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Case Report

An uncommon companion to prolapse: Mönckeberg's medial calcific sclerosis of the uterine vasculature

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ABSTRACT

Mönckeberg's arteriosclerosis, or medial calcific sclerosis, is an uncommon, non-atherosclerotic form of vascular calcification affecting the tunica media of small- and medium-sized muscular arteries. It occurs independently of intimal atherosclerotic disease, is typically non-obstructive, and is most often encountered in the peripheral arteries of the lower limbs in elderly patients, diabetics, and those with chronic kidney disease. Involvement of the uterine vasculature is exceedingly rare, with only a handful of cases described worldwide. We report a 60-year-old postmenopausal woman with no identifiable metabolic risk factors who presented with third-degree utero-vaginal prolapse and underwent vaginal hysterectomy with anterior colporrhaphy. Histopathological examination of the surgical specimen incidentally revealed medial calcific sclerosis of the uterine vessels, in addition to chronic cervicitis and senile cystic atrophy of the endometrium. This case adds to the sparse literature on Mönckeberg's arteriosclerosis at an atypical, visceral site and highlights the importance of histopathological vigilance in recognising this entity even in patients without conventional risk factors.

Keywords: Mönckeberg's arteriosclerosis, Medial calcific sclerosis, Uterine vessels, Utero-vaginal prolapse, Vaginal hysterectomy

INTRODUCTION

Arteriosclerosis, the umbrella term for stiffening and loss of elasticity of the arterial wall, encompasses at least three histopathologically distinct processes: atherosclerosis, which involves lipid-driven inflammatory injury to the tunica intima and is the principal cause of luminal narrowing and ischaemic disease; arteriolosclerosis, characterised by hyaline or hyperplastic thickening of small arterioles, most often in the setting of chronic hypertension and diabetes; and Mönckeberg's medial calcific sclerosis (MMCS), in which calcium hydroxyapatite is deposited circumferentially within the tunica media and, at times, the internal elastic lamina of medium and small muscular arteries.[1] Unlike atherosclerosis, MMCS characteristically spares the intima, so the arterial lumen and endothelial architecture remain largely preserved; the vessel wall instead becomes rigid and non-compliant, producing the classic "pipestem" or "railroad-track" appearance on plain radiography.[2]

The pathogenesis of MMCS is still incompletely understood - a 2025 review reiterated that its aetiology and clinical significance remain debated and that consensus diagnostic criteria are still lacking - but current evidence points to an active, cell-mediated process rather than passive mineral deposition.[3] Vascular smooth muscle cells within the media undergo phenotypic transdifferentiation toward an osteoblast-like lineage, expressing bone-associated regulatory proteins such as osteopontin, osteoprotegerin, matrix Gla protein and bone morphogenetic protein-2, which drive the nucleation of hydroxyapatite crystals along the elastic lamellae.[2] This osteogenic switch is accelerated by chronic

hyperphosphataemia and disordered calcium-phosphate homeostasis, which is why MMCS is most strongly associated with advancing age, type 2 diabetes mellitus, and chronic kidney disease or uraemia.[4] It is typically an incidental finding, identified on imaging, at the time of vascular or surgical procedures, or on histopathological examination, and is most frequently reported in the peripheral arteries of the extremities - the radial, ulnar, tibial, and digital vessels - where it can occasionally be severe enough to compromise perfusion or mimic other vasculopathies, including giant-cell arteritis when it involves the temporal artery.[5]

Involvement of visceral and pelvic vasculature, by contrast, is distinctly uncommon, and MMCS affecting the uterine vessels has been documented only sporadically in the world literature-as an incidental finding accompanying utero-vaginal prolapse, at autopsy in an elderly diabetic patient, and in a case of chronic endometritis presenting with postmenopausal bleeding.[6-8] Because the entity is silent, non-obstructive, and easily overlooked or mistaken for other calcific or degenerative changes, its true prevalence in the female genital tract is likely underestimated. We report a further case of medial calcific sclerosis of the uterine vessels in a postmenopausal woman with third-degree utero-vaginal prolapse, notable for the absence of the diabetic or renal risk factors classically implicated in this condition. In presenting this case, we specifically propose that, at the uterine site, medial calcification may arise through a hormonal and mechanical pathway distinct from the metabolic pathway that predominates elsewhere in the body, namely postmenopausal oestrogen withdrawal acting in combination with chronic mechanical and ischaemic stress from long-standing, unsupported prolapse, and we discuss what further evidence would be needed to substantiate this hypothesis.

CASE REPORT

A 60-year-old woman presented to our outpatient department with a complaint of a mass descending per vaginum for the preceding four years. She was parous with three children, all delivered vaginally at home, and had attained menopause ten years earlier. The mass was initially small and reducible on lying down but had progressively enlarged and become irreducible on digital manipulation. There was no difficulty with initiation of urination, and no history of abdominal pain or distension. On clinical examination, she was found to have third-degree utero-vaginal prolapse with an associated cystocele and rectocele, without evidence of keratinisation or pigmentation of the prolapsed mass. Pre-anaesthetic evaluation, including echocardiography, revealed impaired diastolic function with a preserved ejection fraction of 60%. The patient had no known comorbidities, including no history of diabetes mellitus or renal disease. She underwent vaginal hysterectomy with anterior colporrhaphy. Intraoperatively, the uterus was atrophic with an elongated cervix, and the endometrial thickness on the resected specimen was 2 mm. The specimen was submitted for histopathological examination, which demonstrated chronic cervicitis and senile cystic atrophy of the endometrium, together with circumferential medial calcific sclerosis of the uterine vessels (Figure 1), consistent with Mönckeberg's arteriosclerosis. The patient had an uneventful postoperative recovery.

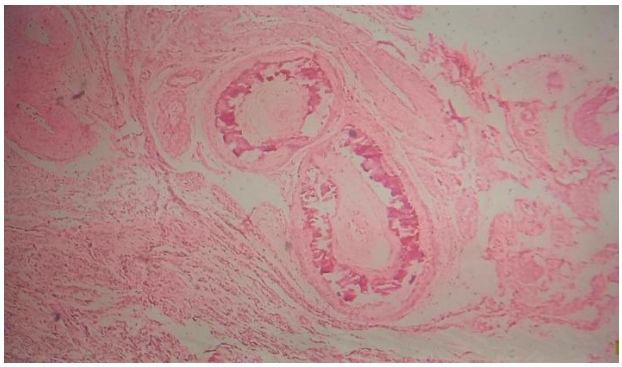

Figure 1: Histopathology showing areas of sclerosis.

DISCUSSION

Mönckeberg's medial calcific sclerosis is a degenerative, non-inflammatory process of the arterial media that is mechanistically and clinically distinct from atherosclerosis: it is generally non-obstructive, is not associated with plaque rupture or thrombosis, and its principal physiological consequence is loss of arterial compliance and elasticity rather than luminal compromise.[1,2] Its recognised risk factors - advanced age, diabetes mellitus and chronic kidney disease with attendant hyperphosphataemia - reflect a shared final pathway of vascular smooth-muscle-cell osteogenic transdifferentiation.[2,4] Our patient, however, had none of these classical predisposing conditions, which is consistent with the small number of previously reported uterine cases, several of which have likewise occurred in women without diabetes or renal impairment.[6] This suggests that, at least in the uterine vasculature, additional or alternative mechanisms may be operative.

One plausible contributor is the hypoestrogenic state of menopause. Oestrogen has a recognised protective effect on vascular smooth muscle and endothelial function, and its withdrawal after menopause is associated with accelerated vascular stiffening and remodelling throughout the circulation, including the uterine vessels, which additionally undergo marked involutional atrophy once ovarian hormonal support is lost. In our patient, this process may have been compounded by chronic mechanical and ischaemic stress on the uterine and cervical vasculature from long-standing, unsupported utero - vaginal prolapse - a possibility also raised in the original description of this association.[6] Similar reasoning has been proposed in the report of Mönckeberg's sclerosis discovered in a postmenopausal woman with chronic endometritis, where the authors speculated that medial

calcification and consequent reduced uterine perfusion might predispose the atrophic postmenopausal endometrium to chronic low-grade infection or inflammation.[8] Taken together, we propose that these observations reflect a distinct, site-specific pathway to medial calcification in the ageing, prolapsed uterus - hormonal withdrawal acting together with chronic mechanical load - that is mechanistically separate from the systemic metabolic pathway (advanced age, diabetes, chronic kidney disease and hyperphosphataemia) that predominates elsewhere in the body. This distinction matters clinically: it implies that MMCS confined to the uterine vessels should not be taken as a proxy for metabolic risk, and that a patient's absence of diabetes or renal disease should not lower the pathologist's index of suspicion when reviewing prolapse or hysterectomy specimens.

This hormonal-mechanical hypothesis remains speculative on the basis of a single case, and cannot establish causation between prolapse, oestrogen withdrawal, and medial calcification. It would be testable directly: by comparing the prevalence of uterine MMCS on histopathology between women undergoing hysterectomy for prolapse and age-matched postmenopausal women undergoing hysterectomy for other indications without prolapse, and by correlating the degree of medial calcification with immunohistochemical markers of vascular smooth-muscle osteogenic transdifferentiation, such as osteopontin or bone morphogenetic protein-2, in atrophic versus non-atrophic endometrial vasculature. Series of this kind, rather than further isolated case reports, are needed to move this association from a plausible hypothesis to an established one.

The rarity of reported uterine involvement almost certainly reflects underdiagnosis rather than true rarity of the underlying process: MMCS is asymptomatic, does not alter gross surgical findings, and is easily overlooked unless the pathologist specifically examines and comments on the vessel media, as illustrated by an autopsy case in which uterine medial calcification was an incidental finding unrelated to the patient's presenting illness.[7] Elsewhere in the body, MMCS is well documented to mimic other, more consequential vasculopathies - for example, temporal artery involvement has been reported to clinically masquerade as giant-cell arteritis, a pitfall highlighted again in a 2025 case report and literature review, and MMCS in a radial artery earmarked for coronary bypass grafting was similarly noted only after histological examination, forcing intraoperative use of an alternative conduit - underscoring the broader importance of histopathological awareness of this entity to avoid diagnostic confusion.[5,9,10] In the uterus specifically, awareness of MMCS is relevant chiefly so that it is correctly distinguished from dystrophic or metastatic calcification associated with malignancy, prior infarction, or fibroid degeneration, none of which were present in our patient. Whether medial calcification confined to the uterine vessels carries any of the systemic cardiovascular implications associated with peripheral MMCS-where arterial stiffening has been linked to increased cardiovascular morbidity - remains uncertain. Our patient's echocardiographic finding of impaired diastolic function, while non-specific and common in this age group, is at least consistent with a broader tendency toward reduced vascular and myocardial compliance. We would argue that this finding, taken alongside the systemic nature of the osteogenic vascular smooth-muscle programme thought to drive MMCS wherever it occurs, is reason enough to recommend a routine cardiovascular assessment - rather than an incidental echocardiographic observation alone - in any patient found to have MMCS at a visceral site, even in the absence of traditional metabolic risk factors; prospective studies correlating visceral MMCS with independently measured indices of arterial stiffness would be needed to confirm whether this recommendation is justified.

CONCLUSION

Mönckeberg's medial calcific sclerosis of the uterine vessels is a rare, incidental histopathological finding that can occur even in postmenopausal women without the diabetic or renal risk factors classically associated with this condition. Building on this case and the small existing body of uterine reports, we propose that its occurrence in the setting of utero-vaginal prolapse reflects a site-specific hormonal-and-mechanical pathway-postmenopausal oestrogen withdrawal compounded by chronic mechanical and ischaemic stress on an unsupported, prolapsed uterus-that is mechanistically distinct from the systemic metabolic pathway driving MMCS elsewhere in the body; this hypothesis is offered as a direction for future comparative and immunohistochemical study rather than as an established mechanism. Increased awareness among pathologists and gynaecologists is warranted so that the entity is correctly recognised and distinguished from other causes of vascular or dystrophic calcification, and further studies - ideally case series comparing prolapse and non-prolapse hysterectomy specimens - are needed to clarify its true prevalence, underlying mechanisms, and any broader implications for vascular health in the ageing female population.

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