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Schistosomiasis-Associated Pulmonary Arterial Hypertension in Brazil: A Call to Action for Enhanced Diagnostic Recognition—2025 Pulmonary Vascular Research Institute Infection in Pulmonary Vascular Disease Consortium Perspectives

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ABSTRACT

Schistosomiasis-associated pulmonary arterial hypertension (Sch-PAH) is one of the most frequent etiologies of pulmonary arterial hypertension in Brazil and globally. The disease is a complication of hepatosplenic schistosomiasis characterized by portal hypertension, egg deposition, and granuloma formation in the pulmonary arteries leading to inflammation, vascular remodeling, increased pulmonary vascular resistance, and progressive right heart failure. During the Infection in Pulmonary Vascular Disease (iPVD) consortium meeting during the Pulmonary Vascular Research Institute (PVRI) annual meeting in January 2025, in Rio de Janeiro, Brazil, a dedicated symposium entitled schistosomiasis-associated pulmonary vascular disease in Latin America addressed the most relevant aspects of the epidemiology, diagnosis, and prevention of Sch-PAH in Brazil. This review summarizes the main discussed topics and serves as a call to increase efforts to understand the disease burden better and enhance diagnostic proficiency.

1 | Introduction

Schistosomiasis is the third most prevalent parasitic disease worldwide, after malaria and amebiasis. There are more than 240 million infected people around the globe and another 800 million individuals at risk of contracting the disease, predominantly in sub-Saharan Africa, South America (particularly in Brazil), China, and the Caribbean. The World Health Organization (WHO) estimates that most schistosomiasis-related deaths and disease-related disabilities occur in Africa [1].

Schistosomiasis-associated pulmonary arterial hypertension (Sch-PAH) is a complication estimated to affect approximately 5%–10% of hepatosplenic schistosomiasis. However, this estimate is based on limited available data [2–5] and the true prevalence of Sch-PAH remains uncertain, particularly in endemic regions

such as Africa, which accounts for nearly 80% of the global schistosomiasis burden [6].

The Pulmonary Vascular Research Institute (PVRI) 2025 Rio Annual Congress hosted the Infection in Pulmonary Vascular Disease (iPVD) Consortium Task Force pre-conference session, which brought together local and international experts to examine the critical intersection of infectious diseases and pulmonary vascular diseases. A dedicated session focused specifically on Sch-PAH in Latin America convened leading researchers and clinicians in the field, including experts from endemic regions with substantial Sch-PAH disease burdens, to present contemporary insights into disease epidemiology, clinical phenotypes, and diagnostic challenges. Through a combination of case-based presentations, epidemiological updates, and discussion of an ongoing National Institutes of Health (NIH)-funded multicenter study, this session aimed to advance understanding of Sch-PAH pathobiology, improve diagnostic accuracy, and address critical gaps in preventive and therapeutic strategies for this parasitic-driven pulmonary vascular disease. Here, we summarize the main topics addressed during the 2025 iPVD Consortium Task Force meeting regarding diagnosis, epidemiology and prevention of Sch-PAH in Brazil.

2 | Hepatosplenic Schistosomiasis and Sch-PAH Pathophysiology

The WHO classifies schistosomiasis into urogenital and intestinal forms, with *Schistosoma mansoni* being highly prevalent in Brazil. The disease can be further categorized into early/acute and late/chronic forms, as shown in Table 1. Up to 10% of individuals in highly endemic areas in Brazil may develop the hepatosplenic form, which may manifest as compensated, decompensated, or complicated disease [7].

In hepatosplenic schistosomiasis, granulomatous inflammation surrounding the parasite eggs leads to fibrosis, resulting in portal hypertension and complications, such as esophageal varices, which can then lead to upper gastrointestinal bleeding [2]. Sch-PAH is another complication resulting from the proliferation and irreversible remodeling of pulmonary vessels induced by *Schistosoma* infection. Egg embolization to the lung via portosystemic shunts, followed by egg mechanical deposition, increased endothelial shear stress, and secondary type 2 immune response contributes to increased pulmonary artery pressure and vascular resistance, which can progress to right ventricular dysfunction and death [8, 9].

Polymorphisms in tumor necrosis factor-alpha (TNF-α) and other host genetic factors may predispose individuals to more severe Sch-PAH. TNF-α has also been shown to influence Schistosoma egg production in a time- and dose-dependent manner. Experimental studies suggest that increased TNF-α levels can accelerate oviposition, resulting in a higher rate of egg deposition, although the eggs produced may display altered maturation. This interaction between host inflammatory signaling and parasite reproduction may contribute to variations in disease burden and immune responses. Although most Sch-PAH cases occur in the setting of hepatosplenic schistosomiasis, pulmonary vascular disease can develop even in the absence of overt hepatosplenic involvement [10].

TABLE 1 | *Schistosomiasis mansoni* clinical classification.

Initial phase
Acute form
1. Asymptomatic
2. Symptomatic
Late phase
Chronic phases (according to the most affected organs)
1. Hepatic-intestinal
2. Hepatic (periportal fibrosis without splenomegaly)
3. Hepatosplenic (periportal fibrosis with splenomegaly)
4. Complicated forms
4.1 Vasculopulmonary
4.2 Glomerulopathy
4.3 Neurological
4.4 Other localizations (eye, skin, urogenital)
4.5 Pseudoneoplastic
4.6 Lymphoproliferative disease

3 | Clinical and Epidemiological Aspects of Schistosomiasis and Sch-PAH in Brazil

Schistosomiasis remains a significant public health issue in Brazil, particularly in the states of Pernambuco, Bahia, and Minas Gerais, despite national prevalence reductions. It is estimated that 5–7 million people are infected in the country, and 25 million live in endemic areas and are at risk of schistosomiasis. Migration from rural areas to urban peripheries has shifted the disease burden, creating new pockets of schistosomiasis prevalence in large cities, such as Recife and Belo Horizonte. Even though living conditions and access to treatment have improved, local transmission persists in areas near rivers [11–13].

Despite the reduction in the overall prevalence of schistosomiasis in Brazil, the rates of severe cases, such as Sch-PAH, remain stable. Sch-PAH underdiagnosis remains a significant challenge owing to limited access to healthcare and a lack of awareness within the medical community. Nevertheless, in pulmonary hypertension reference centers in Brazil, Sch-PAH is one of the most prevalent etiologies, representing up to 30% of PAH cases [14–17].

Schistosomiasis is more common in men due to greater exposure related to work activities. However, Sch-PAH disproportionately affects women, potentially reflecting the biological mechanisms observed in other forms of PAH. Brazilian patients are often middle-aged women presenting with advanced symptoms (NYHA Functional Class III or IV), but performing better on 6-min walk tests compared to idiopathic PAH (IPAH) patients despite a similar hemodynamic profile [18, 19]. Standard PAH therapies have demonstrated efficacy in managing Sch-PAH; however, the long-term prognosis is significantly influenced by the occurrence of the first morbidity event. Early morbidity events, such as hospitalization or disease progression (defined as a decrease of at least 15% in the 6-min walk distance, the need to add a new PAH medication, or worsening of functional class), are associated with an increased mortality risk [17].

4 | Current Challenges in Diagnosing Schistosomiasis and Sch-PAH

Patients with schistosomiasis may be asymptomatic or present with nonspecific symptoms, such as anemia and asthenia. Acute bleeding can be the first clinical presentation. Low platelet and leukocyte counts are common diagnostic clues, while normal or mildly elevated liver enzyme levels are typically observed. Liver ultrasound is the primary imaging study, with periportal fibrosis being highly suggestive of schistosomiasis. Direct tests for schistosomiasis involve the microscopic examination of stool and urine, while indirect methods include immunological tests, such as ELISA. However, these methods are not sensitive when the infection burden is low. Serological tests can identify past exposure to the parasite, but their sensitivity decreases over time, making it challenging to confirm past infections. Liver biopsies, whether percutaneous or surgically obtained, are rarely performed and are reserved for atypical cases [20].

Sch-PAH remains an underdiagnosed condition largely due to delayed symptoms and a lack of screening programs. The most commonly used diagnostic model for Sch-PAH is illustrated in Figure 1 [2]. The signs and symptoms of Sch-PAH are nonspecific, similar to other forms of PAH. Some authors point out that pulmonary artery dilation is a suggestive feature of Sch-PAH [21]. Previous studies have demonstrated that pulmonary artery enlargement is significantly more pronounced in patients with Sch-PAH compared to those with IPAH, irrespective of the degree of mean pulmonary arterial pressure (mPAP) elevation and may represent an adaptive response to longstanding disease [22, 23].

Serologic testing may reflect past exposure or previous infection and, therefore, can remain positive even after treatment. The use of cathodic urine antigen testing for active infection may also lead to false-positive results after prior treatment. Persistent low-level antigenemia due to lingering antigens from dying or

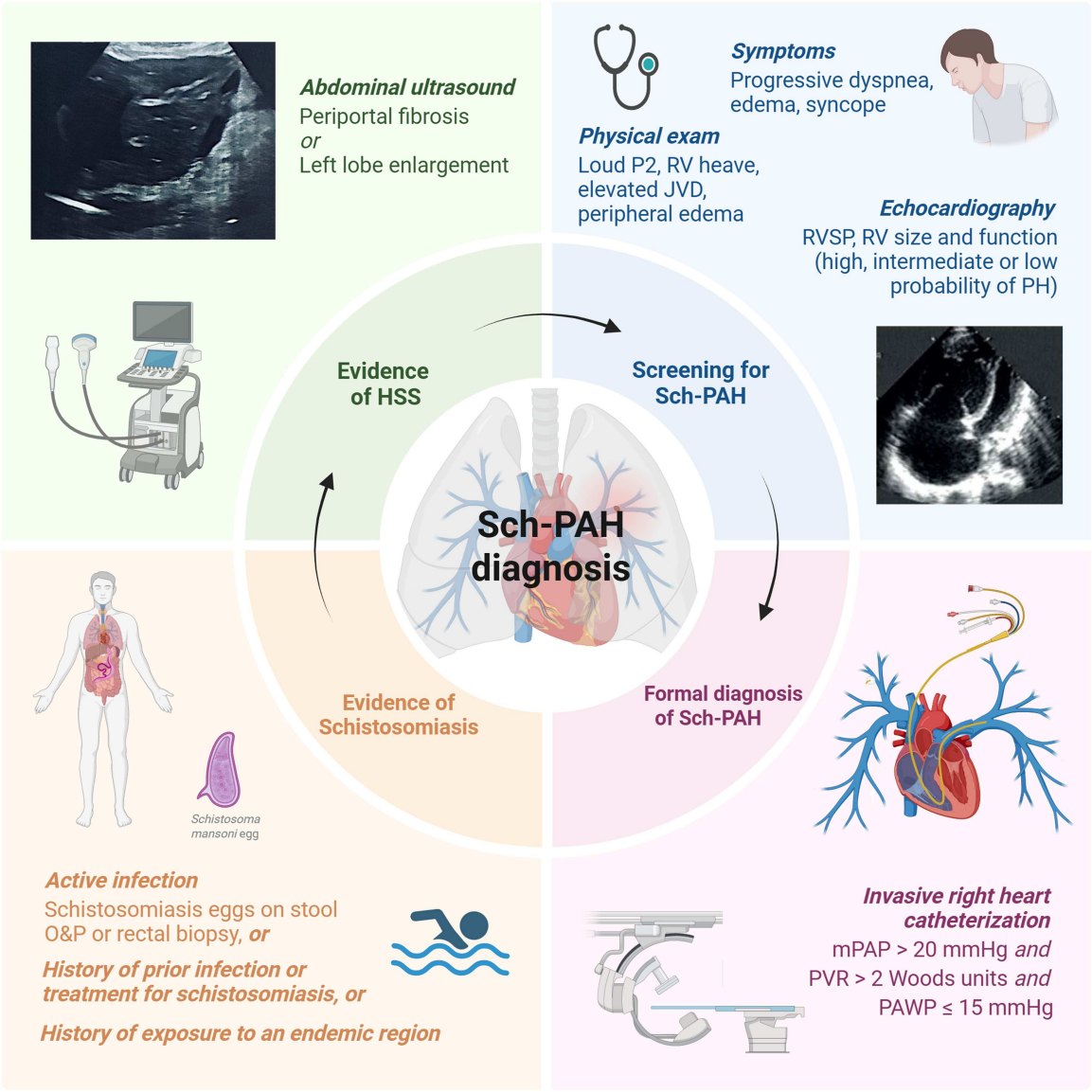

FIGURE 1 | Overview of the diagnostic workup for Sch-PAH in patients presenting with symptoms suggestive of pulmonary arterial hypertension. HSS, hepatosplenic schistosomiasis; P2, pulmonic component of second heart sound; RV, right ventricle; JVD, jugular vein distention; RVSP, right ventricular systolic pressure; PH, pulmonary hypertension; mPAP, mean pulmonary artery pressure; PVR, pulmonary vascular resistance; PAWP, pulmonary arterial wedge pressure.

dead worms or incomplete parasite clearance may be misinterpreted in high-burden or reinfection settings [24].

Although liver ultrasonography is used to detect fibrosis or abnormalities indicative of schistosomiasis-related liver damage, challenges arise when fibrosis is absent after praziquantel treatment. Other noninvasive imaging tests may be complementary tools for detecting hepatosplenic involvement. Liver elastography is a technique that measures liver tissue stiffness to detect, stage, and monitor hepatic fibrosis and cirrhosis: this test is often at the high end of the normal range in hepatosplenic schistosomiasis. Magnetic resonance imaging can detect periportal fibrosis, splenomegaly, and portal hypertension in hepatosplenic disease [25, 26].

Routine echocardiography, a noninvasive, cost-effective tool, is recommended for early detection of Sch-PAH in endemic regions, particularly in resource-limited settings. However, it remains an open question whether screening should be performed among individuals with risk factors for Sch-PAH, such as patients with hepatosplenic disease, portal hypertension, or long-standing schistosomiasis infection, or whether it should be limited to those presenting with PAH-related symptoms. Also, echocardiography can both overestimate and underestimate pulmonary pressures, leading to false positives and negatives. Considering its limited sensitivity and specificity for diagnosing Sch-PAH, a definitive diagnosis of Sch-PAH still requires right heart catheterization (RHC), the gold standard for confirming precapillary pulmonary hypertension [2]. Unfortunately, RHC is usually not available in resource-limited settings where schistosomiasis is endemic.

Distinguishing Sch-PAH from other portal hypertension-related conditions, including portopulmonary hypertension (PoPAH), remains a diagnostic challenge that continues to engage both clinicians and researchers. Individuals with PoPAH have higher cardiac output and vascular shear stress than patients with Sch-PAH, as well as less prominent pulmonary vascular inflammation [27]. Exercise imaging and hemodynamic studies may help identify the clinical and pathophysiological differences between Sch-PAH and PoPAH. Distinct cardiovascular responses and hemodynamic profiles during physical exertion may uncover functional and pulmonary vascular reserve differences between both conditions that are not always apparent at rest, offering insights into their underlying pathophysiology, disease severity, and prognostic trajectories [18, 28].

Differentiating between Sch-PAH and IPAH is also challenging due to overlapping clinical features [19]. The diagnostic foundation for Sch-PAH rests upon two pillars: a documented clinical history of schistosomiasis exposure and hepatosplenic imaging findings indicative of chronic parasitic disease, such as periportal fibrosis and left lobe enlargement. Of note, some Sch-PAH patients exhibit severe pulmonary hypertension without detectable eggs in stool samples, possibly due to prior treatment with praziquantel [2].

5 | Preventive Aspects of Schistosomiasis in Brazil and Its Impact on PAH Development

The potential benefit of population-wide antiparasitic interventions in reducing the incidence of Sch-PAH remains uncertain. Sch-PAH may persist even after schistosomiasis treatment due to irreversible vascular damage. Nevertheless, antiparasitic therapy with praziquantel is generally recommended in patients with Sch-PAH to eradicate any residual infection and prevent ongoing egg deposition. However, the efficacy of praziquantel in reducing the severity of established Sch-PAH is uncertain, as many patients do not carry worms at the time of pulmonary hypertension diagnosis [2]. PAH-specific treatment in Sch-PAH may help prevent further antigenic stimulation and additional egg embolization, potentially stabilizing disease progression even if significant hemodynamic improvement is unlikely. Therefore, management of Sch-PAH relies primarily on PAH-targeted therapies.

Multidisciplinary care for schistosomiasis involving pulmonologists, cardiologists, hepatologists, and primary care physicians, as well as better screening programs in high-risk populations, is essential for timely and accurate Sch-PAH diagnosis, targeted treatment, and improved outcomes. This may be successfully implemented by training healthcare professionals how to detect early signs of PAH, establishing routine echocardiographic screening in endemic areas, developing clear referral pathways to PAH reference centers, and using telemedicine to expand access to specialists in resource-limited settings.

However, the diagnostic imperative of RHC for Sch-PAH confirmation stands in stark contrast to the disease's epidemiological reality: schistosomiasis-associated PAH predominantly burdens low-income endemic regions lacking access to invasive hemodynamic procedures, a critical limitation that fundamentally compromises diagnostic accuracy and clinical outcomes. Therefore, it is imperative that the pulmonary vascular disease community, such as through the PVRI iPVD Consortium, work collaboratively to develop and implement noninvasive, cost-effective Sch-PAH screening and diagnostic programs tailored to resource-limited settings to bridge this critical diagnostic gap and enable earlier disease recognition in vulnerable populations in Brazil and worldwide. Preventive aspects and other priority areas requiring further study are outlined in Table 2.

6 | Conclusion

Schistosomiasis is a neglected tropical parasitic disease, whose hepatosplenic form can evolve into Sch-PAH. Schistosomiasis remains a leading cause of PAH in Brazil. There are significant diagnostic challenges related to the inherently low sensitivity and specificity of current screening modalities for schistosomiasis, which directly contribute to substantial diagnostic uncertainty. Liver abnormalities are critical diagnostic clues but are not always present. The difficulty in distinguishing between active and past infections using serological tests represents another challenge for diagnosis, as well as limited public health policies for systematic screening in high-risk populations. Clinical manifestations of Sch-PAH can potentially vary by geographic region, due to genetic differences, environmental factors, and access to healthcare. Comprehensive epidemiological studies across diverse endemic areas are imperative to unravel this Sch-PAH phenotypic diversity, advance our global understanding of disease pathogenesis and clinical presentation, and ultimately improve disease recognition worldwide. Parallel efforts must also address the critical gap in access to RHC in resource-limited endemic regions, as innovative noninvasive screening algorithms and biomarker strategies should be developed to complement or precede invasive hemodynamic assessment in settings where such procedures remain unavailable.

TABLE 2 | Future research directions in Sch-PAH.

Research area	Key research questions
Disease mechanisms and pathogenesis	Which parasite-derived factors, host cellular responses, and inflammatory mediators drive pulmonary vascular remodeling and disease progression in Sch-PAH?
Biomarkers and early detection	Which biomarkers could improve early diagnosis and risk stratification in Sch-PAH?
Clinical phenotyping	How does disease progression in Sch-PAH differ from other PAH etiologies, such as PoPAH or IPAH?
Therapeutic strategies	Can targeting parasite-driven inflammation or combining antiparasitic treatment with PAH therapies improve clinical outcomes in Sch-PAH?
Prevention and early intervention	Can screening and early treatment of schistosomiasis prevent pulmonary vascular remodeling and reduce the risk of developing Sch-PAH?

Abbreviations: IPAH, idiopathic pulmonary arterial hypertension; PoPAH, portopulmonary hypertension.

Addressing these challenges demands increased clinical awareness, improved diagnostic tools, and advocacy for public health initiatives targeting both prevention and early detection. This strategic agenda requires continued research to elucidate genetic predispositions, immunological responses, and epidemiological patterns across diverse populations. Specifically, prospective cohort studies in endemic areas should prioritize early detection of Sch-PAH through standardized screening, combined with advanced diagnostics. Integration of novel serological and molecular tools with imaging and biomarker data is essential to establish disease prevalence estimates, identify predictors of progression, and reliably distinguish Sch-PAH from other PAH etiologies such as PoPAH or IPAH. Furthermore, longitudinal comparisons across PAH etiologies potentially enhanced by machine learning approaches, could refine diagnostic algorithms and facilitate phenotype-specific management strategies—a vision that the 2025 PVRI iPVD Consortium meeting in Rio has begun to crystallize through its collaborative efforts toward standardized international diagnosis and surveillance frameworks.

The 2025 PVRI iPVD Consortium meeting in Rio represented a pivotal step toward establishing an international framework for standardized Sch-PAH diagnosis and surveillance through structured collaborative efforts. Moving forward, sustained collaboration among local clinicians, international researchers, and public health authorities across endemic regions will be essential to transform current diagnostic and epidemiological uncertainties into actionable clinical strategies and, ultimately, to reduce the global burden of this parasitically driven pulmonary vascular disease.

Author Contributions

All authors contributed to the conception, literature search, writing, and revision of this review article.

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Ethics Statement

The authors have nothing to report.

Conflicts of Interest

The authors declare no conflicts of interest.

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