



Pelvic Atherosclerosis in Women: A Case Report of the Alleviation of Dyspareunia and Vaginal Dryness after Pelvic Artery Revascularisation

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Abstract

Misdiagnosed and inadequately treated vasculogenic sexual dysfunction affects millions of females worldwide, highlighting the importance of a mostly overlooked pathology. Organic female sexual dysfunction, such as vaginal engorgement and clitoral erectile insufficiency, may be associated with hypogastric atherosclerosis.

In the author's vascular medicine setting, they encountered a case of a post-menopausal female patient with symptomatic common iliac and hypogastric artery occlusion. This case highlights the diagnostic management of atherosclerosis and its secondary clinical manifestations, the differentiation between physiological post-menopausal hormonal changes and pathological vasculogenic dysfunction, as well as currently available endovascular treatment options.

The 57-year-old patient presented with symptoms indicative of bilateral peripheral artery disease, Fontaine Stage IIb, along with vaginal dryness and dyspareunia. Upon finding occlusions of the common iliac and hypogastric artery, bare metal stenting of the left distal common iliac artery and percutaneous transluminal angioplasty of the internal iliac artery was performed. At 6-week follow-up, an unlimited pain-free walking distance as well as greatly improved vaginal and clitoral erectile function was reported.

Local oestrogen application for a year showed no clinical improvement, whereas a significant difference in vaginal and clitoral erectile function was reported shortly after endovascular revascularisation. Vaginal engorgement and clitoral erection depend on increased blood inflow and pulsatile, non-calcified arterial walls. In females with cardiovascular risk factors, arterial insufficiency of the hypogastric-cavernous bed is an underestimated atherosclerotic manifestation for sexual impairment. In these patients with arteriogenic sexual dysfunction, endovascular therapy using drug-eluting stents may be considered as an effective treatment option.

Key Points

1. Vasculogenic female sexual dysfunction, a frequently overlooked consequence of atherosclerosis, impacts millions of women worldwide, leading to significant quality-of-life deterioration, especially in postmenopausal women with cardiovascular risk factors.
2. This case report examines a 57-year-old postmenopausal woman with common iliac and hypogastric artery occlusions, illustrating how endovascular revascularisation can address vasculogenic sexual dysfunction and peripheral artery disease symptoms.
3. Endovascular revascularisation emerges as a promising intervention for women with vasculogenic sexual dysfunction, providing symptom relief by restoring pelvic blood flow and highlighting the need for recognising cardiovascular contributions to female sexual health issues.

INTRODUCTION

Pathophysiology and Aetiology of Female Sexual Dysfunction

Vasculogenic female sexual dysfunction syndromes affect millions of ageing women worldwide, whose day-to-day symptoms of coital vaginal discomfort, vaginal dryness, and diminished sexual arousal are underdiagnosed and often inadequately empirically treated with hormone supplementation or simply artificial lubrication.¹ As early as 1997, Park et al.² demonstrated the root of vaginal and clitoral engorgement insufficiency to be of haemodynamic origin, showing significant changes in pelvic nerve-stimulated vaginal haemodynamics such as a decrease in blood flow and vaginal wall pressure in cases of atherosclerotic cavernosal arteries and diffuse vaginal and clitoral fibrosis. Female sexual response is assessed by the appearance of vaginal lubrication and clitoral tumescence, both associated with autonomic nerve-stimulated local increases in blood flow.³ The aetiology of female sexual dysfunction (FSD) is multifactorial, being associated with various hormonal and urogynaecological abnormalities, psychological issues, and degenerative systemic pathologies, such as metabolic syndrome and cardiovascular disease.^{4,5}

Comparable to male erectile dysfunction (ED), the incidence of FSD has been demonstrated to rise with age and correlate bidirectionally with the existence of cardiovascular comorbidities such

as coronary heart disease, myocardial infarction, and stroke.^{6,7} Both conditions, vasculogenic erectile dysfunction and cardiovascular disease, are known to be associated with common predisposing risk factors such as arterial hypertension, obesity, diabetes mellitus, hypercholesterolemia, and smoking, while exhibiting similar underlying pathologies in terms of inflammation and sclerosis of arterial vessels leading to severe endothelial dysfunction.^{7,8} Atherosclerosis of the hypogastric-cavernous arterial bed in predisposed female patients may be an important pathophysiological factor causing organic vasculogenic erectile female sexual dysfunction.

Previous FSD treatment attempts included a range of approaches such as experimental hormone and pharmaceutical therapy, localised mechanical appliances in terms of a vacuum constriction device, and cognitive-behavioural therapy. All in all, results were often inconsistent and none of the above proved to be universally effective. By focusing on the haemodynamic origins and importance of vasculogenic etiological factors of FSD, we aimed to present and further analyse a novel therapeutic approach.

CASE DESCRIPTION

The authors present the case of a 57-year-old woman with a history of hypercholesterolemia, obesity, and smoking. Besides the above-mentioned,

no other cardiovascular risk factors were reported. The patient initially presented in January 2023 with symptoms indicative of bilateral peripheral artery disease Fontaine Stage IIb along with vaginal dryness and dyspareunia for the past year. Prior to endovascular therapy, her female sexual function index (FSFI) was as low as 30, indicating extensive deductions in the domains of desire, arousal, lubrication, orgasm, and satisfaction. Local oestrogen application prescribed by her gynaecologist brought no noticeable clinical improvement. Following a non-invasive vascular workup with oscillography and duplex ultrasound suspecting occlusions of the common iliac and hypogastric artery, bare metal stenting of the left distal common iliac artery and percutaneous transluminal angioplasty of the internal iliac artery was performed. Dual antiplatelet therapy was prescribed over the course of 4 weeks post-interventional.

After an initial period of clinical improvement, the patient's initial complaints of peripheral leg pain after walking short distances as well as the above-described sexual dysfunction re-appeared about 1 year after the initial intervention. Duplex ultrasound confirmed the suspected diagnosis of an in-stent thrombosis in the left common iliac artery. Rotational thrombectomy using a 6F Rotarex® catheter (Rotarex, Luxembourg) was performed, followed by the placement of bare metal stents in the left common and external iliac artery and recanalisation with drug-eluting stent placement in the left internal iliac artery. At the 6-week follow-up, the patient reported an unlimited pain-free walking distance as well as greatly improved vaginal and clitoral erectile function with an FSFI score of 80 points (indicating a maximum score of 5/5 points in the domains of desire, arousal, lubrication, orgasm, and satisfaction, whilst reporting 0/5 points in the aspect of experienced pain). Duplex ultrasound confirmed patency of the common iliac and hypogastric artery. Dual platelet inhibition using aspirin and clopidogrel was prescribed to be taken until the 3-month follow-up appointment.

DISCUSSION

It is well documented that symptoms typical of FSD increase sharply following menopause due to pathophysiological changes in vaginal tissue associated with oestrogen deprivation.²

Post-menopausal women, as well as those predisposed by the existence of cardiovascular risk factors, report noticeably more complaints of vaginal and clitoral dysfunction compared to pre-menopausal counterparts or women of the same age group devoid of cardiovascular risk factors.⁹ Nevertheless, a multicentre Latin American study using the FSFI, a 19-item multidimensional self-reporting measure on primary components of female sexual function quantifying potential dysfunction in six domains (desire, arousal, lubrication, orgasm, satisfaction, pain), as the main validation instrument, showed an unusually young group of women with Type 2 diabetes displaying a high prevalence of sexual dysfunction, with a decrease in vaginal lubrication being the most important associated symptom.⁴

Similar observations were made by a North American cross-sectional cohort study with vaginal dryness due to impaired spontaneous lubrication and a consecutive decline in the number and intensity of reported orgasms being more common amongst insulin-treated diabetic patients compared to non-diabetic women.¹⁰

The role of long-term hormone supplementation in maintaining female sexual health has been widely established. However, in view of the above, additional approaches are worth exploring. Although a vast majority of women discontinuing postmenopausal oestrogen intake develop signs of vulvovaginal atrophy, most epidemiological studies show only little increase in pre-existing dyspareunia with age,¹¹ therefore, pointing towards a primary underlying pathology of the vaginal and clitoral arterial bed, with, irrespective of postmenopausal status, well known vascular pathogens such as dyslipidaemia being confirmed an independent risk, causing

local atrophy and the subsequent clinical symptoms.¹²

In the case of this patient, local oestrogen application over the course of 1 year showed no clinical improvement, whereas a significant difference in vaginal and clitoral erectile function was reported shortly after the procedure. Vaginal engorgement and clitoral erection depend on increased blood inflow and pulsatile, non-calcified arterial walls.¹² Atherosclerosis, strongly associated with cardiovascular risk factors, especially in older patients, increasingly affects a younger population.¹³ Consequently, the age at which secondary pathologies manifest has steadily decreased within the last decade, posing a significant and long-term threat to the individuals' quality of life if not diagnosed and treated early.¹⁴ The prevalence of male erectile dysfunction, especially of vasculogenic aetiology, has increased considerably worldwide, now considered an indicator and independent predictive marker for a disease of generalised atherosclerosis with, in turn, high cardiovascular morbidity and mortality with cardiovascular events.⁷ Besides nicotine abuse, diabetes mellitus is understood to be one of the most important risk that can be influenced; affected patients have shown up to a four-fold increased risk of developing vasculogenic sexual dysfunction.^{9,15}

There is a significant disparity in recognition of vasculogenic sexual dysfunction among genders. The difference in awareness and subsequent diagnosis can largely be attributed to historical and cultural biases in research and clinical practice, in addition to the complexity of female versus male sexual anatomy and reproductive health.

By increasing the index of suspicion for vasculogenic causes of FSD, a significant gap in sexual health care is newly addressed, advocating for heightened awareness among general practitioners to assess for atherosclerosis in women with FSD; as well as to adopt a more proactive approach in evaluating FSD overall, especially in women presenting with risk factors for cardiovascular disease.

It is likely that in the case of this patient, interventional treatment could have been avoided if long-term preventative measures ensuring overall vascular health including weight loss, cessation of smoking, and statin therapy had been implemented early on. Pharmacological administration of vasodilators such as phosphodiesterase-5-inhibitors (PDE-5-I), a routine primary approach in male patients with ED, has not been routinely implemented in the treatment of vasculogenic FSD, and requires further studies.

Going forward, as patients typically do not report their symptoms until the condition has progressed severely, a wider implementation and optimisation of primary and secondary preventive approaches is needed. Encouraging a healthy diet and regular exercise; routine blood sugar and lipid profile checks; and screening and assessment of potential atherosclerotic lesions in other arterial beds such as the lower limb, carotid, and coronary arteries are examples of necessary steps to be taken.

As the most frequent underlying cause is observed to be of vascular origin, a swift path to a confirmed diagnosis is crucial to monitor existing as well as potential risk factors as predictors of future burden of disease to identify patients at risk of severe cardiovascular events early.¹⁶

Male patients with severe atherosclerotic ED unresponsive to PDE-5-Is have been shown to profit greatly from endovascular drug-eluting stent therapy of erection-related arteries.¹⁷ Stable clinical outcomes were reported beyond a 1-year timeframe, proving the safety and effectiveness of this approach especially in long-term follow-up.¹⁸ While the above-described case is a novelty in the field of interventional revascularisation procedures, it demonstrates current and developing endovascular therapy as an effective treatment option for pelvic atherosclerosis in its various clinical manifestations with the overall goal of routinely practised and widely accepted secondary preventive approaches.

A notable limitation of the analysis is the reliance on references that are not recent. This is primarily due to the scarcity of the current research environment. While older studies provide a foundational understanding, the scarcity of recent publications highlights the necessity for further investigation and research of a pathology becoming increasingly frequent. Also to be mentioned is the fact that insights gained from the patient case, while representing a common pathology, may not be generally applicable to primary treatment options of other patients with FSD. The authors' analysis underscores the importance of early preventative measures; however, does not extensively explore potential barriers to implementation or how to effectively encourage patient adherence.

CONCLUSION

Organic sexual dysfunction has a significant impact on the quality of life. Despite vascular pathologies being the most frequent as well as clinically well-manageable aetiological factor, the relevance of cardiovascular integrity in female sexual health remains largely unrecognised. Once clinically manifested, endovascular revascularisation is effective and successful in restoring arterial blood flow of inner pelvic organs and may be a promising minimal-invasive treatment option for patients with severe arteriogenic FSD, improving symptoms within weeks of the intervention. Given the established direct correlation between atherosclerosis and vasculogenic sexual dysfunction, it is imperative for clinicians to consider atherosclerotic disease as

a potential underlying cause of FSD in appropriate patients. Further studies are warranted to assess the role of endovascular therapy on the short- and long-term clinical improvement of FSD of atherosclerotic origin.

PATIENT PERSPECTIVE

The symptoms started over a year ago. First I noticed reoccurring leg pains after reaching a certain walking distance; later on, I additionally experienced intimate discomfort. Initially, I did not know that these two symptoms could be connected and potentially both rooted in the same cause.

My gynaecologist explained that with age and menopausal hormonal changes, vaginal dryness and its consecutive discomfort during intercourse was very common, no reason to worry, and that the prescribed oestrogen cream would help treat the underlying cause of oestrogen-derived vaginal tissue. It was not until an examination revealed blockages in my arteries that it was explained to me that those symptoms might be due to a reduced blood flow in that specific area. After the first intervention and stent placement I noticed an improvement of all the above described quite quickly before the symptoms started to re-appear. Now, 6 weeks after the obstructed artery has been re-opened, I am walking entirely pain-free and have noticed a significant improvement in libido due to almost disappeared local discomfort.

References

1. Laumann EO et al. Sexual problems among women and men aged 40-80 y: prevalence and correlates identified in the Global Study of Sexual Attitudes and Behaviors. *Int J Impot Res.* 2005;17(1):39-57.
2. Park K et al. Vasculogenic female sexual dysfunction: the hemodynamic basis for vaginal engorgement insufficiency and clitoral erectile insufficiency. *Int J Impot Res.* 1998;10(1):67. Corrected and republished from: *Int J Impot Res.* 1997;9(1):27-37.
3. Berman JR. Physiology of female sexual function and dysfunction. *Int J Impot Res.* 2005;1(Suppl 1):S44-51.
4. Blümel JE et al. Sexual dysfunction in middle-aged women: a multicenter Latin American study using the Female Sexual Function Index. *Menopause.* 2009;16(6):1139-48.
5. Wheeler LJ, Guntupalli SR. Female sexual dysfunction: pharmacologic and therapeutic interventions. *Obstet Gynecol.* 2020;136(1):174-86.
6. Dilixiati D et al. Association between cardiovascular disease and risk of female sexual dysfunction: a systematic review and meta-analysis. *Eur J Prev Cardiol.* 2024;31(7):782-800.
7. Uddin SMI et al. Erectile dysfunction as an independent predictor of future cardiovascular events: the multi-ethnic study of atherosclerosis. *Circulation.* 2018;138(5):540-2.
8. Shin D et al. Erectile dysfunction: a disease marker for cardiovascular disease. *Cardiol Rev.* 2011;19(1):5-11.
9. Slob AK et al. Sexuality and psychophysiological functioning in women with diabetes mellitus. *J Sex Marital Ther.* 1990;16(2):59-69.

10. Copeland KL et al. Diabetes mellitus and sexual function in middle-aged and older women. *Obstet Gynecol.* 2012;120(2 Pt 1):331-40.
11. Basson R. Women's sexual function and dysfunction: current uncertainties, future directions. *Int J Impot Res.* 2008;20(5):466-78.
12. Baldassarre M et al. Impaired lipid profile is a risk factor for the development of sexual dysfunction in women. *J Sex Med.* 2016;13(1):46-54.
13. Sun J et al. Global, regional, and national burden of cardiovascular diseases in youths and young adults aged 15-39 years in 204 countries/territories, 1990-2019: a systematic analysis of Global Burden of Disease Study 2019. *BMC Med.* 2023;21(1):222.
14. Jacobs DR Jr et al. Childhood cardiovascular risk factors and adult cardiovascular events. *N Engl J Med.* 2022;386(20):1877-88.
15. Balletshofer B et al. Positionspapier zur diagnostik und therapie der peripheren arteriellen verschlusskrankheit (pAVK) bei menschen mit diabetes mellitus. *Der Diabetologe.* 2021;17(3):283-92.
16. Billups KL. Sexual dysfunction and cardiovascular disease: integrative concepts and strategies. *Am J Cardiol.* 2005;96(12B):57M-61M.
17. Mohan V et al. Long-Term outcomes of drug-eluting stent implantation for patients with atherosclerotic erectile dysfunction not responding to PDE-5-inhibitors. *J Endovasc Ther.* 2023;DOI: 10.1177/15266028231183775.
18. Schönhofen et al. Endovascular therapy for arteriogenic erectile dysfunction with a novel sirolimus-eluting stent. *J Sex Med.* 2021;18(2):315-26.