Conservative Management of Recurrent Bilateral Ovarian Cysts in Pregnancy: A Case Report

S H Teoh, L S J Teoh, W C Teng

ABSTRACT

This is a case report of recurrent bilateral ovarian cysts in pregnancy. From the clinical picture these are likely to be theca lutein cysts. A lookout for complications involving these cysts is important. Patients can be managed conservatively in the absence of complications. Likely mechanisms for the causation of this phenomenon are discussed.

Keywords: conservative management, recurrent bilateral ovarian cysts, pregnancy, pathogenesis

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INTRODUCTION

Ovarian tumours occur about once every 600 pregnancies[1]. The case reported here had recurrence of bilateral ovarian cysts in two separate pregnancies. The diagnosis of theca lutein cysts, non-neoplastic ovarian masses complicating pregnancy, was made clinically and by ultrasound. On both occasions, these cysts were conservatively managed and regressed spontaneously. Different groups have reported or summarised cases identified mainly at surgery in the management of complications such as rupture or torsion[2-4]. Al-Harbi et al reported a case of conservatively managed recurrent theca lutein cysts in pregnancy that included a prior incidence managed surgically due to complication[2]. Foulk et al have reported a conservatively managed case in a single pregnancy[4]. We believe ours is the first reported case of suspected recurrent theca lutein cysts in pregnancy which was managed conservatively. Groups reporting studies on this condittion have highlighted the importance of conservative management. Less attention has been paid to its pathogenesis which is still unclear.

CASE REPORT

TLY, a 24-year-old, Chinese primigravida was first seen in 1992 when she presented with a missed abortion. No adnexal masses were noted on ultrasound scanning of the pelvis at presentation and on follow-up six weeks after evacuation.

She next attended our clinic during her second pregnancy in 1993. She booked at 12 weeks of amenorrhoea. Ultrasound examination showed a single fetus of crown-rump-length (CRL) 55 mm and fetal heart pulsations were seen. A second gestational sac was noted but no foetus was seen. Multiple thin walled cysts were observed in both ovaries and there were no solid areas. The right ovary measured 91 x 53 x 74 mm and the left ovary measured 97 x 53 x 73 mm. In the right ovary, there were three cysts and their average diameters were 56 mm, 42 mm and 24 mm respectively. In the left ovary, there were multiple cysts and the largest cyst had an average diameter of 45 mm. Serum βHCG (human chorionic gonadotrophin) measured at 16 weeks of pregnancy during the time of triple test was 68,840 mIU/ml. This would have been much higher if the patient had gestational trophoblastic disease. The cysts were monitored with serial ultrasound examination. By the 20th week of pregnancy, the left ovarian cysts had completely resolved. Cysts in the right ovary were still present and the ovary measured 57 x 52 x 55 mm. By 26 weeks gestation, the right ovarian cysts were no longer seen. The patient went into spontaneous labour at 39 weeks gestation and delivered a 3.6 kg female baby. At review six weeks post-delivery, no cysts were found on ultrasound scanning of the pelvis.

The patient had a third pregnancy in 1998. She booked at seven weeks and five days gestation. Ultrasound scan showed a viable intrauterine singleton pregnancy. The foetus had a CRL of 11 mm and no adnexal masses were seen. The pregnancy progressed uneventfully till 12 weeks and four days when bilateral multiple thin walled cysts without solid areas were again detected on ultrasound examination. The right ovary measured 128 x 82 x 99 mm and the ovary 105 x 71 x 99 mm. Serum βHCG measured at the time of the triple test was 63,884 mIU/ml. At this time the right ovary measured 62 x 66 x 51 mm and the left ovary 83 x 54 x 83 mm. By 26 weeks of pregnancy, only the right ovary still showed ovarian cysts and these had regressed considerably (Fig. 1). The ovary then measured 48 x 25 x 47 mm. All cysts resolved by

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30 weeks gestation. Pregnancy was uncomplicated throughout and a 3.9 kg infant was delivered vaginally at 39 weeks. Again at postnatal review no adnexal masses were found. The Glucose Tolerance Test done in the second pregnancy was normal.

Of note, all three pregnancies were spontaneous conceptions.

DISCUSSION

Comment on case

The sudden detection of these cysts in the second pregnancy brought concern to the doctor and alarm to the patient. The patient was worried about the implications of these cysts to her health and more importantly, that of her child's. The presence of such cysts placed the obstetrician on alert for possible complications such as rupture or torsion. Although highly unlikely, there was a nagging worry of malignancy. However there were several reassuring features. Firstly, the cysts were never noted in previous examinations of the patient. Secondly, the ultrasound features of multiple anechoic regular thin-walled cysts suggested that these were more likely to be benign. Furthermore such cysts have been observed in both normal and abnormal pregnancies[2-4]. The patient was advised on symptoms of an acute abdomen and told to report them if they arose. In the first pregnancy the period of observation was particularly stressful for the patient. With serial ultrasound examination of the cysts it was possible and important too reassure the patient that the cysts were stable as well as inform her that the baby was growing well.

In her third pregnancy, although the cysts had made both ovaries larger than was so in the previous pregnancy, experience brought us all confidence. Nevertheless the threat of complications remained and there was welcome relief when the cysts began to regress.

Pathogenesis of theca lutein cysts

We believe that this woman developed theca lutein cysts of the ovary in pregnancy. About 50 cases complicating pregnancies unaffected by gestational trophoblastic disease have been reported in the literature[4]. Other reports[3,4] describe the presence of multilocular ovarian cysts and the sizes of the cysts could range from 3 - 20 mm[5]. It has been suggested that these cysts arise from an abnormal response of atretic follicles in the ovary to circulating HCG[2-4]. We would like to expand on this suggestion.

We propose that not all cells in recruited follicles undergo apoptosis following selection of the dominant follicle in the ovarian cycle. Thus these can respond to circulating HCG. This explains why increasing

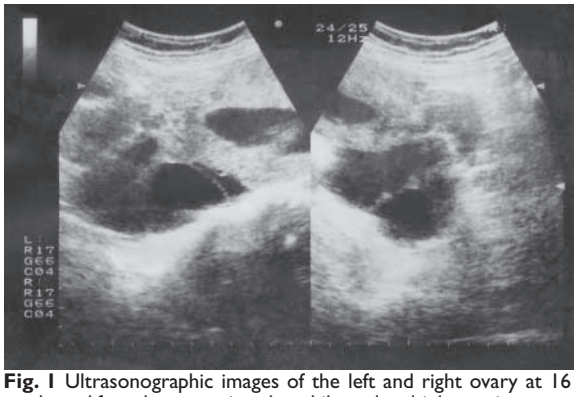

Fig. 1 Ultrasonographic images of the left and right ovary at 16 weeks and four days gestation show bilateral multiple ovarian cysts.

number of cysts are not formed during such pregnancies, being limited by the number of follicles recruited. In patients where theca lutein cysts develop only in isolated pregnancies, the development of cysts could be related to the number of follicles recruited at the start of the ovarian cycle in which conception occurred. In this case an excessively large number of follicles could have been recruited and not all have undergone atresia at the same rate thereby leaving some follicles still capable of response to circulating HCG. The cysts eventually involute with time and this can be seen in our case as well as others where the cysts have been noted to resolve at varying periods of pregnancy[2,3] with some even persisting up till after delivery[4]. Considering those patients like ours and that reported by Al-Harbi et al[3] in whom theca lutein cysts recur in different pregnancies, in such individuals, we would wonder if this is due to excessive recruitment of follicles every ovarian cycle or is there a genetic variation underlying these features. We are unsure if there is any significance in this patient that in both instances cysts on the left ovary resolved before that on the right. The role of oestrogen must be considered also because pregnancy is a hyperoestrogenic state and these follicles have not been noted to develop when an isolated rise in HCG is noted[5]. Oestrogen induces Luteinizing Hormone (LH) receptors in a normal ovarian follicle[6]. It may also have a role in inducing similar receptors in these follicles which are supposed to have undergone atresia. Since LH and HCG are almost structurally identical, it is possible that the LH receptors so induced respond to the circulating levels of HCG and the follicles then develop into cysts.

Generally there is relief to doctor and patient upon resolution of the cysts and the problem is not commonly addressed nor pursued further. Has this phenomenon any implications on the nature of such ovaries in health and disease? We hope that the thoughts raised here may be deemed worthy of future investigation.

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