PRIMARY AMENORRHOEA RESULTING FROM PELVIC TUBERCULOSIS

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In approximately 50% of patients with pelvic tuberculosis menstrual function is normal, [1,15] while in about 25% the patient has menorrhagia.[1,14] Amenorrhoea is the least common (though possibly the best known) menstrual disturbance encountered in the disease, the reported incidence varying from 3 - 20%.[2-4] Such amenorrhoea is usually secondary. Very few cases of primary amenorrhoea caused by genital tuberculosis have been reported,[5-11] and, according to Reiss,[11] only 3 such cases had been described in Britain up to 1956.

Since pelvic tuberculosis should always be considered in the differential diagnosis of primary amenorrhoea, and because so few cases of this type have been reported, the literature is reviewed and 2 additional cases are described.

It is not surprising that *primary* amenorrhoea from pelvic tuberculosis is so uncommon. Genital tuberculosis is almost always secondary to a focus elsewhere in the body,[12,13] the lung being the primary site in 80% of cases. At the stage when the pelvic lesion presents itself, the underlying pulmonary one is active in only about 8% of cases.[10,13] Even though infection of the genital tract usually occurs within 3 years of the appearance of the primary focus, the vast majority of patients with pelvic tuberculosis develop their primary complex after the menarche.[13] This means that if amenorrhoea occurs as a manifestation of the disease it will nearly always be a *secondary* amenorrhoea.

In the rare cases in which *primary* amenorrhoea develops, the menstrual disturbance may be produced in various ways:

1. The endometrium may be destroyed by the tubercular disease, so that no gland-bearing endometrium remains.[7-10] In such cases ovarian function may continue normally, as shown by Netter and Netter-Lambert.[6]

2. Ovarian function may be depressed by the tubercular toxin or by actual involvement of the ovarian tissue in the disease; ovarian-tissue involvement is very rare, but a case has been reported by Schroder.[4]

3. It has been suggested that the hormones may be inactivated by the toxins of the tubercle bacillus, but this is hypothetical.[15]

4. Intra-uterine adhesions may occur and prevent the escape of menstrual fluid.[15-19]

5. Atresia may occur in the lower end of the uterine cavity. In fact Reiss[11] adduced evidence to show that in cases where the amenorrhoea results from intra-uterine adhesions, the obstruction is nearly always in this area. He reported 2 patients with such atresia at the lower end of the uterus — the lesion was clearly demonstrated by the attempted passage of a uterine sound and at subsequent hysterectomy. In one case there was a haematometra and haematosalpinx; in the other there was not enough functioning endometrium to menstruate. He explained such obstruction on the basis of Hughedson's[20] observation that endometrial tuberculosis is particularly liable to affect the region of the uterine isthmus.

Some of the mechanisms by which the amenorrhoea can be produced are demonstrated by the cases described here.

CASE REPORTS

Case 1

T.S., aged 20 years, was first seen on 17 November 1960, complaining of primary amenorrhoea. Breast development and growth of pubic and axillary hair had not occurred until the age of 16 years; at that time she was given oestrogen therapy by her general practitioner — the hair then appeared and the breasts developed, but there was no uterine bleeding. The patient suffered from intermittent lower abdominal pain for several years, and in 1957 a pelvic abscess was drained vaginally; details of this operation were not available.

On examination she was apyrexial. The breasts, axillary hair and pubic hair were normal, but her proportions were somewhat eunuchoidal; her height was 64 inches, span 66 inches, and lower segment 33 inches. The bone age was somewhat delayed. The nuclear sex pattern was 'female' (chromatin positive). Hormone assays gave the following results: FSH — 12·9 rat units per 24 hours, 17-ketosteroids — 5·1 mg. per 24 hours, 17-hydroxycorticosteroids — 9·8 mg. per 24 hours. Vaginal smear showed poor oestrogenization. The erythrocyte sedimentation rate was 12 mm. in the first hour. An X-ray of the chest showed a calcified lesion at the left base, and a calcified gland at the left hilum, but no active lesion. On vaginal examination the uterus was anteverted and fixed, and the left adnexa were enlarged.

On 25 November a curettage was carried out, and large amounts of obvious caseous material were found. Histological examination of the curettings showed a gross degree of caseous tuberculous endometritis. A course of streptomycin, PAS and INH was commenced, and the patient is at present being observed to see the response to treatment.

Case 2

S.F., aged 20 years, was seen on 21 October 1960, complaining of primary amenorrhoea. For the preceding 3 years she had been aware of backache and cramp-like hypogastric pain for 2 - 4 days each month, the last episode of such pain having been from 17 - 20 October. Her breasts, pubic hair and axillary hair developed at the age of 13 years.

On examination she looked well and was apyrexial. Measurements did not indicate eunuchoidal proportions. Her breasts were well developed and the pubic and axillary hair normal. The uterus was retroverted and small, and the adnexa were not palpable. X-ray of the chest showed a healed focus in the left upper zone. The vaginal smear showed good oestrogenization.

On 24 October a dilatation and curettage was carried out. The uterine sound passed for only 1½ inches. Only a single fragment of tissue could be curetted away. Histological examination of this fragment showed several tubercle follicles. Acid-fast bacilli were also demonstrated.

Treatment with streptomycin, PAS and INH was commenced on 30 October. On 1 March 1961 the patient began to menstruate. Treatment and follow-up are still continuing.

DISCUSSION

These 2 patients, presenting with primary amenorrhoea, were proved by histological examination of the endometrium (and the demonstration of acid-fast bacilli in one) to be suffering from genital tuberculosis. They demonstrate different mechanisms whereby the primary amenorrhoea can be produced.

In case 1 the cause was apparently a depression of ovarian function. There was strong evidence in favour of this. The secondary sex characteristics did not develop until the age of 16 years, when oestrogens were given ; the proportions were eunuchoidal and the vaginal smear showed poor oestrogenization. The ovarian failure may have been from toxic depression, but it is more likely that there was parenchymatous involvement of ovarian tissue by the disease, because of the extensive caseation found on curettage and on histological examination. There may well have also been gross or even complete destruction of the gland-bearing area of the endometrium. Follow-up of the case with treatment (which is being undertaken now), especially to see whether she starts menstruating or not, may throw more light on this.

In case 2, the oestrogenized vaginal smear, the normal secondary sex characteristics, and the regular monthly pain, indicated normal ovarian function, and the cause may have been endometrial. However, the endometrial destruction must have been incomplete because the patient began to menstruate after anti-tuberculous therapy. In neither of the 2 cases was any obstruction encountered when the sound was passed before curettage.

SUMMARY

The incidence of primary amenorrhoea caused by genital tuberculosis is discussed, and the reasons for the rarity of the condition are indicated. However, pelvic tuberculosis must be considered in all patients presenting with primary amenorrhoea, unless some other obvious cause is apparent.

The literature on the subject is reviewed, and 2 further cases are described.

The mechanism of production of the primary amenorrhoea is discussed, and is demonstrated by the findings in these cases.

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