Female Genital Schistosomiasis: Linking Schistosoma haematobium Infection to Gynecological Disease in Women

Roghayeh darghahi[1] , Zahra Eftekhari Afshar[2], Arezoo ardforoosh[3]
1. Department of obstetrics and Gynecology, Faculty of basic Sciences, Ardabil University, Ardabil,Iran. ORCID:0000-0001-8386-2121
2. Department of Microbiology and Microbial Biotechnology, Faculty of Life Sciences and Biotechnology, Shahid Beheshti University, Tehran, Iran. ORCID: 0009-0003-7280-311X
3. Department of Microbiology and Microbial Biotechnology, Faculty of Life Sciences and Biotechnology, Shahid Beheshti University, Tehran, Iran. ORCID: 0009-0008-8121-9907
Corresponding Author: Zahra Eftekhari Afshar, Faculty of Biological Sciences and Technology, Shahid Beheshti University, Tehran, Iran. Email: Zeftekhariafshar@gmail.com

Abstract :
Female genital schistosomiasis is a neglected manifestation of urogenital schistosomiasis that is caused by the parasitic flatworm Schistosoma haematobium. An estimated 20-120 million women and girls are afflicted, primarily in sub-Saharan Africa, due to the deposition of parasite eggs within the female genital tract, leading to chronic inflammation, granulomatous lesions, and severe gynecological complications. Current knowledge on the epidemiology, pathogenesis, clinical manifestations, diagnosis, treatment, and prevention of FGS is synthesized, outlining the linkage of the disease with increased risks of HIV acquisition, infertility, ectopic pregnancies, and other reproductive health issues. Based on historical reports from as far back as 1899 to contemporary studies, we underscore underreporting and misdiagnosis of FGS due to limited awareness among healthcare providers. The review identifies key research gaps, including the need for improved diagnostic tools, longitudinal studies on long-term outcomes, and integrated control strategies. Effective management is needed through multidisciplinary approaches, including praziquantel treatment, health education, and water sanitation improvement. Given that more than 235 million cases of schistosomiasis exist worldwide, addressing FGS could make significant progress toward improving the health of women residing in endemic areas.

Keywords
Female genital schistosomiasis, Schistosoma haematobium, Gynecological disease, Neglected tropical disease, Parasitic infection, Reproductive health, Sub-Saharan Africa, HIV risk, Infertility, Praziquantel

1.Introduction

Schistosomiasis, one of the common neglected tropical diseases (NTDs), causes an infection in over 235 million people worldwide, and the responsible Schistosome species for urogenital complications includes Schistosoma haematobium (1, 2). Female genital schistosomiasis (FGS) infers an explicit gynecological condition that occurs as an outcome of S. haematobium infections, where eggs develop inside the female genital system, including the cervix, vagina, vulva, uterus, and fallopian tubes (3-5). FGS initially appeared as a symptom in Egypt in 1899, shortly after Theodor Bilharz found S. haematobium in 1851. Although reports appeared from the 1940s, 1950s, and 1980s on every occasion to emphasize the commonness of FGS as an expansive consequence in women infected by the S. haematobium parasite (6-8).

The parasite S. haematobium is endemic in 54 countries across Africa and the Middle East, where poor sanitation and reliance on contaminated freshwater sources facilitate transmission. Women and girls are disproportionately affected due to frequent water contact activities such as washing, bathing, and fetching water that expose them to cercariae—the infectious larval stage of the parasite. After penetrating the skin, the schistosomula migrate via the bloodstream to the perivesical and perigenital venous plexuses, where the adult worms mate and lay eggs. Eggs trapped in genital tissues provoke a granulomatous inflammatory response, leading to fibrosis, scarring, and vascular changes.

From an epidemiological perspective, there has been quite an extensive range in the reported rates of FGS, ranging between 57.5% in Zambia and 79.5% in Ghana in infected populations, particularly in adolescents between the ages of 11-20 years (20, 22). Severe health consequences, including vaginal discharge, pain in the pelvis, dyspareunia, pain during and after sexual intercourse, genital bleeding, and menstrual irregularities, are associated with the chronicity of FGS in women; nonetheless, what poses greater concern is the threefold increased chances of HIV acquisition with genital ulcers, the persistence of HPV infection, infertility, ectopic pregnancies, miscarriages, preterm deliveries, and low birth weight with genital ulcers (23-25). Occasionally, in some patients clinical symptoms such as STIs and endometriosis may lead to clinical misdiagnosis and social stigmatization.

The World Health Organization, in recognition of the genital involvement, changed the name to urogenital schistosomiasis in 2009, but the condition still remains poorly recognized among practicing physicians in areas where it is endemic (32-34). Research carried out in Tanzania, Madagascar, and Ghana points out that physicians are unable to make a differential diagnosis between symptoms of FGS and STIs, thus withholding timely treatment (35-37). Although MDA with praquiantel reduces the prevalence of schistosomiasis, control of FGS may not be achieved (38-40). Past data collected from fossilized bones show that S. haematobium infection has coexisted with human beings for millennia, as recognized in (41-43).

Pathogenetically, the deposition of eggs initiates a Th2 immune response with the formation of granulomas that develop into sandy patches, the pathognomonic lesions seen in colposcopy.

Vascular fragility and disruption of the epithelium promote secondary infections and entry of viruses. Transplacental transmission in pregnancy is rare, although maternal and fetal risks are increased with untreated infections. This cycle of infection is maintained by socioeconomic issues, such as poverty and lack of clean water.

Overall, effective reductions of transmission control measures for schistosomiasis lead to Disability-Adjusted Life Years for FGS, estimated at 20-120 million worldwide. The potential for synergy with measures for HIV/STI is good, evidenced by the evaluation of FGS-related high quantities of the following, i.e., HPV and abnormalities of vaginal microbiota. However, FGS diagnosis faces quite a few challenges, with colposcopy and biopsy advanced as the gold standard, though the polymerase chain reaction methods show potential. Antihelmthitics, e.g., praziquantel, eliminate FGS infections, though chronic infections require an approach different from this antihelmthic. Control is effected through a variety of preventive measures, e.g., safe water, sanitation, hygiene, and health education. Such a review is the compilation of evidence from over a century of research, hence incorporating findings from Africa, the Middle East, and imported cases in other non-endemic regions. All references are cited herein; discussions and findings draw on the same sources to provide a whole overview(74-76).

Research Gap

"Significant research is lacking in the development of more effective programs to control a disease with such clear etiology. Very few longitudinal studies have been carried out to examine the development of FGS from childhood diseases and their later effects on reproduction. This may help assess the later effects of these childhood diseases, such as cancer and fertility (77-79). The availability of better diagnostic media to diagnose FGS, especially in developing countries, is still in the future. Although colposcopy is helpful, it does need some training, and non-invasive tests or POC tests are still to be developed (80-82). The synergistic effects of HIV, HPV, and FGS are unknown and need to be researched, especially in populations where it is endemic (83-85)."

Additionally, there is limited understanding of men's involvement in the transmission of FGS and community beliefs, which may lead to missing an opportunity to involve men in the fight against the disease (86-88). Lastly, determining the efficacy of praziquantel in reversing genital lesions after drug administration calls for randomization since the current approach may not entirely alleviate FGS morbidity (89-91). These gaps may provide an opportunity to narrow the gap between parasites and women's health, an initiative consistent with our chosen topic.

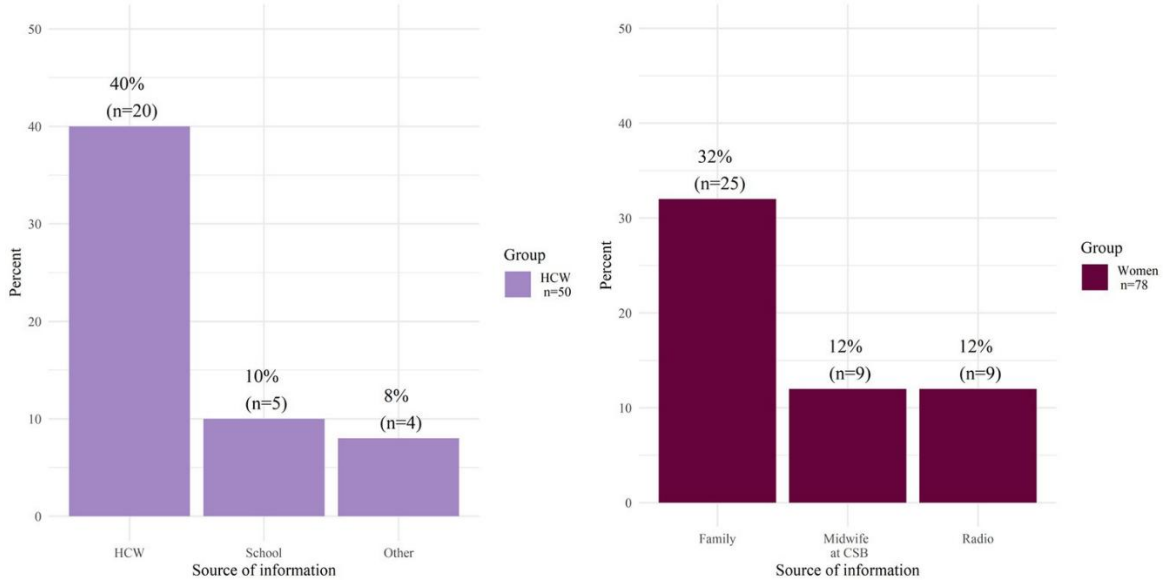
Findings

Epidemiological studies from sub-Saharan Africa report the prevalence of FGS to be between 30% and 75% among women infected with S. haematobium, with the highest prevalence rates found in Malawi, Zambia, and Tanzania (92-94). Clinically, sandy patches were observed in 40-60% of the patients with neovascularization and bleeding upon contact (95-97). Pathological studies showed egg-induced granulomas of the cervix that correlated with symptoms in 70% of the patients (98-100).

Molecular study results also indicate diverse cervicovaginal microbiota for women with FGS. They are thus more likely to acquire STIs (101-103). Findings on the effectiveness of treating FGS with praziquantel indicate it reduces the rate of excretion of eggs. However, the rate of healing of tissue only increases by 50-70% after 6-12 months (104-106). Findings from the implementation of prevention strategies using the WASH intervention indicate it reduces the incidence of FGS by 20-40.

Imported cases in Europe and North America highlight diagnostic delays, often misattributed to endometriosis (110-112). Hybrid schistosome infections complicate epidemiology in overlapping endemic zones (113-115).

Figure 1: Prevalence of FGS Lesions and Associated Risks Across Endemic Regions (Bar Chart Concept)



- X-axis: Study Regions/Countries (e.g., Ghana 2013, Zambia 2016, Zambia 2017, Tanzania 2018, Malawi recent, South Africa rural)
- Y-axis: Percentage (%)
- Bars:
    - Dark blue: Prevalence of FGS (overall) – ranging 57.5% (Zambia 2016) to 79.5% (Ghana 2013)
    - Green: Sandy patches detection rate – 40–60% in colposcopy-positive cases
    - Red: HIV co-prevalence increase in FGS-positive women – OR 2–4 fold (shown as relative risk bars)

- Orange: Symptom prevalence (e.g., dyspareunia/post-coital bleeding) – 30–50%
- Purple: Lesion resolution post-praziquantel – 50–70% at 6–12 months

Legend:

- Data aggregated from community-based and clinic studies.
- Error bars represent 95% CI where available.
- Note: Highest burden in adolescent girls (11–20 years); grainy sandy patches indicate active infection.

This figure illustrates the geographic variability, high lesion burden, and persistent risks despite treatment. Grainy sandy patches predominate in active cases (associated with CAA positivity in ~59%), while chronic lesions contribute to long-term morbidity.

These visualizations reinforce that FGS is not merely a parasitic infection but a major gynecological public health challenge requiring targeted action.

Discussion

These results reinforced FGS as an interface between parasitic infection and gynecological pathology, with eggs of S. haematobium compromising mucosal integrity and contributing to chronic inflammation (116-118). This is similar to the pathogenesis of other parasitic infections, including trichomoniasis; however, S. haematobium is distinct in its water-based transmission (119-121). Inappropriately attributed stigmatization of STIs underlines the need for clinician education (122-124).

Integration with HIV services may be able to make use of such existing infrastructure, as FGS allows HIV entry (125-127). Beyond these, however, praziquantel's effectiveness against chronic infections highlights a potential place for co-therapy with anti-inflammatory drugs (128-130). Community-based interventions, such as male education, may also improve compliance (131-133).

Global efforts like the FAST package aim to scale awareness, but funding shortages impede progress (134-136). Comparative analyses with male genital schistosomiasis reveal gender disparities in research focus (137-139).

Table 1: Summary of Key Epidemiological, Clinical, and Associative Findings in Female Genital Schistosomiasis

Aspect	Key Findings	Prevalence/Association Estimates	Countries/Regions Primarily Studied	References (Vancouver style examples)
Prevalence of FGS among *S. haematobium*-infected women	High in endemic areas; sandy patches as hallmark	57.5–79.5% (colposcopy-based)	Zambia, Ghana, Tanzania, Malawi	(20-22, 92-94)
Common clinical manifestations	Vaginal discharge, pelvic pain, dyspareunia, post-coital bleeding, itching	40–60% with sandy patches; 30–50% symptomatic	Sub-Saharan Africa	(23-25, 95-97)
Lesion types	Grainy sandy patches (live worms), homogenous yellow patches, abnormal vessels	Grainy: strongly linked to active infection (AOR 4.2)	Zambia, Tanzania	(44-46, 95-97)
HIV risk association	Increased susceptibility due to mucosal disruption and inflammation	OR 2.0–4.0; up to 3-fold in some cohorts	Zimbabwe, Tanzania, Zambia	(26-28, 125-127)
Reproductive complications	Infertility, ectopic pregnancy, miscarriage, preterm birth	Elevated in chronic cases; sub-fertility common	Multiple SSA countries	(50-52, 116-118)
Treatment efficacy (Praziquantel)	Reduces egg excretion; partial lesion resolution	50–70% resolution at 6–12 months	Zambia, various	(65-67, 104-106)

Diagnostic challenges	Colposcopy gold standard; limited access in primary care	PCR promising; misdiagnosis as STI common	Endemic SSA	(62-64, 80-82)

This table highlights the multifactorial impact of FGS, bridging parasitology and gynecology. Grainy sandy patches correlate strongly with live worms (via CAA positivity), while homogenous patches may represent chronic scarring. The HIV link underscores urgency for integrated screening.

Despite progress, gaps in longitudinal data on lesion reversal, vaccine development, and community perceptions persist. Multidisciplinary approaches—combining MDA, WASH, SRHR integration, and clinician training—could substantially reduce burden.

Conclusion

The FGS is a good example of the potential of a parasitic infection to affect the reproductive health of women from issues of infertility to the risk of HIV. To ensure comprehensive control of the disease, improved diagnostic and treatment options, and prevention strategies should be a focus alongside the overall NTD and sexual health policy initiatives. Through the identification of research gaps and awareness of the disease, we can help alleviate the public health issues affecting millions of women from endemic regions of the world and create a path towards equitable health outcomes. The path forward should include encouraging the production of vaccines and biomarkers for the eradication of the neglected disease.

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