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Review

Intracranial Aneurysms in Autosomal Dominant Polycystic Kidney Disease: Current State of Practice

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Abstract

Autosomal dominant polycystic kidney disease (ADPKD) is a genetic disorder primarily known for progressive kidney cysts, and it is the most common hereditary syndrome linked to intracranial aneurysms (IAs). Approximately 5–20% of ADPKD patients have IAs (versus ~3% in the general population). Key risk factors for IAs in ADPKD include a family history of aneurysmal subarachnoid hemorrhage (SAH), early-onset or poorly controlled hypertension, and possibly more severe kidney disease (e.g., large total kidney volume and reduced kidney function). The PKD1 and PKD2 mutations in ADPKD lead to polycystin-1/-2 dysfunction in vascular cells, causing intrinsic vessel wall weakness. This weakness—compounded by chronic hemodynamic stress and inflammation—predisposes ADPKD patients to aneurysm formation. Clinically, most aneurysms in ADPKD are small (<7 mm), asymptomatic, and located in the anterior cerebral circulation. Their growth and rupture risk appears similar to aneurysms in non-ADPKD patients; however, ruptures in ADPKD occur at younger ages, underscoring the need for vigilant management. This narrative review provides a nephrology-oriented overview of intracranial aneurysms in ADPKD, including pathophysiology, epidemiology, and clinical management. Key Messages: -ADPKD carries a higher prevalence of intracranial aneurysms (≈5–20%) than the general population (≈3%). Key risk factors include a family history of aneurysm/SAH, early or poorly controlled hypertension, and possibly advanced renal disease. -Guidelines support targeted rather than universal screening, mainly in patients with family history or prior SAH. -Non-contrast MRA is the preferred modality, usually initiated around age 30 in at-risk individuals. -Most aneurysms are small and asymptomatic; small lesions are monitored with BP control and imaging, while larger or high-risk aneurysms are treated prophylactically. -Broader screening remains debated. Future genetic insights may improve risk stratification, but current practice requires balancing rupture prevention against over screening.

Keywords: autosomal dominant polycystic kidney disease; intracranial aneurysm; polycystin; subarachnoid hemorrhage; aneurysm screening; vascular complications; nephrology; cerebrovascular risk

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1. Introduction

Autosomal dominant polycystic kidney disease (ADPKD) is an inherited systemic disorder characterized by numerous renal cysts leading to progressive kidney enlargement and chronic kidney disease. Beyond its renal manifestations, ADPKD has multiple extrarenal complications—notably vascular abnormalities such as intracranial aneurysms (IAs). An intracranial aneurysm is a saccular outouching of a cerebral artery that can rupture and cause subarachnoid hemorrhage (SAH), a life-threatening event. The association between ADPKD and intracranial aneurysms has been recognized for decades: ADPKD is the most common genetic syndrome linked to cerebral aneurysms. Recognizing this link is vital, as aneurysm rupture carries high morbidity and mortality, and ADPKD patients are often relatively young, requiring long-term medical management [1,2].

The prevalence of intracranial aneurysms in ADPKD far exceeds that in the general population. Roughly 2–4% of the general adult population may harbor an unruptured intracranial aneurysm, whereas studies in ADPKD report prevalence rates on the order of 5–20%, varying with cohort characteristics and risk factors. This elevated risk in ADPKD is thought to arise from a combination of genetic and pathophysiological factors inherent to the disease (such as mutations in PKD1 or PKD2 that compromise vascular wall integrity), as well as conventional cerebrovascular risk factors (e.g., hypertension, which is common and often early-onset in ADPKD) [3,4].

Several key clinical questions arise in managing this problem:

1. Who should be screened for intracranial aneurysms in ADPKD, and at what intervals?
2. What is the optimal imaging modality for screening?
3. How should an unruptured aneurysm identified in an ADPKD patient be managed—should we intervene earlier given the genetic context or manage it similarly to a sporadic aneurysm?
4. Does the natural history of aneurysms differ in ADPKD (for example, do they tend to rupture at smaller sizes or younger ages)?
5. What are the pathophysiological mechanisms by which ADPKD predisposes patients to aneurysm formation?

This narrative review aims to address these questions by synthesizing current knowledge on intracranial aneurysms in the setting of ADPKD. We focus on nephrology-related considerations (acknowledging that many readers are nephrologists or general internists caring for ADPKD patients) while also covering essential neurological and neurosurgical aspects. The scope is limited to intracranial aneurysms in ADPKD (without delving into other cerebrovascular disorders in ADPKD) to maintain a clear focus on aneurysm-related issues.

2. Materials and Methods

We performed a comprehensive literature search and review to gather information on intracranial aneurysms in the context of ADPKD. Sources included peer-reviewed journal articles, systematic reviews, meta-analyses, and clinical guidelines up to the most recent evidence available. We searched databases PubMed and Scopus using keywords including "ADPKD and intracranial aneurysm," "polycystic kidney and aneurysm," "polycystic and vascular," "aneurysm screening ADPKD," and related terms. Both the clinical and basic science literature were reviewed to capture aspects of pathophysiology and genetics in addition to epidemiology and management. Relevant guideline documents and consensus statements were also consulted to extract current recommendations on screening and management. Given that this is a narrative review, formal study quality assessments were not performed; instead, we aimed to integrate findings and expert opinion from key publications to provide a comprehensive overview. Where appropriate, we included figures and tables to summarize critical data. Headings were organized into thematic

sub-sections for clarity. This review does not contain any new studies involving human or animal subjects performed by the authors and thus did not require ethical approval.

3. Results and Discussion

3.1. Genetics and Vascular Pathophysiology of Aneurysm Formation in ADPKD

Genetic Basis and Polycystin Function: ADPKD results from heterozygous mutations in PKD1 (chromosome 16) or PKD2 (chromosome 4), encoding polycystin-1 and polycystin-2, respectively. Polycystin-1 is a receptor-like transmembrane protein, while polycystin-2 is a calcium-permeable channel. These proteins interact within cellular compartments such as primary cilia, regulating mechanosensation and intracellular signaling. Beyond the kidney, polycystins are expressed in vascular endothelial and smooth muscle cells, including cerebral arteries, and have been demonstrated within intracranial aneurysm walls in ADPKD, linking PKD gene dysfunction to cerebrovascular structure [5–7].

Kidney cyst formation follows a "two-hit" mechanism, where the somatic loss of the normal allele drives focal disease. A similar concept has been proposed for vascular lesions, in which the localized loss or reduced function of polycystins may create focal vessel wall weakness. Even without a clear second hit, the systemic haploinsufficiency of polycystin-1/2 may gradually impair vascular integrity [6,8,9].

Polycystin Signaling in Vessels: Polycystin-1 functions as a mechanosensor and adhesion molecule, whereas polycystin-2 regulates intracellular Ca^{2+} signaling. Together they modulate calcium-dependent and cyclic AMP pathways that govern cellular differentiation, proliferation, and structural maintenance. In vessels, these complexes support endothelial responses to shear stress and preserve smooth muscle contractility and wall elasticity, thereby maintaining vascular stability [10–12].

Vessel Wall Abnormalities: PKD mutations likely increase aneurysm susceptibility through impaired mechanosensing and structural support in endothelial and smooth muscle cells. Defective polycystin signaling may promote endothelial dysfunction, extracellular matrix weakening, and reduced smooth muscle integrity. Hemodynamic stress—often amplified by early-onset hypertension—and inflammatory remodeling further strain vulnerable vessel segments, promoting saccular aneurysm formation. Even without aneurysms, ADPKD patients may show diffuse vascular changes such as dolichoectasia or enlarged intracranial arteries, suggesting generalized loss of vessel wall strength. Histological studies demonstrate features similar to sporadic aneurysms, including elastic lamina degeneration, smooth muscle loss, and inflammation, although whether these changes are accelerated by polycystin dysfunction remains under investigation. Overall, PKD1/PKD2 mutations produce intrinsic vascular fragility that, combined with hemodynamic and inflammatory factors, predisposes patients to aneurysm development [5,7,13].

Polycystin-2 appears to contribute to vascular smooth muscle cell (SMC) contractility through the regulation of the actin cytoskeleton. Experimental models show that the loss of polycystin-2 impairs actin organization, reducing SMC contractile capacity and vascular tone. The resulting decrease in vessel wall resilience may promote focal dilation and aneurysm formation under chronic conditions [14–16].

Hemodynamic Stress and Hypertension: Hemodynamic stress is a major driver of aneurysm formation and is particularly relevant in ADPKD, where hypertension develops early and affects most patients by mid-adulthood. Chronic hypertension increases mechanical stress at arterial bifurcations, especially in the circle of Willis. In ADPKD, systemic hypertension combined with intrinsic vessel wall fragility from polycystin dysfunction creates a synergistic risk, and hypertension has been identified as an independent predictor of aneurysm presence (entire process is shown in Figure 1). Effective blood pressure control is therefore important not only for renal protection but also for reducing aneurysm risk [2,17].

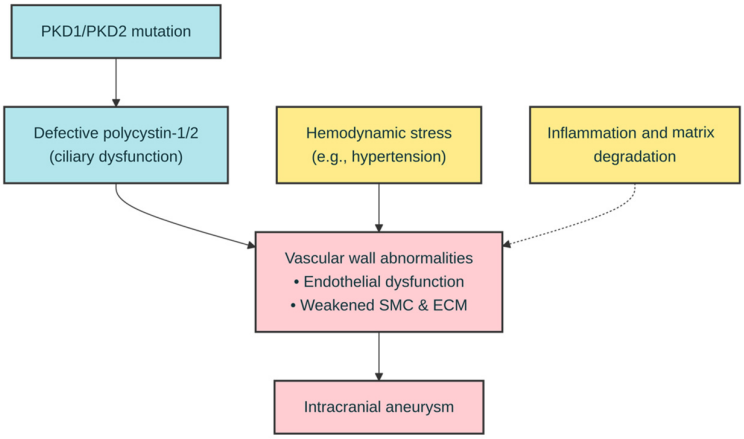


Figure 1. Schematic representation of pathophysiology leading to intracranial aneurysm formation in ADPKD.

In addition, endothelial dysfunction may impair local shear stress responses. Because polycystin-1 normally mediates endothelial mechanosensing, its dysfunction may blunt protective vascular responses to flow, leaving focal arterial regions more vulnerable to aneurysm initiation [18].

Inflammation and Matrix Degradation: Aneurysm development involves chronic vessel wall inflammation, with macrophages, T cells, cytokines, and matrix metalloproteinases (MMPs) driving the degradation of collagen and elastin. In ADPKD, early hypertension, oxidative stress, and systemic inflammatory influences may amplify these processes, accelerating extracellular matrix breakdown. Together, genetic defects, hemodynamic stress, and inflammatory remodeling create a pro-aneurysmal vascular environment [2,7].

3.2. Epidemiology of Intracranial Aneurysms in ADPKD

Intracranial aneurysms represent one of the most clinically relevant cerebrovascular manifestations of ADPKD, with a prevalence several times higher than in the general population and a tendency toward earlier occurrence. Risk is influenced by factors such as family history, age, and vascular phenotype, although aneurysms may also develop in patients without traditional risk markers [2,3,6,9,17,19–21]. The main epidemiologic features and their clinical implications are summarized in Table 1.

Table 1. Epidemiologic and morphologic characteristics of intracranial aneurysms in ADPKD.

Parameter	Key Findings	Clinical Implications
Prevalence	ADPKD patients have a higher prevalence of unruptured intracranial aneurysms (~5–20%; meta-analytic estimate ~10%) compared with ~3% in the general population.	Confirms ADPKD as a high-risk population and supports structured screening strategies.
Family history	Prevalence is ~6–10% without family history and ~20–30% with a positive family history of aneurysm/SAH; however, many affected patients lack family history.	Family history is a major risk marker but cannot be the sole criterion for screening.
Age	Aneurysms are rare in young patients, increase from the 3rd–4th decade, and may affect up to ~20% by the 6th decade; rupture occurs earlier (late 30s–early 40s) than in sporadic cases.	Supports screening in adulthood and earlier vigilance compared with the general population.

Table 1. *Cont.*

Parameter	Key Findings	Clinical Implications
Multiplicity	Multiple aneurysms occur in ~20–30% of ADPKD patients with aneurysms, higher than in sporadic series (~15%).	The identification of one aneurysm warrants complete intracranial vascular evaluation.
Location	Distribution resembles the general population, predominantly in the anterior circulation (ACOM, MCA bifurcation, PCOM–ICA junction), without a distinct ADPKD pattern.	Full vascular imaging is necessary during screening.
Infundibulum vs. aneurysm	Infundibula are usually <3 mm conical branch-origin dilations; lesions that are ≥3 mm or rounded or lacking a branch are more likely true aneurysms.	Accurate interpretation and follow-up reduce misclassification and overtreatment.

3.3. Risk Factors and Risk Stratification in ADPKD

Several factors have been identified that influence the risk of intracranial aneurysm in ADPKD. These factors also guide decisions about which patients to screen and how to counsel patients regarding their risk. Intracranial aneurysm risk in ADPKD is multifactorial, reflecting genetic susceptibility, vascular stressors, and overall disease burden. Family history remains the strongest clinical predictor (risk increase based on family history is shown in Figure 2), but age, hypertension, renal disease severity, and lifestyle factors also contribute, and aneurysms frequently occur in patients without obvious risk markers. These epidemiologic and clinical risk determinants are summarized in Table 2 and form the basis for current risk-adapted screening strategies.

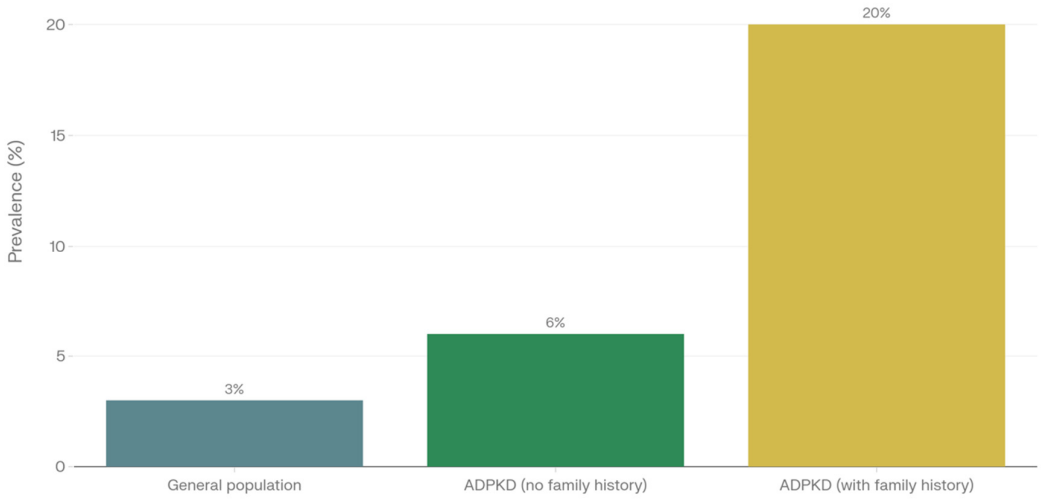


Figure 2. Risk increase based on presence of condition and family history of condition.

Table 2. Key risk determinants of intracranial aneurysms in ADPKD.

Risk Factor	Key Findings	Clinical Implications
Family history of aneurysm/SAH	Strongest established predictor; prevalence is markedly higher in patients with affected first-degree relatives, although many aneurysms still occur without familial history.	Central criterion for screening, but the absence of family history does not exclude risk.
Age	Risk increases from the 3rd–4th decade and continues to rise with age; rupture tends to occur earlier than in non-ADPKD populations.	Supports screening in adulthood and earlier vigilance compared with the general population.

Table 2. *Cont.*

Risk Factor	Key Findings	Clinical Implications
Sex	Some studies suggest a female predominance, but the findings are inconsistent, and both sexes remain at significant risk.	Sex alone is not sufficient to guide screening decisions.
Hypertension	Common and often early in ADPKD; contributes to aneurysm formation and rupture through chronic hemodynamic stress.	Strict blood pressure control is a key preventive measure for all patients.
Genotype (PKD1 vs. PKD2)	Severe PKD1 mutations may be associated with higher vascular burden, though evidence is still emerging.	Not routinely used for screening decisions but may influence individualized assessment.
Kidney disease severity	Larger kidney volume and more advanced renal dysfunction may correlate with higher aneurysm prevalence, suggesting shared disease severity.	May justify a lower screening threshold in patients with aggressive renal phenotypes.
Smoking/heavy alcohol use	Smoking promotes vascular degeneration and may increase aneurysm risk; heavy alcohol use may exacerbate hemodynamic stress.	Lifestyle modification is recommended as part of vascular risk management.
Prior aneurysm or SAH	History of aneurysm increases the risk of de novo or recurrent lesions.	Lifelong imaging surveillance and long-term follow-up are advised.

In summary, the highest-risk profile for intracranial aneurysm in ADPKD would be a patient who is middle-aged or older, with a family history of aneurysmal hemorrhage, with long-standing hypertension (especially if previously suboptimally controlled), who is possibly female, with a severe PKD1 mutation, and with very large polycystic kidneys (reflecting high disease burden). However, it bears emphasis that aneurysms can also occur in ADPKD patients without any of these "red flags." Indeed, many ADPKD patients who are found to have aneurysms have no obvious risk factors. This reality underlies the ongoing debate: should all ADPKD patients (or at least a broader subset than currently recommended) be offered screening given the inherent aneurysm risk of the disease? The next section will discuss current screening strategies and guidelines in light of these risk considerations [2,3,6,21–27].

3.4. Natural History and Outcomes of Aneurysms in ADPKD

Rupture Risk of Unruptured Aneurysms: A central question is whether aneurysms in ADPKD behave more aggressively than sporadic aneurysms. Current evidence suggests that small unruptured aneurysms in ADPKD follow a similar natural history to those in non-ADPKD populations. Long-term follow-up studies, including the Mayo screening cohort, reported no ruptures and minimal growth among small observed aneurysms, consistent with general population data in which most aneurysms <7 mm remain stable. A meta-analysis estimated an annual rupture rate of ~0.1–0.2% for predominantly small aneurysms, comparable to non-ADPKD cohorts [2,3,27]. However, interpretation is limited by observational bias, as many aneurysms are closely monitored or treated preventively, and by the exclusion of patients presenting initially with rupture [2,17].

Insights from Ruptured Aneurysms: Studies of aneurysmal SAH in ADPKD show that rupture often occurs at younger ages (30s–40s versus 50s–60s in the general population), and many patients lack prior family history, meaning that risk-based screening may miss some individuals. Some series suggest rupture at slightly smaller sizes (median ~6 mm), raising concern that vessel fragility could increase risk at lower diameters, although definitive evidence is limited. Consequently, some clinicians favor treating moderately sized aneurysms (~5 mm) in younger ADPKD patients rather than prolonged observation [1,2]. Still, longitudinal data indicate that many small aneurysms remain stable for

years, supporting surveillance unless growth, irregular morphology, or higher-risk features develop [1,2].

Outcomes of Aneurysm Rupture: Outcomes after rupture are severe and similar to the general population, with high mortality and disability rates. Management follows standard SAH principles (early aneurysm securing, neurocritical care, vasospasm prevention), though ADPKD may complicate care due to chronic hypertension or renal dysfunction requiring multidisciplinary management. In some cases, aneurysmal SAH leads to the initial diagnosis of previously unrecognized ADPKD [2,3,20,26].

Summary: ADPKD increases overall aneurysm prevalence, but small unruptured aneurysms appear to carry a low short-term rupture risk similar to sporadic lesions. Because rupture tends to occur at younger ages and with severe consequences, proactive risk-adapted management and screening remain central to care.

3.5. Screening Strategies for Intracranial Aneurysms in ADPKD

Screening for intracranial aneurysms in ADPKD aims to detect lesions before rupture, enabling surveillance or preventive treatment to reduce the risk of subarachnoid hemorrhage. Because aneurysm prevalence is elevated in ADPKD, screening has a clear rationale; however, it also carries disadvantages such as cost, patient anxiety, and the detection of small lesions with uncertain clinical relevance. Current practice therefore favors the targeted screening of higher-risk patients rather than the universal screening of all individuals with ADPKD [1,2,26].

Non-contrast magnetic resonance angiography (MRA), especially time-of-flight techniques, is the preferred screening modality because it is noninvasive and free of radiation and avoids contrast exposure while maintaining good sensitivity for aneurysms $\geq$2–3 mm. Computed tomography angiography (CTA) offers high spatial resolution but requires iodinated contrast and radiation, making it mainly a secondary option when MRI is contraindicated or inconclusive. Digital subtraction angiography (DSA) remains the diagnostic gold standard but is invasive and carries a small procedural risk; it is therefore reserved for confirmatory evaluation or treatment planning rather than routine screening [1,9,28–33]. The details of these modalities are given in Table 3.

Table 3. Imaging modalities for intracranial aneurysm screening in ADPKD.

Modality	Key Features	Advantages	Limitations/Typical Use
MRA (non-contrast)	Time-of-flight imaging; detects aneurysms $\geq$2–3 mm	Noninvasive, no radiation, no nephrotoxic contrast	Preferred screening tool; may miss very small aneurysms or produce flow artifacts
CTA	Contrast-enhanced CT angiography	High spatial resolution, good for small aneurysms	Radiation and iodinated contrast; used when MRI is unavailable/contraindicated or for clarification
DSA	Catheter-based angiography (gold standard)	Highest anatomical detail and accuracy	Invasive with small stroke risk; used for confirmation or pre-intervention planning

Screening recommendations in ADPKD favor a targeted, risk-based approach rather than universal testing. The goal is to maximize detection in patients most likely to benefit while avoiding unnecessary imaging, anxiety, and downstream interventions in low-risk individuals. Across major guidelines, the strongest indication is a family history of aneurysm or aneurysmal SAH, followed by prior personal aneurysm history. Additional situations that may justify screening include high-risk occupations, pre-transplant or major elective surgery evaluation, and strong informed patient preference. In patients without recognized

risk factors, routine screening is generally not recommended, and decisions should be individualized through shared clinician–patient discussion [1,34,35]. Indications are shown in Table 4.

Table 4. Indications for intracranial aneurysm screening in ADPKD.

Indication	Rationale	Practical Implication
Family history of aneurysm or SAH	Highest diagnostic yield; strongest established risk factor	Screening generally recommended
Personal history of SAH or treated aneurysm	Risk of de novo or recurrent aneurysm	Periodic lifelong surveillance
High-risk occupation or circumstance	Sudden neurological collapse could endanger others	Individualized screening decision
Before major elective surgery (e.g., transplant)	Perioperative hemodynamic stress may increase rupture risk	Often considered as part of preoperative assessment
Patient anxiety or informed preference	Shared decision-making and psychological reassurance	Screening may be offered after counseling
No risk factors present	Low yield; risk–benefit ratio uncertain	Routine screening not recommended

Screening for intracranial aneurysms in ADPKD is generally initiated in adulthood, most often around age 30–40, as aneurysms are rare in children and adolescents and early screening would lead to many years of repeated imaging with limited benefit. Earlier screening may be considered in selected high-risk situations, such as a strong family history of early aneurysm rupture. When an initial screening MRA is negative, repeat imaging is commonly recommended at intervals of about 5–10 years in patients who remain at elevated risk, particularly if screening was done at a younger age or if new risk factors emerge. There is no universally accepted interval, and frequency is typically individualized according to risk profile and clinical context [32,36].

Geographic practice differs. In Japan, many centers adopt a more inclusive approach, often offering at least one screening brain MRA to all ADPKD patients by early adulthood regardless of family history. This strategy reflects both higher reported aneurysm prevalence and a proactive screening culture. In addition, Japanese practice tends to favor the earlier elective treatment of detected aneurysms, supported by strong endovascular expertise and a preventive philosophy aimed at minimizing even low-probability catastrophic events [37–39].

Despite these regional differences, core principles are consistent across practices: prioritize screening in higher-risk individuals, use noninvasive MRA as the preferred modality, and manage detected aneurysms according to established neurosurgical principles while considering the ADPKD context. There is also a clear shift toward patient-centered decision-making, with increasing willingness to offer screening to well-informed patients who request it, acknowledging both patient values and the imperfect ability of current risk models to predict who will develop aneurysms [40,41].

Crucially, any decision about screening should be made with informed consent and shared decision-making. The patient should understand the potential benefits (e.g., finding and treating an aneurysm before it ruptures) as well as the potential downsides (e.g., false positives on imaging, the anxiety of living with a known small aneurysm, or the risks associated with possible interventions) of undergoing screening. With this understanding, the decision can be tailored to the individual patient's risk tolerance and values.

Table 5 provides a comparison of key recommendations from various guidelines and expert consensus statements regarding the screening and management of intracranial aneurysms in ADPKD. As seen in Table 1, there is broad agreement across guidelines on

who should be screened (those with family or personal history of aneurysms, etc.) and how to screen (MRI/MRA preferred). The differences lie in how aggressive to be beyond high-risk groups. The trend in recent years, especially influenced by patient advocates and newer data, is a more patient-centered approach: if an ADPKD patient is aware of the risk and wants to be screened, even without classic risk factors, many clinicians will proceed with screening after counseling.

Table 5. Screening and management recommendations for intracranial aneurysms in ADPKD across guidelines.

Guideline	Screening Indications	Preferred Imaging	Management Principles
KHA-CARI (2015)	• Family history of IA/SAH • Prior IA/SAH • High-risk occupation or pre-transplant	• Non-contrast MRA • Repeat only if high-risk (≈5–10 yrs)	• Targeted screening only • Observe small aneurysms
KDIGO 2025 ADPKD	• Personal or family IA/SAH • Consider pre-transplant/major surgery • Patient-informed request	• First-line MRA • Individualized follow-up	• Shared decision-making • No ADPKD-specific treatment thresholds
AHA/ASA 2015	• ADPKD with additional risk factors • ≥2 first-degree relatives with IA/SAH	• MRA or CTA	• General rupture risk models (e.g., PHASES) • Treat ≥5–7 mm (lower if posterior)
Japanese practice	• Broad/near-universal adult screening	• Baseline MRA ± periodic rescreening	• Lower treatment threshold • Prevention-oriented strategy

Pros and Cons of Screening

Screening ADPKD patients for intracranial aneurysms has clear potential benefits but also important drawbacks. The decision to screen should balance the prevention of catastrophic hemorrhage against the risks of overdiagnosis, anxiety, and unnecessary interventions, ideally through shared decision-making [42–45].

Pros

- The early detection of unruptured aneurysms allows for monitoring or treatment before rupture, potentially preventing fatal or disabling SAH.
- Identifying an aneurysm encourages stricter risk factor control (blood pressure, smoking avoidance, lifestyle modification).
- A negative screening result may provide reassurance for patients and families.
- The detection of small aneurysms enables structured surveillance instead of leaving lesions undiscovered.
- Screening supports proactive rather than reactive care.

Cons

- Most screened ADPKD patients will not have aneurysms, raising cost-effectiveness concerns.
- False-positive or indeterminate findings may cause anxiety and lead to unnecessary invasive testing.
- The discovery of very small aneurysms can create long-term surveillance burdens or overtreatment risks.
- The psychological impact (“ticking time bomb” effect) may reduce quality of life in some patients.
- In certain settings, a known aneurysm may affect insurance, licensing, or occupational clearance.

Screening decisions in ADPKD should be individualized and guided by shared decision-making, balancing potential benefit against the risks of overdiagnosis and patient anxiety. Clinicians should discuss the possible outcomes of screening, including incidental findings and long-term surveillance implications, so patients can make informed choices aligned with their values and risk profile. Regardless of whether screening is performed, the aggressive management of modifiable vascular risk factors—particularly blood pressure control—and education about the warning symptoms of aneurysm rupture remain essential. When screening is negative, patients should be reassured while also being informed about whether repeat imaging may be appropriate based on ongoing risk [42–45].

3.6. Management of Intracranial Aneurysms in ADPKD

Once an intracranial aneurysm is identified in an ADPKD patient, management follows the general principles used for unruptured aneurysms, with added attention to renal status and lifelong vascular risk. The key decision is between observation and preventive intervention, based on aneurysm features, patient factors, and preferences. Table 6 summarizes major decision-making factors.

Table 6. Major factors guiding management of unruptured intracranial aneurysms in ADPKD.

Factor	Key Information	Clinical Implication
Aneurysm size	Most screened aneurysms are small (<7 mm); rupture risk rises with size, though ADPKD lesions may rupture at slightly smaller diameters [2,24,46].	Small lesions often observed; 5–6 mm aneurysms may be treated in high-risk young patients.
Aneurysm location	Posterior circulation aneurysms have higher rupture risk than anterior circulation lesions.	Lower threshold for treatment in higher-risk locations.
Age and overall health	Younger patients have longer lifetime risk; older or comorbid patients face higher procedural risk.	Younger patients treated more proactively; older patients often observed.
Renal function	Advanced kidney disease affects contrast use and peri-procedural planning but does not preclude treatment.	Requires tailored peri-procedural management.
Patient preference	Anxiety and individual risk tolerance influence management choice.	Shared decision-making is essential.

All ADPKD patients with aneurysms should receive strict blood pressure control, lifestyle counseling, and education on rupture warning symptoms [2,24,46].

For conservative management, surveillance imaging is typically performed at ~6 months and 12 months, then spaced to every 2–5 years if stable, although some clinicians continue periodic long-term imaging due to the risk of new aneurysm formation [2,20]. Growth or morphological change usually triggers reevaluation for intervention; multiple aneurysms are assessed individually, with priority given to the highest-risk lesion [47–49].

If treatment is indicated, options include endovascular therapy and microsurgical clipping, selected according to aneurysm anatomy and local expertise. Endovascular coiling is often favored because it is minimally invasive and widely applicable, with outcomes in ADPKD comparable to non-ADPKD patients. Microsurgical clipping remains important for complex or unfavorable aneurysm morphologies and offers durable long-term exclusion. The elective treatment of unruptured aneurysms carries a pooled major complication risk of roughly 3–6%, with coiling generally being less invasive but clipping more durable [2,3].

Outcomes after elective treatment are usually excellent, with most patients recovering fully when treated before rupture. Follow-up imaging is required after coiling, and periodic long-term screening is often advised even after treatment, as ADPKD patients remain at risk for de novo aneurysms [36,50]. Preventing rupture avoids the high morbidity and

mortality associated with SAH, which historically contributed significantly to ADPKD-related mortality [2,17].

Special considerations include the continuation of ADPKD therapies such as tolvaptan, which has no known effect on aneurysm risk. Tight blood pressure control remains central to both renal and cerebrovascular protection, and acute SAH management follows standard protocols with attention to chronic hypertension and renal function [51].

4. Conclusions

Intracranial aneurysm is a major extrarenal complication of autosomal dominant polycystic kidney disease. Advances in ADPKD genetics and vascular biology show that polycystin-1 and -2 deficiency weakens the arterial wall; combined with chronic hypertension, this predisposes patients to aneurysm formation and rupture. The detection of an aneurysm in an ADPKD patient is therefore clinically critical, given the risk of subarachnoid hemorrhage.

Management requires individualized risk stratification. High-risk features include a family history of aneurysm or SAH, early-onset or poorly controlled hypertension, and markers of severe renal disease (e.g., large kidney volume). Current guidelines favor the targeted screening of such subgroups using non-contrast MRA rather than universal screening, although broader strategies are debated because many aneurysms occur without family history. Small, low-risk aneurysms are monitored with strict blood pressure control and imaging surveillance, whereas larger or morphologically high-risk lesions are treated prophylactically.

Outcomes have improved with earlier detection and elective treatment, yet rupture may still be the first manifestation, underscoring limitations in current prediction models. Optimal care is multidisciplinary and patient-centered, emphasizing risk factor control, the prudent screening of high-risk individuals, and timely intervention to reduce hemorrhagic complications while avoiding overtreatment.

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Abbreviations

The following abbreviations are used in this manuscript:

ADPKD	Autosomal dominant polycystic kidney disease
IA	Intracranial aneurysm (typically saccular)
SAH	Subarachnoid hemorrhage
PKD1	Gene mutated in ADPKD (encodes polycystin-1)
PKD2	Gene mutated in ADPKD (encodes polycystin-2)
MRA	Magnetic resonance angiography
CTA	Computed tomography angiography
DSA	Digital subtraction angiography (catheter angiography)

ECM	Extracellular matrix
SMC	Smooth muscle cell
BP	Blood pressure
FHx	Family history
KDIGO	Kidney Disease: Improving Global Outcomes
KHA-CARI	Kidney Health Australia—Caring for Australians with Renal Impairment
AHA/ASA	American Heart Association/American Stroke Association
CKD	Chronic kidney disease
TKV	Total kidney volume
PCOM	Posterior communicating artery
ACOM	Anterior communicating artery
MCA	Middle cerebral artery
PHASES	Risk score for aneurysm rupture (Population, Hypertension, Age, Size, Earlier SAH, Site)

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