recommendations, the likelihood of identifying a clinically significant intracranial structural cause of syncope was expected to be very low.

Despite these recommendations, clinical decision-making in the ED often requires integration of guideline-based practice with physician judgment and individual patient characteristics. In our patient, brain MRI was obtained because 2 consecutive syncopal episodes occurred within a short period and no definitive explanation was established after the initial ED assessment. Although MRI unexpectedly revealed an intracranial lesion, the radiological characteristics and accompanying clinical findings did not support a causal relationship between the lesion and the syncopal episodes. In retrospect, given the normal neurological examination and absence of established high-risk features, brain MRI would not have been routinely indicated according to current syncope guidelines [4,5]. The incidental discovery in this case therefore illustrates one of the potential consequences of extending neuroimaging beyond guideline-supported indications.

The present case also highlights an important challenge frequently encountered in emergency medicine: balancing adherence to evidence-based guidelines with individualized clinical decision-making. While indiscriminate neuroimaging can increase healthcare costs and identify clinically irrelevant abnormalities, selective imaging based on persistent diagnostic uncertainty can occasionally reveal unexpected findings requiring further evaluation [11,12]. Consequently, neuroimaging results should always be interpreted within the broader clinical context rather than being viewed in isolation. Careful clinicoradiological correlation remains essential to distinguish incidental abnormalities from clinically significant pathology and to avoid inappropriate attribution of symptoms to unrelated imaging findings.

The widespread availability of advanced neuroimaging has substantially increased the detection of incidental intracranial abnormalities during the evaluation of patients presenting with unrelated neurological or non-neurological problems. Although these unexpected findings may initially appear clinically significant, their relevance to the presenting symptoms may be uncertain and should be interpreted cautiously within the overall clinical context [11,12]. Failure to distinguish temporal association from true causality can lead to unnecessary specialist consultations, additional diagnostic procedures, increased healthcare costs, patient anxiety, and potentially avoidable interventions. In the present case, the incidental MRI finding directly prompted neurosurgical consultation, hospital admission, and consideration of microsurgical treatment, illustrating how an unexpected imaging abnormality can initiate a cascade of further healthcare utilization even when its relationship to the presenting symptom remains uncertain.

Intracranial epidermoid cysts are rare benign congenital lesions that arise from ectodermal inclusions during neural tube closure and account for approximately 0.2-1.8% of all intracranial tumors [6,7]. They are characteristically slow-growing and frequently remain asymptomatic for many years until they become sufficiently large to compress adjacent neural structures [6,7]. The cerebellopontine angle is the most common site of intracranial epidermoid cysts, whereas the quadrigeminal cistern is an uncommon location, with only isolated case reports and small case series described in the literature [6,7,13,14]. Clinical manifestations are therefore largely determined by lesion location and the degree of mass effect rather than by

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the histological nature of the lesion itself. Symptomatic lesions have been reported to present with diverse neurological manifestations, including hydrocephalus, headache, cranial nerve dysfunction, cerebellar symptoms, mutism, and signs of increased intracranial pressure [13-15]. None of these findings were present in our patient.

MRI plays a central role in the diagnosis of epidermoid cysts because their imaging characteristics are highly distinctive. Classical epidermoid cysts typically demonstrate signal intensity similar to cerebrospinal fluid on conventional T1- and T2-weighted images but exhibit incomplete suppression on FLAIR sequences together with marked diffusion restriction and low ADC values because of their keratinaceous contents [6,7]. These imaging characteristics are particularly useful in distinguishing epidermoid cysts from arachnoid cysts, which follow cerebrospinal fluid signal intensity on all sequences and do not demonstrate restricted diffusion [6,7]. However, restricted diffusion is not specific to epidermoid cysts, and other diffusion-restricting lesions, particularly intracranial abscesses and selected cystic neoplasms, should be considered in the appropriate clinical context [16]. Additional differential diagnostic considerations include dermoid cysts and neurenteric cysts [17]. Dermoid cysts are typically distinguished by their fat-containing components and characteristic T1 hyperintensity, whereas neurenteric cysts generally demonstrate variable T1 and T2 signal characteristics related to their proteinaceous content and usually lack the marked diffusion restriction characteristic of epidermoid cysts [17]. Extraparenchymal neurocysticercosis, particularly the racemose form, can also involve the subarachnoid cisterns and should therefore be considered in the differential diagnosis of cisternal cystic lesions; however, the solitary well-circumscribed lesion with marked diffusion restriction, low ADC values, incomplete FLAIR suppression, and the overall characteristic MRI appearance favored an epidermoid cyst in the present case [18]. Cystic neoplasms can be differentiated by the presence of solid components, contrast enhancement, and/or surrounding edema [16]. In the present case, the combination of FLAIR hyperintensity relative to cerebrospinal fluid, pronounced diffusion restriction, absence of contrast enhancement, and characteristic anatomical location supported a radiological diagnosis of an epidermoid cyst; however, in the absence of histopathological confirmation, this diagnosis remains presumptive.

An important educational message emerging from this case is the risk of diagnostic anchoring, a cognitive bias in which clinicians place disproportionate weight on an initial impression or salient diagnostic finding and insufficiently adjust their diagnostic reasoning when subsequent information becomes available [19]. The incidental discovery of an intracranial lesion during syncope evaluation can understandably increase the temptation to assume a causal relationship, particularly when the lesion is anatomically adjacent to structures involved in autonomic regulation. However, causality should not be inferred solely from temporal coexistence or anatomical proximity. Instead, radiological abnormalities must always be interpreted together with the patient's clinical history, neurological examination, objective imaging findings, and subsequent clinical course. In our patient, the history suggestive of hair-grooming syncope, the absence of focal neurological deficits, and the lack of radiological evidence of brainstem compression, obstructive hydrocephalus, or vascular compromise collectively suggested that the lesion was most likely an incidental finding rather than the primary cause of syncope. At approximately 6-month follow-up, the absence of recurrent syncope or new neurological symptoms, despite occasional mild dizziness, was also clinically reassuring. Nevertheless, because no repeat neuroimaging, neurosurgical reassessment, or additional cardiac evaluation was performed during follow-up, the subsequent clinical course alone cannot definitively establish or exclude a causal relationship.

From a practical perspective, this case reinforces several important principles for emergency physicians. First, meticulous history-taking remains the cornerstone of syncope evaluation and frequently establishes the correct diagnosis even when unexpected abnormalities are identified during subsequent investigations [4,5]. Second, incidental neuroimaging findings should not automatically alter the diagnostic impression unless they are supported by convincing clinical and radiological evidence. Finally, multidisciplinary collaboration among emergency physicians, radiologists, neurologists, and neurosurgeons facilitates balanced interpretation of unexpected imaging findings and helps avoid unnecessary diagnostic or therapeutic interventions while ensuring patient safety.

From a neuroanatomical perspective, trigeminally mediated cardiovascular reflexes involve sensory afferents of the trigeminal nerve and brainstem autonomic pathways, with efferent responses mediated through cardiac vagal neurons located in the medulla oblongata [20,21]. In contrast, the lesion in our patient was extra-axial, centered within the quadrigeminal cistern, and abutted the posterior mesencephalon without evidence of brainstem compression. This anatomical mismatch, together with the absence of hydrocephalus, vascular compromise, or neurological deficits, further argues against a direct causal relationship between the lesion and the syncopal episodes.

A systematic review by Viau et al, including 3361 patients with syncope, reported a diagnostic yield of approximately 1.1-3.8% for head CT despite neuroimaging being performed in more than half of the included patients. Factors associated with a higher likelihood of positive neuroimaging included abnormal neurological findings, age ≥65 years, trauma, anticoagulant use, and a history of seizure or stroke [22]. Similarly, in a large retrospective multicenter study of 10 036 adults presenting with syncope, the positivity rate of brain MRI was only 0.99%, further demonstrating

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the very low yield of advanced neuroimaging in unselected syncope evaluations [23]. None of the clinical features identified in the systematic review as being associated with a higher likelihood of positive neuroimaging were present in our patient.

Limitations

This case report has several limitations. First, histopathological confirmation was unavailable because the patient declined surgical intervention; therefore, the lesion is described as radiologically consistent with an epidermoid cyst on the basis of its MRI features, rather than as a confirmed epidermoid cyst. In addition, dedicated heavily T2-weighted 3D steady-state sequences such as CISS or FIESTA were not obtained, which may have limited more detailed assessment of the lesion margins and its relationship to adjacent neurovascular structures.

Second, orthostatic blood pressure and heart rate measurements, which are a recommended component of the initial syncope evaluation [4,5], were not obtained. The initial syncope assessment was therefore incomplete, which limits the exclusion of alternative causes, including orthostatic hypotension; consequently, the diagnosis of situational reflex syncope should be regarded as probable rather than definitive. Third, selectively indicated second-line investigations, including ambulatory electrocardiographic monitoring, echocardiography, and tilt-table testing, were not performed. Although the clinical presentation was suggestive of situational reflex syncope and the ED evaluation revealed no significant cardiac or neurological abnormalities, the absence of these investigations further limits the ability to definitively exclude alternative causes of syncope, including an intermittent arrhythmic or other cardiac etiology.

Fourth, follow-up was limited to a telephone assessment at approximately 6 months. Although the patient reported no recurrent syncope or new neurological symptoms and remained functionally well, he had not attended the recommended neurosurgical follow-up, undergone repeat neuroimaging, or received additional cardiological evaluation. Consequently, interval radiological stability of the lesion and a cardiac etiology of the original syncopal episodes could not be objectively reassessed.

Finally, as this report describes a single patient, it cannot establish or definitively exclude a causal relationship between the quadrigeminal cistern lesion and the syncopal episodes.

Conclusions

In this patient, the clinical presentation was most consistent with probable situational reflex syncope associated with hair washing, and the quadrigeminal cistern lesion, radiologically consistent with but not histopathologically confirmed as an epidermoid cyst, was considered likely incidental rather than definitively unrelated to the episodes. Hair-grooming syncope should be considered in young patients when loss of consciousness is temporally linked to grooming activities and the neurological examination is normal. In uncomplicated syncope, the diagnostic yield of cranial neuroimaging is low, and routine imaging should generally be avoided in the absence of neurological abnormalities or other established high-risk features. Our patient remained free of recurrent syncope and new neurological symptoms during approximately 6 months of follow-up, supporting a clinically benign course, although this observation alone does not establish or exclude causality. When an unexpected intracranial lesion is identified, its causal relevance should be assessed according to its anatomical relationship to the proposed pathophysiological mechanism and objective evidence of mass effect or neurological dysfunction rather than its mere presence.

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Institution Where Work Was Done

Erzurum City Hospital, Erzurum, Türkiye.

Patient Consent

Written informed consent for publication of the patient's clinical information and accompanying anonymized images was obtained from the patient.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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