Endometrial Osseous Metaplasia: A Narrative Review of Pathogenesis, Diagnostic Pitfalls, and Clinical Management

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Abstract

Background: Endometrial osseous metaplasia (EOM), also known as endometrial ossification or heterotopic intrauterine bone, is a rare benign condition characterized by the presence of mature or immature bone in the endometrium. While rare, it is an under-recognized, reversible cause of secondary infertility and abnormal uterine bleeding, and sonographic appearance is easily confused with a copper intrauterine device (IUD).

Objective: To present a complete review of the terminology, the first description, the biological background, epidemiology, pathogenesis, clinical presentation, diagnostic evaluation, differential diagnosis, and treatment of EOM with a particular focus on the frequent clinical mistake of transvaginal sonography misdiagnosing intracavitary bone as a copper intrauterine device, thus delaying definitive treatment for the woman's infertility.

Methods: Review of published literature (case reports, case series, histopathological and molecular analysis, and previous reviews on endometrial ossification and osseous metaplasia).

Findings: EOM is primarily a disease of women of reproductive age, with most women having a previous pregnancy loss or uterine instrumentation. There are two main and possibly separate mechanisms suggested: (1) fetal skeletal tissue persisting after an abortion and (2) the actual transformation of maternal endometrial stromal cells to osteoblasts due to chronic inflammation and repeated injury. The intracavitary bone acts as an endogenous foreign body, causing mechanical and inflammatory (prostaglandin-mediated) implantation problems just like an IUD. The diagnosis is confirmed by transvaginal ultrasonography (which usually shows hyperechoic, shadowing endometrial foci that strongly resemble a copper IUD), the reference standard for diagnosis, and the definitive treatment is hysteroscopy; histopathology is useful to exclude malignant heterologous elements. Rapid symptom clearance and often spontaneous reversion to fertility are seen with complete hysteroscopic removal.

Conclusion: An echogenic focus within the cavity of the uterus should alert the clinician to the possibility of EOM in any woman of reproductive age who has secondary infertility or abnormal uterine bleeding, especially in women who have not had an IUD inserted. High index of suspicion, hysteroscopic diagnosis, and complete surgical removal under direct vision have a very good prognosis.

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Keywords: endometrial osseous metaplasia; endometrial ossification; heterotopic intrauterine bone; secondary infertility; abnormal uterine bleeding; hysteroscopy; copper intrauterine device; retained fetal bone.

Introduction

Endometrial osseous metaplasia (EOM), or endometrial ossification or heterotopic intrauterine bone, is a rare benign condition in which bone forms inside the uterine cavity. It has been known for more than 100 years in the gynecological and pathological literature. It has been reported almost exclusively in isolated case reports and small case series, both because it is rare and because

it has been underdiagnosed in clinical practice. EOM is part of a larger group of heterologous endometrial changes characterized by the presence of an endometrial component that contains a tissue type other than normally present endometrial tissue, most often bone, but occasionally cartilage, smooth muscle, glia, or adipose tissue.

EOM is not that common, given how clinically important it is. The ossified material stuck inside the cavity acts as a natural foreign body which mechanically interferes with embryo apposition and implantation, alters the shape of the cavity, and stimulates a chronic local inflammatory response that is pro-inflammatory to a developing pregnancy [1, 2]. This produces a clinical picture that is characterized by secondary infertility, irregular menstruation, and a persistent dull pain in the pelvic area. The causal link is easily missed, especially when the overwhelming majority of affected women have a history of a pregnancy that ended with a miscarriage or termination (often months or years before presentation) and the infertility is often blamed on other more common factors. The other and just as significant challenge is diagnostic. The sonographic appearance of a copper intrauterine device (IUD) is similar to that of the strongly echogenic, shadowing intracavital foci seen on transvaginal ultrasonography [18], and it has been proposed to use this appearance to detect the presence of copper IUDs in the uterus. This resemblance can lead to a misdiagnosis of the entire gynecologic evaluation process, to a delay in definitive treatment, and to unnecessary prolonged involuntary childlessness, especially in a woman with no previous IUD placement. The lesson in the condition is the central clinical one: If the IUD is reported in a woman who has never had an IUD, then there is a pressing need to re-evaluate the diagnosis.

This review intends to summarize the current knowledge of EOM including its terminology, historical background, biological basis, epidemiology, pathogenesis, clinical presentation, diagnostic evaluation, differential diagnosis, management and prognosis with a particular emphasis on the sonographic mimicry of a copper IUD and on the simple hysteroscopic intervention that resolves this mimicry. The published case reports, case series, histopathological studies, molecular studies, and previously reported narrative reviews are the basis for the discussion. As the condition is uncommon, there is necessarily a small evidence base, and recommendations for management are based on accumulated clinical experience, not on data from randomized trials.

2. Definition and Terminology

EOM is characterized by the presence of bone in the endometrium. The terms “endometrial ossification” and “heterotopic intrauterine bone” are synonymous in the literature, and “osseous metaplasia” is often used indiscriminately for any intrauterine bone, regardless of its site of origin. But when used properly, “metaplasia” will refer to a true transformation of a differentiated tissue — in this instance, differentiation of endometrial stromal cells into an osteoblastic lineage — rather than to the mere mechanical holding of pre-formed fetal bone. This distinction is pathophysiologically significant and is often not possible solely based on conventional histology, since both retained fetal bone and metaplastic maternal bone can be identical at the microscopic level. Only in cases examined by molecular methods that can distinguish maternal from fetal or paternal DNA can a definitive statement of origin be made [5, 7].

The disease has a broad spectrum of histopathology. Isolated spicules or small plaques of mature lamellar bone represent one extreme. In contrast, more extensive trabecular areas with active osteoblasts, osteoclasts, and osteoid and, at times, foci of hematopoietic marrow represent the other extreme. In a few cases, the cartilage is present along with the bone, and in many cases, the surrounding endometrium shows a chronic inflammatory reaction with plasma-cell infiltration, which is suggestive of chronic endometritis. The bone may be present in the functionalis or in the basalis, is often visible in the cavity, and can be easily removed by hysteroscopy.

3. Historical Background

The presence of bone in the uterus was reported as far back as the nineteenth century, when intrauterine bony tissue was noted after pregnancy loss. Until the latter part of the twentieth century the phenomenon was believed to be largely retained fetal bones, thus justified by the regular clinical linkage with a previous miscarriage or abortion [24, 28]. The modern concept of EOM as a cause of secondary infertility was crystallized when retained intrauterine bone was observed to have the effect of an intra uterine contraceptive device and its removal resulted in restoration of fertility [1]. This is because retained intrauterine fetal bone has been shown to be linked to increased production of prostaglandins in the endometrium, which explains the contraceptive effect and the abnormal bleeding [2].

The introduction and spread of hysteroscopy changed the diagnosis as well as the treatment for the condition, with direct visualization of the bony fragments and the removal of these fragments under direct vision instead of blind curettage [3, 4, 25]. Recently, molecular studies of bone have overturned the belief that all intrauterine bone is fetal, and in some instances, bone is of maternal origin and is thus true metaplasia, not retention [5, 7]. The current literature is framed by this changing paradigm: retained fetal bone, foreign body effect on the cause of infertility, and a heterogeneous problem: a retentive and metaplastic form.

4. Biological and Embryological Basis

An understanding of the biological context assists in understanding why the competing explanations for EOM exist. The human fetal skeleton starts to ossify early with the appearance of primary ossification centers in the clavicle and long bones during the late embryonic and early fetal period, and by the end of the first trimester, a significant amount of the fetal skeleton already begins to ossify and is radiologically and histologically visible. As a result of this development, if a pregnancy ends or is terminated at or after this time, a significant amount of mineralized fetal bone may remain and become embedded and incorporated within the maternal endometrium [1, 24].

In contrast, true metaplastic ossification takes advantage of the well-known ability of mesenchymal cells to be plastic. Endometrial stromal cells also have multipotent fibroblast-like properties and, when stimulated, can also develop along osteoblastic pathways, similar to the phenomenon of heterotopic ossification which occurs in soft tissues other than the endometrium following trauma or chronic inflammation. The mechanisms that regulate osteogenic differentiation of the cells of the mesenchyme are very similar to those in other organs: bone morphogenetic proteins and their downstream osteogenic transcription factor are recognized as regulators of this process in other organs and so the same mechanisms are presumed to apply in the

endometrium for metaplastic bone [5, 7]. Chronic endometritis in the majority of cases is well known to exist in parallel with an inflammation-related metaplastic pathway.

5. Epidemiology

EOM is rare. It is relatively uncommon and has been reported in few hundred cases in the world literature, and is only occasionally seen at most large gynecological units. It is undoubtedly under reported and its true incidence remains unknown as cases of asymptomatic disease are not recorded and cases that are mis-reported as a retained IUD or as a case of endometrial calcification are not recorded. It is a preponderance of a disorder of women of reproductive age and the majority of reported patients occur in the third and fourth decades of life [3, 6, 13].

The most common epidemiological characteristic is its association with a preceding pregnancy event. The vast majority of reported patients have a history of previous miscarriage or induced abortion, often, but not always, after the first trimester of pregnancy, and sometimes a long time after, ranging from a few weeks to multiple years [1, 21]. There is a smaller group of women with no documented history of pregnancy loss whatsoever, and a retention of fetal tissue is a good argument that there is more than one mechanism that can give rise to intrauterine bone, and that the retention of fetal tissue is not the only explanation [5]. Other reported associations are previous uterine instrumentation, chronic endometritis and in endemic areas, genital tuberculosis.

6. Etiology and Pathogenesis

There is no single mechanism for all cases of EOM, and multiple hypotheses have been reported in the literature and are summarized in Table 1. The two most popular explanations are fetal bone retention and true metaplastic ossification of maternal tissues; both of the above are not mutually exclusive and the proportion of each may differ between patients.

6.1. Retained fetal bone

It is generally believed that the most likely cause of the condition, especially if the fetus is ossified after a miscarriage that occurred after the first trimester of pregnancy, is that the fetus' skeletal tissue is retained and persists in the womb after the fetus has died. By the end of the first trimester, fetal bone has started to ossify significantly (as mentioned above), so that fragments of fetal bone can be found in the endometrium following miscarriage or termination at or after this point [1, 24]. The embedded bone fragments remain a silent foreign body within the endometrium and basalis for many years, if at all, and are eventually found to be causing symptoms. The best evidence for this mechanism is provided by molecular studies which have demonstrated fetal or paternally derived DNA in the bony tissue, in selected cases [7].

6.2. True osseous metaplasia

In women who have not had any advanced gestation pregnancy loss, and where molecular analysis has shown the bone to be maternal, a process of true metaplasia is invoked [5]. The pluripotent endometrial stromal cells are believed to change to osteoblasts during chronic inflammation, repeated injury to the endometrium and altered local signals. These are consistently mentioned as triggers that create a microenvironment of chronic endometritis, in which osteoblastic differentiation can take place. These are always reported as triggers that create a microenvironment of chronic endometritis that allow osteoblastic differentiation. The finding of osseous metaplasia at other sites in the female genital tract (cervix) and the recurrence of such metaplasia after complete removal are additional arguments for a true metaplastic process rather than retention [11].

6.3. Dystrophic calcification

A third pathway is dystrophic calcification of necrotic decidual or chorionic tissue that can then go on to become bone. This mechanism can cause a degree of calcification and normal ossification and could form a transition stage between the two in some patients, especially those having retained necrotic tissue after an abortion.

6.4. Molecular and inflammatory mechanisms.

Irrespective of the initial cause, a chronic inflammatory microenvironment seems to play a key role in the persistence and clinical consequences of intrauterine bone. Physiological studies have demonstrated that physiological conditions that cause the retention of intrauterine fetal bone lead to the production of prostaglandins E2 and F2-alpha in the endometrium, which is a nidus for chronic endometrial inflammation [2]. The mechanisms proposed for the contraceptive and bleeding effects of intrauterine devices are also implicated in the increased local prostaglandins in causing abnormal uterine bleeding and an endometrial environment that is unfavorable to implantation. Metaplastic, the inflammatory signals that lead to osteogenic differentiation of stromal cells could also continue to maintain a bad environment in the same areas, forming a vicious cycle.

6.5. Other proposed contributors

A variety of other causes have been reported, some with inconsistent and largely circumstantial evidence, such as disordered calcium and phosphate metabolism, hypervitaminosis D, prolonged or unopposed estrogenic stimulation and chronic infective state (genital tuberculosis in endemic areas). The exact cause can not be proven in most patients and multiple factors may contribute.

6.6. Mechanism of infertility

Two mechanisms of EOM causing infertility are mechanical and inflammatory. The intracavitary bone acts as an intrauterine contraceptive device because it physically blocks embryo apposition and implantation, fills and distorts the cavity and can interfere with the transport of sperm and embryos [1]. This mechanical effect is complicated by chronic inflammatory response to the foreign body that leads to elevated local prostaglandin production and endometrium refractivity to implantation [2]. The reason the secondary infertility tends to be what brings patients to light, and the very quick recovery of fertility once the ossified tissue has been totally removed, can be explained by this dual mechanism.

Table 1: Proposed pathogenic mechanisms of endometrial osseous metaplasia.

Mechanism	Key features and supporting evidence
Retained fetal bone	Persistence of fetal skeletal fragments after a pregnancy loss beyond the first trimester; fetal skeleton ossifies substantially by ~12 weeks; supported by detection of fetal/paternal DNA in the bone.
True osseous metaplasia	Maternal endometrial stromal cells differentiate into osteoblasts under chronic inflammation/injury; supported by maternal-origin DNA, extra-uterine sites (e.g. cervix), and recurrence after removal.
Dystrophic calcification	Calcification of necrotic decidual or retained chorionic tissue progressing to ossification; possible intermediate step.
Inflammatory / prostaglandin axis	Retained bone raises endometrial prostaglandins (PGE2, PGF2-alpha), promoting bleeding and an implantation-hostile endometrium.
Contributory factors	Prior dilatation and curettage, retained products, chronic endometritis/infection (incl. tuberculosis), disordered calcium–vitamin D metabolism, oestrogenic stimulation.

7. Risk Factors

The mechanisms of EOM are outlined and parallel to the risk factors. The one association that has been seen in almost every pregnancy loss case reported is a history of pregnancy loss, especially pregnancy losses after the first trimester, regardless if the loss was spontaneous or induced [1, 21]. The instrumentation of the uterus, particularly dilatation and curettage, is a common procedure and also leaves a place for the retention of tissue and repeatedly injures the endometrium, stimulating metaplasia. Chronic endometritis and a history of pelvic inflammatory disease are common associations and retained products of conception and failure to evacuate the uterus mechanistically. In areas where genital tuberculosis is still endemic, it is accepted as another predisposing factor. Metabolic and hormonal disturbances, such as disordered calcium/ vitamin D metabolism and excessive estrogen stimulation are less consistently implicated. If these factors are present in a single patient, there should be a high index of suspicion for the presence of an echogenic intracavitary focus.

8. Clinical Presentation

There is a wide and non-specific clinical spectrum of EOM, which also adds to the delay in diagnosis. The most crucial presentation is the one of secondary infertility: many women come to present just because their conception has not succeeded after an earlier one and in a significant percentage EOM is the only factor found. According to the mechanism described above, the diagnosis should be suspected particularly in the evaluation of secondary infertility [1, 3, 13]. Abnormal uterine bleeding is the second most common symptom and can manifest as menorrhagia or irregular, long periods of bleeding between periods (intermenstrual bleeding), due to both the foreign-body effect and the higher levels of endometrial prostaglandins [2]. It is common for people to experience chronic pelvic pain and dysmenorrhea and a persistent or malodorous discharge from the vagina may be a sign of chronic endometritis. Occasionally a few women report passing any bony fragments or spicules per vaginam; this is uncommon and is seen only in a few other disorders. Lastly, a small proportion of cases are completely asymptomatic and identified on imaging or as an incidental finding in hysteroscopy for another reason. The common presentations are summarized in Table 2.

Table 2: Clinical presentations of endometrial osseous metaplasia.

Presentation	Comment
Secondary infertility	Most clinically important feature; retained bone acts as a foreign body impairing implantation.
Abnormal uterine bleeding	Menorrhagia, intermenstrual or prolonged, irregular bleeding; linked to raised prostaglandins.
Pelvic pain / dysmenorrhoea	Often longstanding and non-specific.
Vaginal discharge	May be persistent or malodorous; associated with chronic endometritis.
Passage of bony spicules	Uncommon but strongly suggestive of the diagnosis when present.
Asymptomatic / incidental	Detected during imaging or hysteroscopy for another indication.

9. Diagnostic Evaluation

9.1. Transvaginal ultrasonography

Transvaginal ultrasonography is the most commonly ordered initial test and the area where the typical diagnostic quandary occurs. EOM is most often seen as a focal or multiple strongly hyperechoic areas or linear or branching echogenic structures in the endometrium, often with posterior acoustic shadowing [18]. This appearance is very similar to a copper intrauterine device (IUD) which is the major sonographic mimic and somewhat resembles intrauterine adhesions or focal calcification. The key factor to remember is that an "IUD" in a woman who has never had an IUD inserted should raise suspicion of osseous metaplasia, rather than simply accepting the "IUD" label. The use of descriptive terminology for the endometrium and uterine cavity that is standardized helps to consistently report these results [18].

9.2. 3D Ultrasound and Sonohysterography

Saline infusion sonohysterography distends the cavity with fluid, which enhances the delineation of the location, extent and attachment of the tissue, and separates truly intracavity lesions from lesions embedded within the myometrium. In addition, three-dimensional ultrasonography with coronal reconstruction of the uterine cavity further elucidates the spatial relationship of the bone to the endometrium to help plan hysteroscopic surgery. Colour Doppler typically shows the bony focus to be avascular, which helps to distinguish it from vascularized retained products of conception.

9.3. Hysteroscopy

The diagnosis is made by the hysteroscopy and this is the definitive treatment. Hard, whitish, spiculated or plaque-like fragments can be seen directly in the endometrium, and can

be easily distinguished from the intrauterine device, intrauterine adhesions, and retained products of conception [3, 4]. It is the same procedure that allows the operator to see the bone and remove it, and then at the conclusion of the procedure to be certain that the bone has been completely removed, an important factor in outcome.

9.4. Histopathology

Definitive diagnosis is made with histopathological study of the excised tissue. It shows mature lamellar or woven bone, osteoid, osteoblast and osteoclastic lining, sometimes containing hematopoietic marrow or foci of cartilage in or near the endometrial lining (stroma). There is often a chronic inflammatory change in the surrounding endometrium. Histology also plays a crucial role in ruling out the presence of the malignant heterologous components of a carcinosarcoma (malignant mixed Müllerian tumor) which is the most important pathological differential diagnosis and has a completely different prognosis and management.

9.5. Other imaging and the problem of diagnostic delay

A Hysterosalpingogram may also detect filling defects in the cavity and computed tomography (CT) or magnetic resonance imaging (MRI) occasionally shows the presence of ossified intrauterine tissue, but these methods are not first line and only used if the case is unusual or complicated. The symptoms are non-specific and can be confused with a common contraceptive, and this can delay diagnosis (sometimes for years) until the fertility treatment is misdirected. The most important ways to reduce this delay are to be aware of the entity, have a careful contraceptive and obstetric history, and have a low threshold for diagnostic hysteroscopy.

10. Differential Diagnosis

The differential diagnosis of an echogenic, shadowing intrauterine focus is the essence of the clinical challenge of EOM, and is outlined in Table 3. The most significant and most common misinterpretation is the retained or embedded copper IUD which is often identified with a careful contraceptive history and is definitively identified by hysteroscopy. Recent pregnancy, Doppler vascularity, and histology or serum β-hCG help to diagnose retained products of conception with calcification. Further benign considerations are dystrophic endometrial calcification and intrauterine adhesions (Asherman syndrome) which is seen with fibrous bands and decreased distensibility of the cavity at hysteroscopy. The most important malignant disorder to rule out is carcinosarcoma (the presence of heterologous osseous or cartilaginous elements in a malignant tumor, usually seen in older, or postmenopausal, women); this is an aggressive malignancy which can be differentiated from benign metaplasia by histopathology. The mature teratomatous elements and other heterologous metaplasias rarely appear in the differential diagnosis.

Table 3: Differential diagnosis of an echogenic intrauterine focus

Entity	Features	Key discriminator
Copper IUD	Regular echogenic shadowing intracavitary structure.	History of insertion; hysteroscopic visualisation.
Osseous metaplasia	Irregular hard whitish bony spicules/plaques embedded in endometrium.	Hysteroscopy and histology showing bone.
Retained products	Mixed echogenicity, possible vascularity, recent pregnancy.	β-hCG; Doppler; histology.
Intrauterine adhesions	Fibrous bands across cavity; reduced distensibility.	Hysteroscopic appearance; instrumentation history.
Endometrial calcification	Punctate echogenic foci without organised bone.	Histology; absence of trabecular bone.
Carcinosarcoma	Heterologous bone/cartilage within a malignant tumour; older women.	Histopathology demonstrating malignancy.

11. Management

11.1. Hysteroscopic removal

Total removal of the ossified tissue under hysteroscopic guidance is the treatment of choice. The bony fragments can be identified and removed using hysteroscopy and the surrounding healthy endometrium spared, and the operator can confirm that the cavity is clear at the end of the hysteroscopy [3, 4, 25]. The complete removal is the most crucial factor of success for both symptom relief and restoration of fertility; there should be no remnants, as this will result in ongoing bleeding and infertility requiring additional procedure.

11.2. Surgical Technique

The tissue can be removed with grasping forceps, hysteroscopic scissors, a resectoscope or, in more recent practice, via hysteroscopic tissue morcellation or ancillary sources of energy, depending on the size, number, and depth of attachment of the fragments and the equipment available [19, 20]. Concurrently using transabdominal ultrasound has been described to enhance the safety and completeness of fragment removal, as it would enable real-time monitoring of the depth of resection, and decrease the chance of perforation; this is a particular advantage when the fragments are deep within the cavity or the cavity is distorted [9].

11.3. Blind Curettage

Historically, blind dilatation and curettage (BDC) was performed prior to the spread of hysteroscopy; it is generally not recommended. It may lead to the incomplete removal of the embedded fragments, it might injure the basal endometrium and is associated with the risks of uterine perforation and of the formation of intrauterine adhesions, which are exactly the ones that may undermine future fertility. There are no such risks with direct hysteroscopic excision and the tissue obtained can be used for definitive histological diagnosis.

11.4. Peri-operative and post-operative care

If chronic endometritis and/or active infection occur, adequate antimicrobial treatment is provided, as continued inflammation may prevent healing and fertility. Once the

cavity is removed, it is recommended to repeat hysteroscopy or ultrasonography to confirm that the cavity is empty before fertility treatment can be resumed [22]. If extensive resection has been done, consideration may be given to reducing the formation of intrauterine adhesions and a brief wait before attempting a conception gives time for the endometrium to regenerate.

11.5. Subsequent fertility management

However, in many women, spontaneous conception occurs after complete removal without additional procedures, which indicates the mainly mechanical and inflammatory nature of the cause of infertility [1, 3]. If other factors contribute to the infertility, or if conception does not occur within a reasonable time, the standard assessment of subfertility and, if necessary, assisted reproductive technology could be considered; the removal of intrauterine bone obviates a discrete and correctable barrier to implantation and enhances the chances of assisted reproductive technology.

12. Prognosis

With total removal of intrauterine bone, the prognosis is good. Whether the abnormal bleeding or pelvic pain is secondary or not, resolution is the norm and often a woman with secondary infertility will conceive spontaneously within a few months after treatment, with little or no further intervention [1, 3, 13]. It is such a quick recovery of fertility after removal of mechanical and inflammatory obstruction that it reinforces the theory that the retained bone acts very much like an intrauterine contraceptive device. Recurrence following total resection is rare but has been reported, especially in the metaplastic variant, and requires follow-up to confirm an intact cavity and be alert for reoccurrence of symptoms [11]. These results combined, highlight the clinical message: an intentionally simple hysteroscopic procedure can solve years of involuntary childlessness if a diagnosis is given and there is complete removal of tissue.

13. Discussion

The literature on EOM is entirely from case reports and small series, but, with regard to the issues of clinical relevance, it is consistent within itself. One is that the condition is strongly linked with a previous pregnancy event and that the condition should be specifically suspected in women who have secondary infertility and a pregnancy history relevant to the condition [1, 21]. Second, the effect of the bone is primarily foreign body effect—that is, mechanical and via prostaglandin-mediated inflammatory response—and this is why the clinical similarity to the intrauterine device and the rapid return of fertility after its removal. [1,2] Third, it is the definitive diagnostic test and treatment of choice with excellent results achieved if the entire lesion is removed [3, 4, 25].

The principal conceptual debate is about the source of the bone. The classical theory, attributing the presence of intrauterine bone to retention of fetal skeletal tissue is well supported, and has been substantiated by the presence of fetal or paternal DNA within the tissue [7, 24]. The fact that some cases have bone of maternal origin, however, does prove that true metaplasia does exist, and that EOM is a multiple process rather than a single one [5]. In a subpopulation of patients, the diagnosis of metaplasia is further supported by the presence of osseous metaplasia at extra-uterine locations and by the reports of recurrence [11]. Practically, the difference does not affect immediate management (total hysteroscopic removal) but it does for counselling and also for the expectation of recurrence and for molecular study of tissue in cases of academic or clinical interest.

The most clinically relevant and preventable is the diagnostic delay due to sonographic mimicry of a copper IUD. It is possible to minimize this by standardized reporting of intracavitary echogenic foci and a disciplined history of the contraceptive, but the clinican remains the key safety: an “IUD” reported in a woman who has never used one should be interpreted as a warning sign for hysteroscopy, not a conclusive finding [18]. The cost of not being diagnosed is so great — years of involuntary childlessness — that it is not worth missing, especially if the diagnosis and correction is so simple and effective.

14. Limitations and Future Research

Due to the rarity of EOM, there is a limited evidence base. It is based largely on individual case reports and small groups with the inherent limitations of publication and selection bias, and is lacking in data from randomization and large comparative studies to inform management. Reported results are good, although there is no systematic long-term follow-up so there is an incomplete definition of rates of recurrence and the relative effectiveness of different hysteroscopic techniques. Multicenter registries collecting cases in a prospective manner would help future progress, as would routine molecular characterization of the bone to better understand the prevalence of the retentive versus metaplastic forms and standardized reporting of reproductive outcomes following removal. If there was greater awareness among sonographers and gynecologists of the copper-IUD mimic, then the number of cases ascertained would increase and the time to treatment would decrease.

15. Conclusion

Secondary infertility and abnormal uterine bleeding are relatively uncommon and reversible disorders caused by endometrial osseous metaplasia. In any woman of reproductive age who presents with these complaints, a history of prior pregnancy loss and uterine instrumentation, and an echogenic intracavital focus on ultrasound should be actively considered, particularly if the focus is reported as an intrauterine device in a woman who never had one inserted. Diagnostic and therapeutic benefit is provided in one; histopathology is confirmation and exclusion of malignancy, and complete removal of the ossified tissue is associated with a good prognosis, with frequent restoration of fertility. The growing clinical awareness of this entity and its highly characteristic sonographic mimicry of a copper IUD will help prevent diagnostic delay and unnecessary delay in childbearing.

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