

A rare cause of antigen cancer elevation I25 (CA 125): a case of uterine adenomyosis

Abstract

Until the recent past, the delays entered the first sign and the diagnosis of adenomyosis was several years. In the absence of a specific sign, its frequency is hardly appreciable because of the great heterogeneity of the studied populations (all on parts of hysterectomy). All research is converging towards reducing the time of diagnosis of adenomyosis and endometriosis. The dosage of CA 125 could be promising. The physiopathology of its synthesis and its secretion shows us that it can be elevated during endometriosis in general, sometimes in the absence of clinical signs. We believe that even in the absence of functional signs, its dosage should be systematic in the presence of uterine pathology even in the absence of endometriosis. We report here a case of isolated adenomyosis (in the absence of any uterine fibroid) associated with an elevation of Ca 125 and whose kinetics of values were influenced by hormonal treatment.

Volume 4 Issue 3 - 2017

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Received: February 13, 2017 | **Published:** February 16, 2017

Introduction

Adenomyosis is an ectopic localization of endometrial tissue (glands and stroma) and autonomous development in the thickness of the uterine muscle. The prevalence of adenomyosis ranges from 5 and 70%¹ after hysterectomy according to the surgical indication,² whatever their indications. Transvaginal ultrasound and magnetic resonance imaging exam (MRI) have currently an important place in the diagnosis before surgery. The antigen cancer 125 (CA 125) is currently, apart the ovarian's epithelium cancer, used in the diagnosis of deep endometriosis, and does not appear as a determining element in the diagnosis of adenomyosis. We report here, a case of isolated adenomyosis (in the absence of any uterine fibroid) associated with an elevation of CA 125 and whose kinetics of values were influenced by hormonal treatment.

Observation

A 35-year-old woman presented for an incidental finding of an increased level of antigen cancer 125 performed in systematic blood test check up. She reported pelvic chronic pain and abnormal menstrual bleeding for several months. The transvaginal ultrasonography showed a large antero-posterior asymmetry of the uterine walls, a striated appearance of the myometrium, and a neof ormation rounded with

fuzzy limits without mass effect and calcifications. No adnexal mass was observed. These signs were strongly suggestive of adenomyosis lesion (Figure 1). The magnetic resonance imaging (MRI) confirmed a diffuse adenomyosis without uterine fibroids (Figure 2). Neither the ultrasound nor the MRI revealed deep infiltrating endometriosis (DIE). A Positron Emission Tomography (PET) scan was performed to rule out other differential diagnosis of an increased blood level of CA125. Focal hypertense hypermetabolism was only observed in the myometrium (Figure 3). Standard pipelle endometrial sampling did not reveal atypic cells. The other tumor's markers including ACE, CA153 were normal. Conservative management was proposed as first line therapy. Therefore successive hormonal suppression was given (gonadotropin-releasing hormone agonist-GnrHa, and then Ulipristal Acetate). The Figure 4 summarizes the changes of CA 125 level and menorrhagia with treatment. However, symptoms did not release and a radical management was performed. At the laparoscopy, a globally enlarged uterus was found. No other macroscopic focus suggestive of pelvic endometriosis was highlighted. No complications occurred after hysterectomy. The histological examination of the specimen showed numerous foci and diffuse adenomyosis consisting of heterotopic glands subtended by a cytogenic chorion and hyperplasia muscle fibers (Figure 5). Also the endometrium was hypotrophic and the Fallopian tubes were normal.

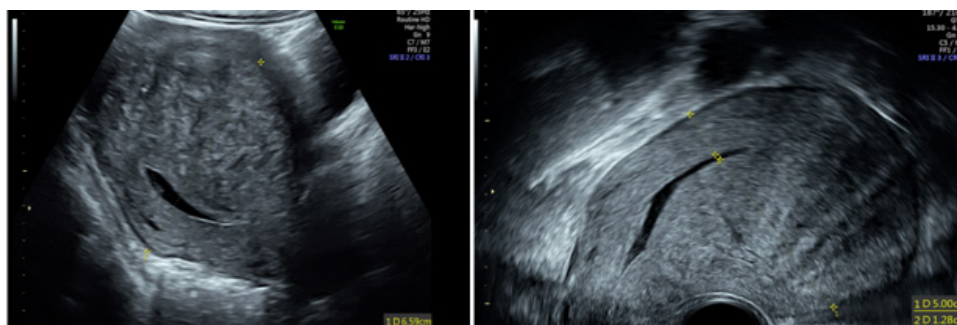


Figure 1 Pelvic ultrasound images: Important asymmetry between the uterine walls. The anterior wall had a striated appearance, with a neof ormation rounded with fuzzy limits without mass effect and calcifications.

- i. Left: Abdominal route, with a laminated aspect of the anterior wall that is blown by an adenomyoma.
- ii. Right: Vaginal route, significant asymmetry of thickness of the anterior and posterior walls of the uterus.

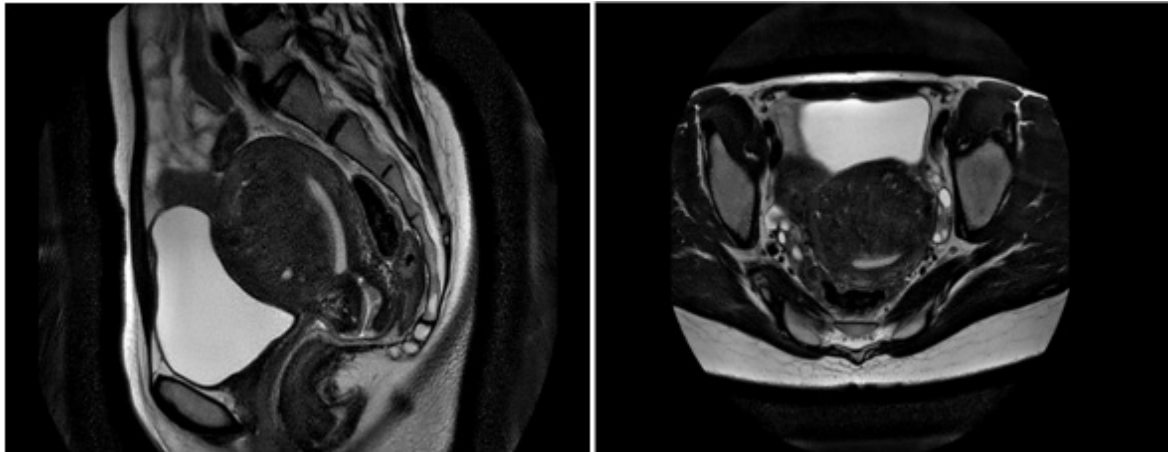


Figure 2 Pelvic MRI: A myometrial hypertrophic area of the anterior surface poorly limited, without defined wall with many hyper-intense micro-signals.
 i. Left: Sagittal cut in T2 sequence.
 ii. Right: Axial cut in T2 séquence.

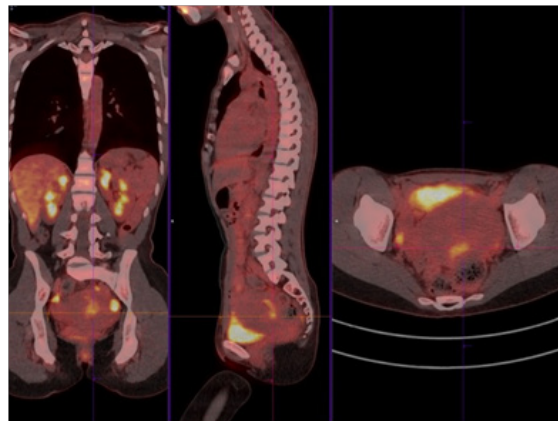


Figure 3 PET scan demonstrating endometrial and uterine hypermetabolism.

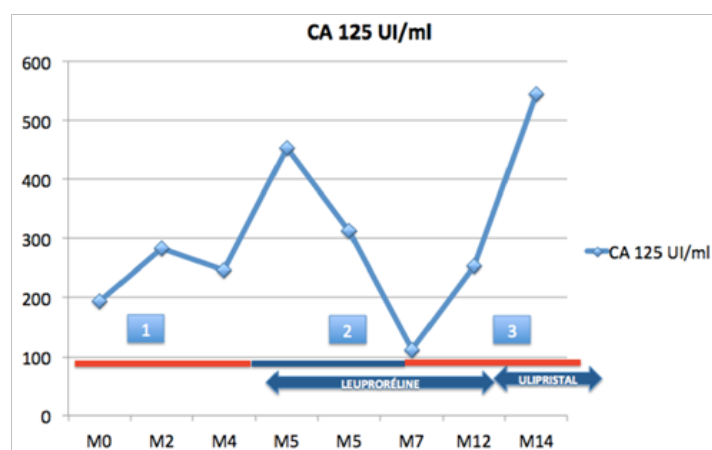


Figure 4 Change in CA 125 and menorrhagia during treatment.
 i. 1 and 3 red lines: Periods of Menorrhagia.
 ii. Blue line: Period of normal menstruation bleeding.

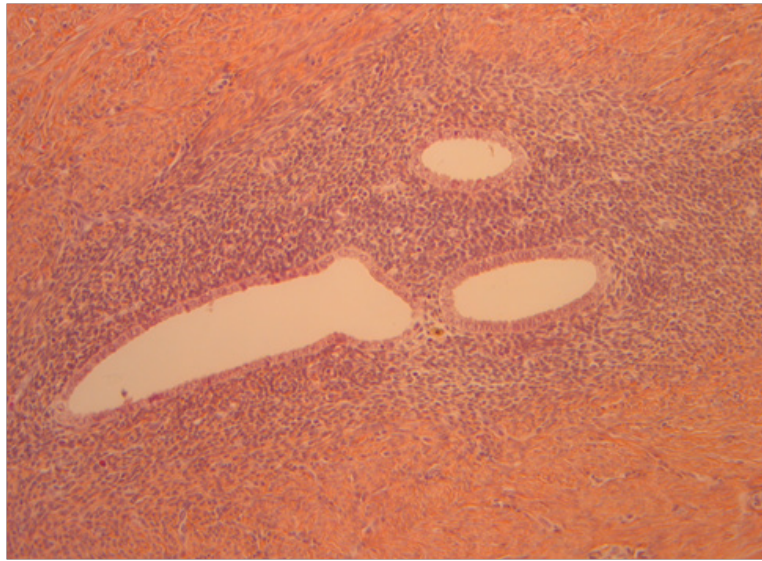


Figure 5 Histological aspect showing endometrial heterotopic glands with a highly cytogenic chorion within the myometrium. Magnificationx200. HES coloring.

Discussion

The prevalence of adenomyosis ranges from 5 and 70%¹ after hysterectomy according to the surgical indication.² While the diagnosis of adenomyosis is histopathologic, transvaginal ultrasound and MRI can be strongly suggestive. The antigen cancer 125 (CA 125) is currently, apart the ovarian's epithelium cancer, used in the diagnosis of deep endometriosis, and does not appear as a determining element in the diagnosis of adenomyosis. The finding of an incidental increased level of CA 125 challenges the diagnosis. Ovarian epithelial tumors, severe endometriosis, cardiac and chronic renal failure should be ruled out. Our case demonstrates that adenomyosis could be another differential diagnosis.

- i. The glycoprotein CA 125 can be, secreted by several cells and enhanced by the inflammatory cytokines.^{1,2} An increase in cytokines and inflammatory mediators had been observed in women with adenomyosis without endometriosis.³ The focal hyperintense metabolism at PET scan, may reflect this intense inflammatory process in the myometrium with adenomyosis.
- ii. The secretion of local oestrogen is increased in adenomyosis. Oestrogen may promote local inflammation. Hormonal suppression may decrease local secretion of steroids and therefore reduce the inflammatory process. In our case, the decrease of CA 125 level after GnRH α may be supported both by the experimental findings and its beneficial use to improve the result of in vitro fertilization (IVF) in case of endometriosis.⁴

This case raises further questions

- i. In the absence of symptoms, the benefit of routine CA 125 blood test remains unclear.
- ii. CA 125 could be associated with severe adenomyosis and unsuccessful hormonal therapy with factor of poor prognosis in the case of hormonal treatment.

- iii. And in the presence of uterine hypermetabolism to PET scan, adenomyosis should be discussed as a differential diagnosis of uterus cancer (sarcoma and endometrial adenocarcinoma).

Conclusion

Increase blood level of CA125 can be found in adenomyosis and should be considered in the differential diagnosis. The effect of hormonal therapy on the CA125 level remains unclear.

Acknowledgements

None.

Conflict of interest

The author declares no conflict of interest.

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