

CASE REPORT

Acute spontaneous subdural hematoma in a patient with autosomal dominant polycystic kidney disease

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ABSTRACT

Central nervous system (CNS) manifestations in autosomal dominant polycystic kidney disease (ADPKD) typically include intracranial aneurysms, arachnoid cysts, and dolichoectasia, which can lead to subarachnoid or chronic subdural hemorrhages. A review of the literature identified limited case of acute SDH in a patient with ADPKD, along with several reports of chronic SDH in this population. This suggests that acute SDH may be underrecognized in ADPKD, potentially progressing to the chronic stage and resulting in higher morbidity and mortality. In this report, we describe a case of acute SDH in a 42-year-old woman with ADPKD who presented with a severe right-sided headache, facial pain, and focal seizures in the absence of conventional risk factors or identifiable intracranial pathology. She was managed conservatively, and her neurological symptoms gradually improved. Comprehensive cerebral imaging, including magnetic resonance angiography and magnetic resonance venography, revealed no evidence of aneurysms, arachnoid cysts, venous sinus thrombosis, or other vascular abnormalities commonly associated with ADPKD. Although acute SDH is rare in ADPKD, and may be easily overlooked, allowing progression to chronic SDH. Therefore, maintaining a high index of clinical suspicion in patients with ADPKD is essential for early diagnosis and prevention of SDH-related complications

Keywords: Acute Subdural hematoma, Autosomal dominant polycystic kidney disease, Intracranial aneurysm, Chronic kidney disease.

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Introduction

Autosomal dominant polycystic kidney disease (ADPKD) is a systemic inherited disorder characterized by the development of multiple cysts, predominantly in the kidneys, with frequent extrarenal involvement of the liver and cardiovascular and central nervous systems (1). Intracranial aneurysms represent an important vascular manifestation of ADPKD, occurring in approximately 5% to 9% of affected individuals, with a prevalence three to fivefold higher than that in the general population (2). The interpretation of acute neurological symptoms in patients with ADPKD can be challenging, particularly because hypertension is common and may independently contribute to cerebrovascular complications. Therefore, new neurological symptoms in patients with ADPKD warrant careful evaluation for neurological and intravascular abnormalities.

While intracranial aneurysms, arachnoid cysts, and dolichoectasia are well-documented neurological manifestations of ADPKD, acute subdural hematoma (SDH) remains an uncommon complication (3). Spontaneous SDH has previously been reported in patients with ADPKD, although most published cases have involved chronic SDH, and the underlying mechanisms remain incompletely understood (4-6). Proposed mechanisms include structural abnormalities such as arachnoid cysts, alterations in cerebrospinal fluid dynamics, and impaired hemostasis associ-

ated with advanced kidney disease (4-6).

We report a case of acute spontaneous SDH in a patient with ADPKD without a history of head trauma or anticoagulant use. Comprehensive neurovascular imaging revealed no intracranial aneurysm, vascular malformation, or cerebral venous thrombosis. This case highlights an uncommon neurological presentation of ADPKD.

Case Presentation

A 42-year-old woman with a recent diagnosis of ADPKD presented with a 3-day history of acute-onset, right-sided headache and facial pain. She had no prior history of hypertension, diabetes mellitus, anticoagulant use, or head trauma. The headache was severe, rated 8/10 on the visual analog scale (VAS), and was associated with slurred speech. She also experienced three episodes of focal seizures. On examination, her vital signs were as follows: blood pressure (BP) of 130/90 mmHg, pulse rate (PR) of 86 beats per minute, respiratory rate (RR) of 22 breaths per minute, and peripheral oxygen saturation (SpO₂) of 98% on room air. Her Glasgow Coma Scale (GCS) score was E4V5M6. Neurological examination revealed a left-sided extensor plantar response and slurred speech, while the rest of the neurological findings were within normal limits. A non-contrast computed tomography (NCCT) of the brain was urgently performed which revealed a right-sided frontoparietotemporal subdural hematoma, as illustrated in Figure 1. She was further evaluated with MRI brain, magnetic resonance angiography (MRA), and magnetic resonance

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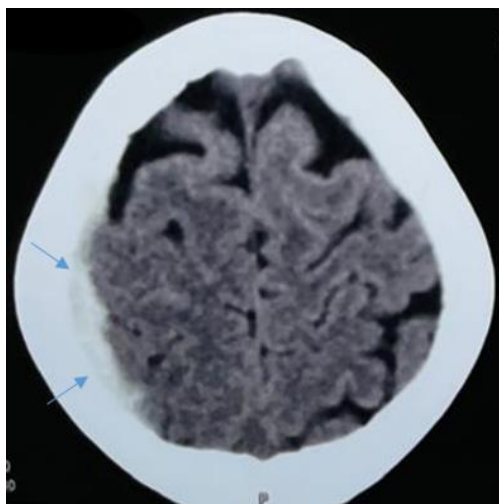


Figure 1. Axial CT image of the brain demonstrating an acute right-sided subdural hematoma (SDH), indicated by blue arrows.

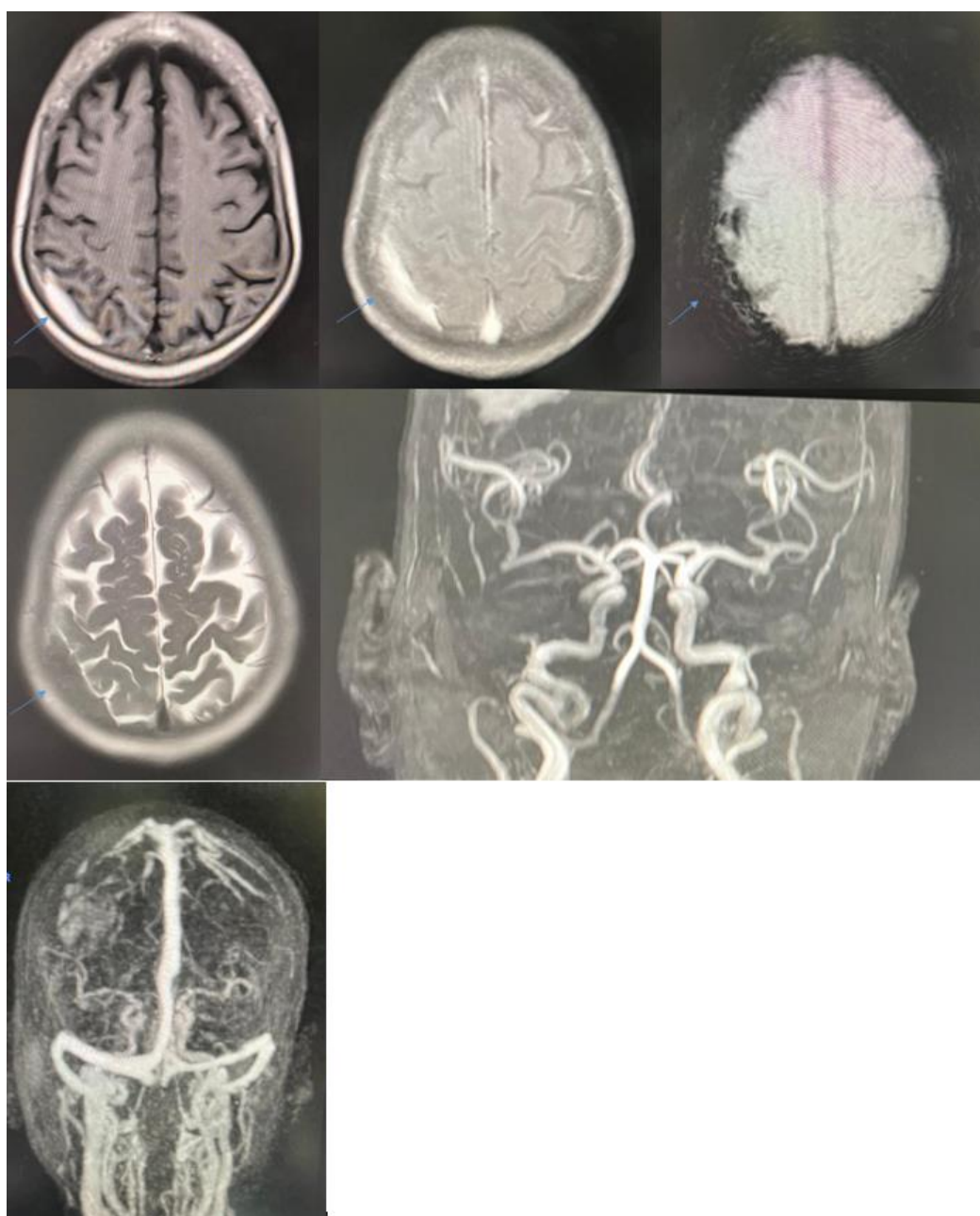


Figure 2. Axial section of MRI brain demonstrating both hypointense and hyperintense lesions indicative of acute subdural hematoma (SDH), as marked by the blue arrow. Magnetic Resonance Angiography (MRA) and Magnetic Resonance Venography (MRV) revealed no vascular abnormalities.

venography (MRV) to rule out intracranial aneurysms, arachnoid cysts, or other vascular abnormalities commonly associated with ADPKD as potential sources of bleeding. However, the MRI brain was suggestive of acute subdural hematoma and subsequent MRA and MRV showed no vascular malformations or thrombosis as depicted in Figure 2. Laboratory investigations showed a hemoglobin level of 9.5 g/dL and mild thrombocytopenia with a platelet count of 145,000/ μ L. Renal function was impaired, with a serum creatinine of 3 mg/dL and a blood urea nitrogen of 60 mg/dL. Liver function tests and coagulation profiles were within normal. The inflammatory markers, including ESR, CRP, and ANA profile, were all within normal limits, effectively ruling out underlying inflammatory or autoimmune conditions. The patient was managed conservatively with osmotherapy, antiepileptics, antihypertensives, analgesics (including paracetamol and pregabalin), and corticosteroids (dexamethasone). She was closely monitored through regular assessments by neurology and neurosurgery teams. As her clinical condition improved and follow-up neuroimaging showed no progression of the hematoma, conservative management was continued. Her blood pressure consistently remained below 140/90 mmHg throughout her hospital stay, indicating stable hemodynamics during the course of treatment. The patient showed gradual clinical improvement with the given treatment, experienced no further episodes of seizures, and reported significant relief from headache. She was discharged in a hemodynamically stable condition with full GCS. At the two-week follow-up, she remained asymptomatic, and her functional recovery was complete, with a Modified Rankin Scale (mRS) score of 0.

Discussions

In patients with ADPKD, neurological symptoms such as headache, cranial nerve palsies, seizures, and altered consciousness may be associated with underlying intracranial abnormalities (7). Acute SDH is an uncommon but clinically significant neurological complication reported in patients with ADPKD. Although the relationship between SDH and ADPKD remains uncertain, previous reports indicate that SDH can occur in individuals with ADPKD. Arachnoid cysts have been reported more frequently in patients with ADPKD and may predispose to subdural bleeding through alterations in cerebrospinal fluid dynamics and increased traction on bridging veins. Additionally, spinal meningeal diverticula, another recognized manifestation of ADPKD, may contribute to cerebrospinal fluid leakage and intracranial hypotension, which can increase traction on bridging veins and potentially predispose to SDH (3).

Although traumatic brain injury is the most common cause of SDH, several other factors may contribute to its occurrence in patients with ADPKD in the absence of preceding trauma. These include structural abnormalities such as arachnoid cysts, intracranial vascular abnormali-

ties, renal dysfunction with associated platelet dysfunction, antiplatelet or anticoagulant therapy, and systemic hypertension. Advanced CKD and uremia may impair platelet function and increase bleeding susceptibility, while antiplatelet or anticoagulant medications may further increase this risk in patients with relevant comorbidities (1,4,6-9). Hypertension, which is common in ADPKD, may also contribute to cerebrovascular complications through vascular injury and is an established risk factor for intracranial hemorrhage. However, these factors are not specific to ADPKD and may occur independently of the underlying genetic disorder.

Patients with ADPKD also exhibit endothelial dysfunction, even with preserved kidney function, characterized by reduced nitric oxide availability and increased oxidative stress (10). These abnormalities may contribute to the broader vascular phenotype of ADPKD, including the development of intracranial aneurysms. However, current evidence does not establish endothelial dysfunction as a direct mechanism for spontaneous SDH.

Large-scale studies involving more than 10,000 participants have identified smoking, hypertension, and family history as important risk factors for intracranial aneurysm formation, consistent with findings in the general population (2). Intracranial aneurysms are an important cerebrovascular manifestation of ADPKD; therefore, the development of acute neurological symptoms in these patients warrants appropriate neurovascular evaluation. In our patient, magnetic resonance angiography and venography did not demonstrate an intracranial aneurysm, vascular malformation, or cerebral venous thrombosis.

Previous reports have described both chronic and spontaneous SDH in patients with ADPKD, although the available literature consists predominantly of individual case reports and small case series (4-6,8,11). Therefore, the prognosis and clinical course of SDH specifically associated with ADPKD cannot be reliably generalized from the limited published literature. In our patient, the hematoma remained stable on follow-up imaging, neurological symptoms resolved with conservative management, and complete functional recovery was achieved.

Management of SDH depends on the size and location of the hematoma, mass effect, neurological status, and evidence of radiological progression. Selected patients without significant mass effect or neurological deterioration may be managed conservatively with close neurological monitoring and serial neuroimaging. Patients with significant mass effect, neurological deterioration, or progressive hematoma may require surgical evacuation, including burr-hole drainage or craniotomy. In our patient, conservative management was appropriate because there was no clinical or radiological evidence of deterioration, and follow-up imaging demonstrated stability of the hematoma.

Comprehensive genetic testing for inherited disorders associated with vascular fragility was not performed; therefore, an alternative genetic predisposition cannot be com-

pletely excluded. Thus, this case demonstrates an association between ADPKD and acute spontaneous SDH but does not establish a causal relationship.

In our case, acute SDH occurred without head trauma, anticoagulant therapy, significant thrombocytopenia, poorly controlled hypertension, or identifiable intracranial vascular abnormalities. Although this presentation is uncommon, the underlying mechanism remains uncertain. Clinicians should consider acute SDH in patients with ADPKD who develop new neurological symptoms and perform timely neuroimaging when clinically indicated.

Conclusion

Acute spontaneous SDH is an uncommon neurological presentation in patients with ADPKD. In the absence of trauma, anticoagulant use, or identifiable intracranial vascular abnormalities, its underlying mechanism may remain uncertain. New neurological symptoms in patients with ADPKD should prompt timely neuroimaging to facilitate early diagnosis and appropriate management.

Author contributions

Conceptualization: ADJ, MS, IJ, RKS; Methodology: ADJ, MS, IJ, RKS; Data curation: ADJ, MS, RKS; Validation: ADJ, MS, IJ, RKS; Writing - Original Draft: ADJ; Writing - Review & Editing: ADJ, MS, IJ, RKS; Validation: ADJ, MS, IJ, RKS; Supervision: RKS.

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Conflict of Interest

We declare that we do not have any conflict of interest.

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