An Unusual Case of Massive Hypertrophy of the Breasts

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SUMMARY

A case of giant hypertrophic breasts is described, occurring during a fifth pregnancy and while the patient was receiving treatment for pulmonary tuberculosis. Regression to normal proportions took place after the termination of pregnancy and after antituberculosis treatment, which consisted of INH-containing Thiazina tablets, had been discontinued.

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Although massive hypertrophy of the breasts is an uncommon condition, it is occasionally seen during adolescence following normal puberty, in both females and males. It has been more frequently reported in association with pregnancy and following administration of isoniazid and digitalis. The hypertrophy is thought to be due to an increased sensitivity of the mammary gland to oestrogen.

The following case, associated with pregnancy and Thiazina therapy, is presented.

CASE REPORT

In August 1971 a 22-year-old Zambian female visited the filter clinic of the University Teaching Hospital in Lusaka, complaining of swollen breasts for one week. She was not breastfeeding but had missed 2 menstrual periods and was thought to be pregnant. She was prescribed analgesics and stilboestrol for the breast engorgement, but she did not improve and was admitted to the gynaecological ward in October 1971. She presented bilateral massively enlarged, tender breasts with no circumscribed lumps palpable. There was no discharge from the nipples. She was found to be 8 weeks' pregnant. This was her fifth pregnancy, with 3 live births and one abortion in May 1971.

Her relevant past history revealed an upper respiratory tract infection in June 1970. Two months later she had been admitted to the TB ward of Kabwe General Hospital for pulmonary tuberculosis, and discharged in October 1970. She had been treated with Thiazina 150 mg thiacetazone and isoniazid 300 mg daily.

On admission to the gynaecological ward she was first treated with stilboestrol and analgesics, later combined with diuretics, but no improvement resulted. A surgeon who was consulted thought that cellulitis and carcinoma of the breast could be excluded and that hormone-induced engorgement was the most likely diagnosis.

Three weeks later the patient was taken home by her relatives to try her luck with a traditional healer, and when this too was unsuccessful, she was seen at Mpanshya Mission Hospital. On admission there she was pyrexial, pale and distressed. The breasts were acutely tender with superficial infection around the incisions of the traditional healer, causing *peau d'orange* in places. The superficial blood vessels were grossly distended, but no pathological lymph glands were felt.

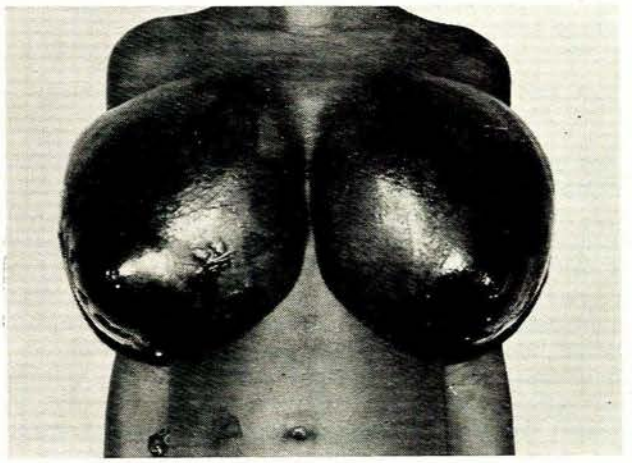

Fig. 1. The patient on 28 October 1971, immediately after termination of the pregnancy. The lesion where the biopsy specimen was taken is visible.

Fig. 2. The patient 6 months later.

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The patient was referred to the surgical ward of Lusaka's University Teaching Hospital, where the secondary infection was successfully treated and stilboestrol discontinued. Termination of the pregnancy was performed and a biopsy specimen was taken from the right breast. The histopathology report indicated that the section showed predominantly adenomatous hyperplasia with, in places, features of fibro-adenoma and evidence of secretion in glands.

After termination of the pregnancy the breasts started to lactate and methyltestosterone 10 mg *t.d.s.* was prescribed. No change in the breasts was noted and the lactation continued. By this time Thiazina had been discontinued.

By 20 December 1971, nearly 2 months after termination of the pregnancy, lactation had stopped, but the size and consistency of the breasts were unchanged and testosterone had to be discontinued because of signs of hirsutism. By April 1972 the breasts had returned to normal and the patient was again menstruating regularly. Contraceptive advice was given and an intra-uterine contraceptive device was fitted. Regular follow-up up to October 1973 showed no abnormality of the breasts, the patient being well in all respects.

DISCUSSION

Despite evidence indicating a complex, multiple glandular control of the growth of the mammary gland, it remains a basic postulate of all theories that growth results from hormonal stimuli initially set in motion by one or both of the ovarian hormones.[1] Oestrogen and progesterone, secreted cyclically, control the proliferation of ducts, lobules and alveoli. This process is greatly accentuated during adolescence. During gestation, when there is a continuous and prolonged production of both oestrogen and progesterone by the ovaries and the placenta, the greatest development takes place. Throughout pregnancy the mammary gland undergoes extensive changes in preparation for lactation.[2]

Tremendous overgrowth of the mammary gland is apparently due to extreme sensitivity to mainly the oestrogenic hormone. Besides the development of the condition of hypertrophic breasts during pregnancy and adolescence, gynaecomasty and hypertrophy of the breasts has occurred in patients treated with isoniazid. This might be due to impaired inactivation of oestrogen in the liver[3] or due to a direct effect of isoniazid on the hypothalamus and hypophysis.[4] However, gynaecomasty is not reported in the international co-operative investigation into Thiazina side-effects.[5] Digitalis may also cause gynaecomasty, by an oestrogen-like effect, the mechanism of which is still obscure.[6]

Haagensen[7] describes two types of true hypertrophy of the female breasts: the adolescent type with excessive growth of connective tissue and fat; some endocrine abnormality may be present but the menstrual cycle is usually comparatively normal. Sometimes the growth is asymmetrical, which is difficult to explain on an endocrinological basis. The massive hypertrophy of the breasts in pregnancy begins soon after the onset of pregnancy, usually a first or second pregnancy.[3,7-9] Only Luchsinger[3] reports spontaneous regression after the pregnancy; most other authors recommend surgery.[9-11]

There is little information in the literature concerning recurrence of this condition in a subsequent pregnancy; Luchsinger's patient had a recurrence in two succeeding pregnancies and Williams[9] reports hypertrophy of axillar breast tissue during a second pregnancy. Dewhurst and Burslem[11] feel that patients should be warned of the possibility of a recurrence in a following pregnancy.

The patient described in this report was in more than one way an unusual case of massive hypertrophy of the breasts. It occurred for the first time during her fifth pregnancy. She was also taking isoniazid in her Thiazina tablets, but the hypertrophy was only noted when she became pregnant while she was taking Thiazina. We postulate that the normal increased oestrogen level during pregnancy was accentuated by poor inactivation of oestrogen by the liver due to some ill-understood effect of isoniazid on the liver. After termination of the pregnancy the breasts returned to normal, taking nearly 5 months to do so. We wonder if the authors quoted waited long enough before they operated.

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