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CASE REPORT

Isolated Endometrial Calcification Presenting As Primary Amenorrhoea

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About the Author

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Introduction

Isolated endometrial calcification without any other calcification in pelvis or elsewhere in body as a cause for primary amenorrhoea is very rare. In May 2005, Duffield [1] and Eugenio Gerscovich from the University of California determined the clinical and histologic importance of small echogenic foci in endometrium/endocervix on pelvic sonography. Their result of ultrasound examination from 62 women was small hyperechoic foci in endometrium in 76 % and endocervix in 19 % or in a combination of endometrium/endocervix in 3 % or endometrium/myometrium in 2 %. The echogenic foci measured a few millimetres each, were mostly non-shadowing, and often appeared in small clusters. They commented that Asher-mann's syndrome, associated with haemorrhage, inflammation, and tissue destruction may have a causal role in endometrial calcification.

Zreik and Rutherford [2] illustrated psammoma bodies in association with benign conditions like oral contraceptive pills, benign papillary structures of the ovaries, intrauterine devices, ruptured ovarian cysts, endosalpingiosis, etc. Multiple psammoma bodies are also reported in lower endometrium following clomiphene citrate administration and diagnostic curettage for infertility evaluation. Premenopausal, perimenopausal, and postmenopausal endometrial calcification are reported in women where average age of menarche was 12.3–13 years. Sexually transmitted disease was present as a cause in menstruating women. Thus in most of cases secondary amenorrhoea was present.

Here, a case of primary amenorrhoea depicted isolated calcification of entire endometrium.

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Case Report

Miss P.S., 17 years, presented with primary amenorrhoea. She had average built with normal secondary sexual characters without any other constitutional symptoms or systemic problems. There was no malaise, fever, headache, or galactorrhoea. TCDC of WBC (TC = 7200; N-62, L-32, M-1, E-0) and ESR = 10 mm were normal. Hb = 11.6 g; calcium = 9.6 mg; thyroid function, serum prolactin, LH, and FSH were within normal limits. Mild mucopurulent discharge was seen at external os which on Gram staining revealed occasional Gram-negative bacilli and Gram-positive cocci in pairs and singles. Gram-positive bacilli were also present. Moderate numbers of epithelial cells (10–15/hpf) and occasional pus cell (0–1/hpf) were seen. Any pathogens were not grown. AFB stain and culture were negative.

Transabdominal ultrasound depicted size of uterus to be 61.2 × 43.2 × 20.8 mm (Fig. 1). Right ovarian volume was 7 ml (Fig. 2) and that of left ovarian volume was 13.9 ml (Fig. 3). Both ovaries contained multiple tiny follicles. Any other calcific focus was not identified in pelvis or abdomen. Colour doppler exhibited high resistance flow in both uterine and ovarian arteries (Figs. 4, 5). Endometrium had 7.5 mm thick calcification in entirety (Fig. 6). Cervical canal was moderately echogenic. Concentration hovered around endometrial causes of primary amenorrhoea.

Decision was taken for laparo-hysteroscopy. But patient party refused laparoscopy and agreed for hysteroscopy.

Hysteroscopy was done under G.A. First cervico-endometrial aspirate was taken for bacteriology. Lower 1 cm of cervical canal was accessible (Fig. 7) whereas upper part had intense fibrosis. Uterine cavity could not be entered as it had grating sensation accompanied with frank fibrosis and amorphous calcification. Gradual dilatation of uterine cavity under hysteroscopic control resulted in 3 cm of non-bleeding uterine cavity (Fig. 8) wherein one multiload 375 (Fig. 9) was

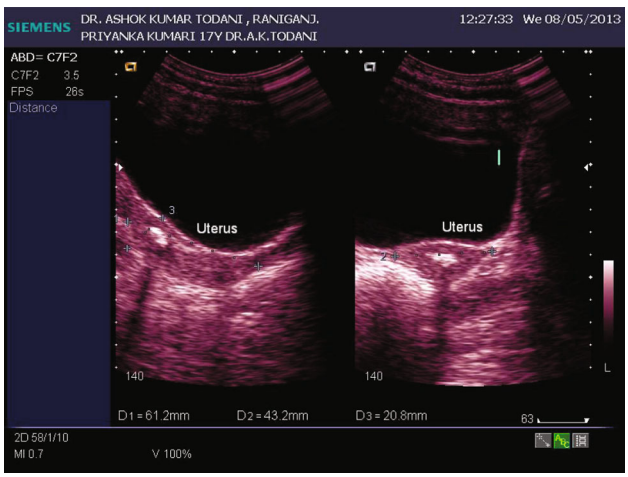

Fig. 1 Transabdominal pelvic ultrasound (uterus)

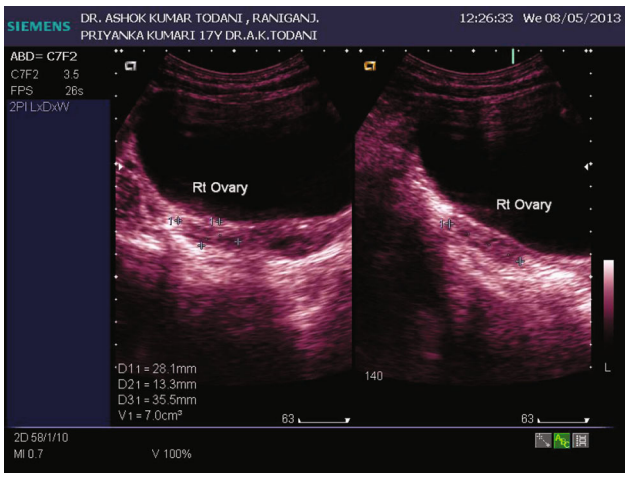

Fig. 2 Transabdominal pelvic ultrasound (right ovary)

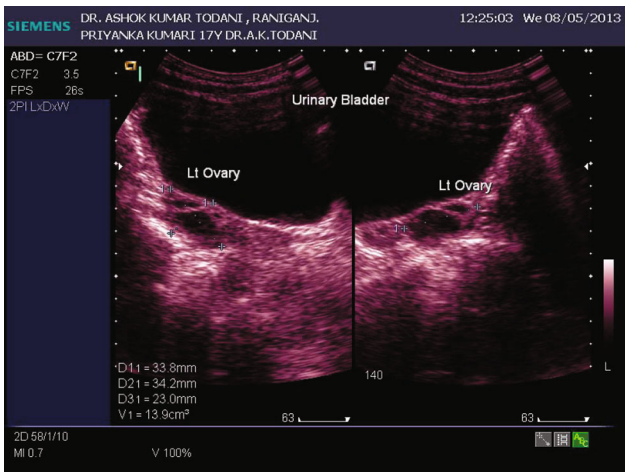

Fig. 3 Transabdominal pelvic ultrasound (left ovary)

put in to maintain its existence. Very few tiny tissue bits could be recovered, which were endometrial bits but inadequate for opinion as per histo-pathology report. Endocervical and endometrial aspirate had occasional Gram-positive spore-bearing bacilli. Any AFB was not found.

Discussion

Causes of intrauterine calcification [3] are:

1. Congenital as part of intra-abdominal echogenic foci in intrauterine life continuing later in life.
2. Acquired causes:
   (a) Traumatic → Intrauterine haematoma getting organised, then fibrosed and ultimately calcified;
   (b) Inflammatory → Different acute and chronic PID including tuberculosis;

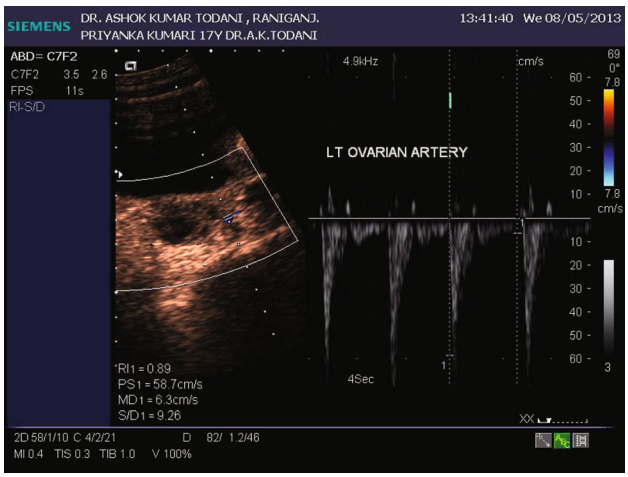

Fig. 4 Colour Doppler of left ovarian artery

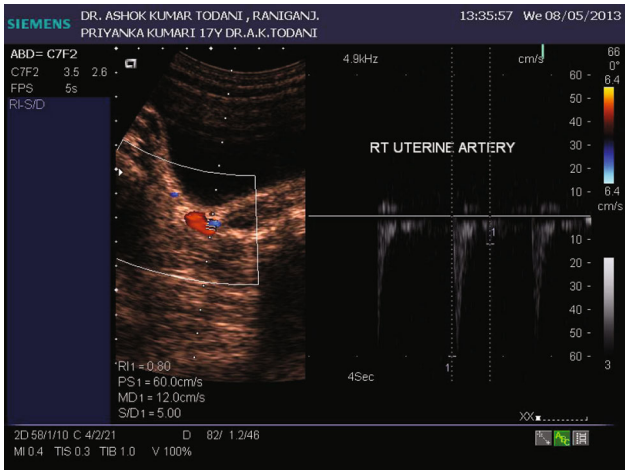

Fig. 5 Colour Doppler of right uterine artery

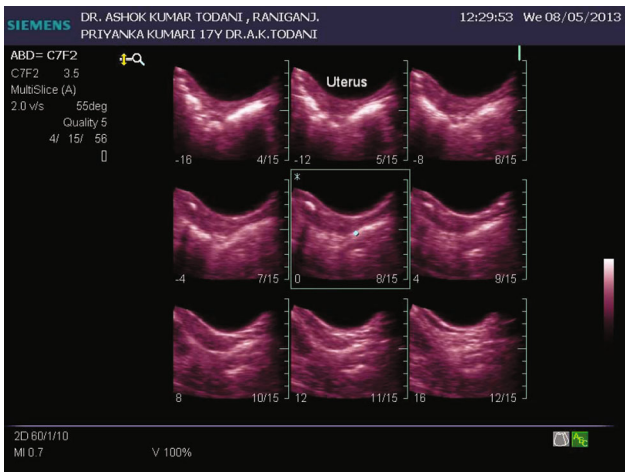

Fig. 6 Transabdominal ultrasound uterine multislice

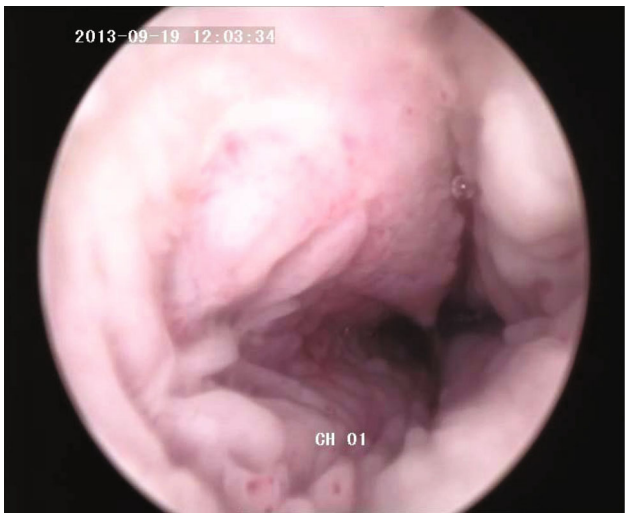

Fig. 7 Hysteroscopy of cervical canal

(c) Neoplastic → Benign conditions like fibroid/polyp calcification; malignancies;
(d) Miscellaneous → Psammoma bodies, metabolic disorders, iatrogenic like Ashermann's syndrome.

Calcification is deposition of calcium. If we consider types of calcification, they can be physiological or pathological. Pathological calcification is accumulation of calcium salts in soft tissues.

Aetiology

It may be either dystrophic or metastatic.

(1) Dystrophic calcification occurs in damaged tissues either dead or degenerative

(a) Progressive disintegration of dead cells leads to denatured proteins.
(b) Exposes groups capable of binding to phosphates.
(c) Acts as nucleation sites for precipitation of calcium.
(d) Process is accentuated by hypercalcemia.

(2) Metastatic calcification

(a) May occur in normal tissues whenever there is hypercalcemia, which may be due to

i. Hyperparathyroidism,
ii. Malignancy,
iii. Vitamin D intoxication,
iv. High bone turnover,
v. Renal failure.

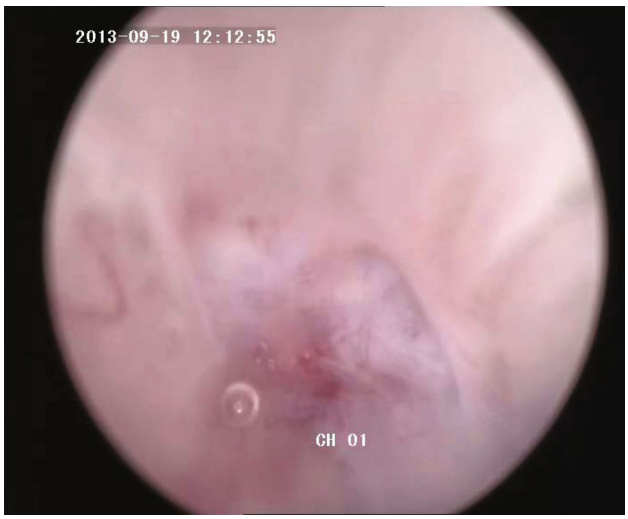

Fig. 8 Hysteroscopy restored uterine cavity

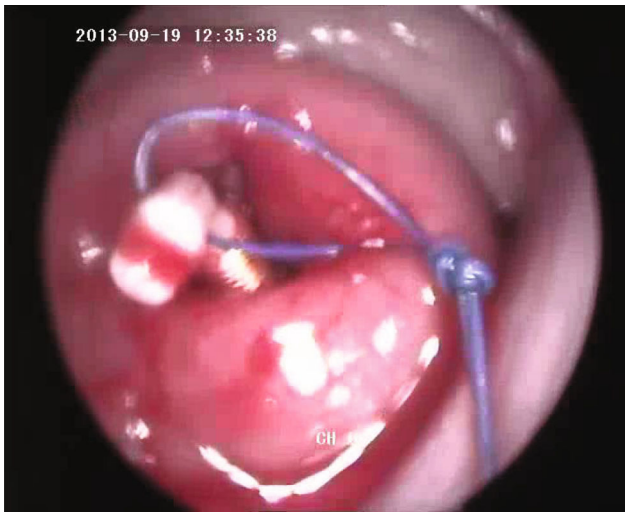

Fig. 9 CuT inserted

(b) Exact mechanism is unknown.
(c) Possibly related to localised high pH.
(d) Favours deposition of basic calcium salts.

Pathology

1. In dystrophic calcification, deposits are usually amorphous and non-crystalline. It may be in the form of
   (a) Phosphates,
   (b) Carbonates,
   (c) Oxalates,
   (d) Often mixed with iron salts.

Then, hydroxy appatite crystals may form progressing to ossification. It often causes organ dysfunction.

- Dead tissues like caseation, parasites, fat necrosis, infarcts, thrombi, and haematomas.
- Degenerative tissues in atherosclerosis, damaged heart valves, infected lymph nodes, degenerative tumours and chondrocalcinosis, etc. invite calcification.

2. Metastatic variety occurs more widely.
   (a) It predominantly involves interstitial tissues of blood vessels, kidneys, lungs, gastric mucosa.
   (b) Calcium salt resembles that seen in dystrophic calcification.
   (c) Rarely causes organ dysfunction.

Calcification may be macro or micro (less than 2 mm).

Conclusion

Patient had macrocalcification of endometrium as a cause of amenorrhoea. It appeared to be a case of dystrophic calcification, where deposits were amorphous and non-crystalline leading to organ dysfunction. Dead tissues like caseation, parasites, fat necrosis, infarcts, thrombi, and haematoma invite calcification. Degenerative tissues in atherosclerosis, damaged heart valves, infected lymph nodes, degenerated thrombus and chondrocalcinosis form nidus for calcium deposition. Calcification and fibrosis surrounding IUCD are also well known. Psammoma bodies are well circumscribed, laminated, calcified structures that may be seen in female genital tract in association with serous tube neoplasms, which can be benign, borderline, or malignant. Good example for this is "burnt out" or atrophic endosalpingiosis. Ashermann's syndrome follows D&C.

Perioperative endometrial aspirate showed Gram-positive spore-forming bacilli (i) where aerobes are *Bacillus anthracis* (cutaneous, pulmonary, and GI) and *Bacillus cereus* found in food rice, potato, meat etc. and (ii) anaerobes are *Clostridium perfringens*, *C. difficile*, *C. tetani*, and *C. botulinum*.

Mycobacterium tuberculosis and *M. leprae* are non-spore forming acid-fast bacilli. Punctate calcification could be seen in cystic deneration of endometrial glands ending in calcification.

The patient came from a farmer's family who was used to pass urine and stool in open cultivable fields where gram-positive spore-forming aerobic bacilli found ascending genital tract to destroy the endometrium. It was a slow process and never caused any form of acute local or systemic disturbances in patient so as to draw attention. Ultimately entire endometrium was destroyed and calcified. Chronic discharge and inflammation prompted

endocervical fibrosis as well. Thus, local sanitation and outdoor field toilet habits need attention and counselling.

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