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SHORT REPORTS

Congenital syphilis as an unusual cause of abnormal cardiotocogram

Prelabour fetal heart monitoring is widely used to monitor the wellbeing of babies at risk of intrauterine growth retardation. We describe an unusual case of intrauterine growth retardation presenting with diminished fetal movements. Fetal distress was shown by cardiotocography and confirmed at caesarean section.

Case report

An unmarried 32-year-old multigravida presented to the antenatal clinic on 8 April 1981. Her last menstrual period had been on 3 July 1980 and the expected date of delivery was 10 April. She was a somewhat unreliable historian but a letter of referral dated September 1980 summarised her medical history. She had had four previous full-term normal deliveries, all the babies weighing over 3700 g, and had been a heroin addict for several years. In 1979 she had developed septicaemia and subacute bacterial endocarditis, which had damaged the mitral valve. During two months as an inpatient she had been weaned off drugs of addiction. She had been in Germany since September 1980 and as she had felt well had not sought antenatal care. Two weeks earlier she had noted decreased fetal movements and had returned to England for delivery.

At examination she looked well. She had a heart murmur consistent with mitral incompetence. There was no evidence of heart failure or pre-eclamptic toxemia. The uterus was compatible with a gestation of 36 weeks. The fetal heart was heard and the rate noted to be 60 beats/min after abdominal palpation but rising to 130 beats/min. Fetal weight was estimated as less than 3000 g.

She was admitted direct from the clinic after undergoing routine antenatal blood tests and testing for Australia antigen. The first cardiotocogram on 8 April showed deep type II decelerations with uterine contractions, but the rate and baseline variability were within the normal range. A fetal movement chart was started and confirmed little activity. On 9 April the cardiotocogram showed loss of baseline variability and several decelerations.

Elective caesarean section was performed. A live female infant in very poor condition, weighing 2800 g, and with Apgar scores of one at one minute and six at 10 minutes was delivered covered in thick meconium. After resuscitation the baby was noted to have a fine petechial rash, hepatosplenomegaly, and ascites. Intrauterine infection was diagnosed and the baby transferred to the neonatal intensive care unit.

Routine investigations were begun to determine the nature of the infection, but syphilis was diagnosed when treponemes were seen in the refrigerated ascitic fluid examined two days after birth. Serological tests for syphilis were positive on both the mother's blood and cord blood. Both mother and baby were treated with penicillin with good results, though the infant was seriously ill with thrombocytopenic purpura, haemolytic anaemia, and disseminated intravascular coagulation and was jaundiced for several weeks. She was discharged on 2 July and taken into care. She did not have any skin lesions apart from the petechial rash, nor were there the expected radiological bony changes of congenital syphilis. The mother had been treated for early syphilis and gonorrhoea in February 1980, but a new infection had probably been acquired in December 1980, when she would have been about 24 weeks pregnant.

Comment

Congenital syphilis is now uncommon in Britain, only 19 cases having been reported in 1979 and eight in 1980.¹ This is probably largely due to antenatal screening, though penicillin has made a contribution. Treponemes cross the placenta even in the first trimester, but the fetus does not seem to react to this process until after the fourth month.² Intrauterine death may result or a child may be born with or soon develop signs of syphilis. Intrauterine growth retardation may precede the intrauterine death, and without fetal monitoring, prompt intervention, and skilled paediatric intensive care this baby would probably have died. We have been unable to find a report of congenital syphilis presenting with diminished movements or fetal distress detected by monitoring before labour.

As syphilis becomes less common it will be considered less often.

This case serves as a timely reminder of the importance of sterile and no touch techniques when dealing with undiagnosed infection, and that women who book late are an unusual group and all investigations on them should be processed urgently.

We thank Dr N Johnston of the Venereal Disease Reference Laboratory who made the diagnosis of syphilis by examining the ascitic fluid, and the nursing, paediatric, and venereological colleagues who helped to care for the patient and her baby.

¹ Anonymous. *Communicable diseases report* 1981;47, 27 November:3.

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Benoxaprofen: effect on cutaneous lesions in psoriasis

Benoxaprofen is an anti-inflammatory drug that is as effective an inhibitor of inflammation experimentally as indomethacin and phenylbutazone.¹ It has little effect on prostaglandin synthesis but does appear to inhibit lipoxygenase activity² and modify inflammatory cell function. Our interest in the drug was stimulated by the chance finding that the skin lesions in a patient with arthritis and psoriasis cleared shortly after the drug was started. We conducted a small pilot study in 13 patients.

Patients, methods, and results

The 13 patients selected for the study were attending our department with severe psoriasis that had failed to respond to standard measures, including, in most cases, cytotoxic drugs. All other treatment was withdrawn for four weeks before benoxaprofen was introduced in a dose of 600 mg daily. Treatment was on an open basis and the drug was continued for at least four to eight weeks. The table summarises clinical details.

Of the 13 patients treated the psoriasis was unchanged in five, greatly improved in four, and cleared in four. In two of the four whose psoriasis was cleared (cases 2 and 8) treatment was discontinued after eight weeks: one patient (case 8) has remained free from trouble, but the other (case 2) suffered a further attack after five months without treatment; this was brought rapidly under control with a further course of benoxaprofen. In the other two patients (cases 5 and 10) the drug has been continued. A fifth patient (case 4) greatly improved with treatment and her psoriasis would almost certainly have cleared had she not discontinued treatment because of dyspepsia.

Comment

It is most unlikely, given the severity of the condition, that the improvement was spontaneous: thus, eight out of 13 patients benefited

Details of 13 patients treated for psoriasis with benoxaprofen

Case No	Sex	Age	Duration of psoriasis (years)	Extent of lesions (%)	Response
1	F	71	21	30	No change
2	M	21	14	50	Cleared
3	F	35	15	50	Improved
4	F	66	30	90	Improved*
5	M	52	19	80	Cleared
6	F	35	20	40	No change
7	F	23	11	70	No change†
8	F	61	5	40	Cleared
9	M	53	32	70	Improved
10	M	29	17	80	Cleared
11	M	30	15	90	Improved
12	M	66	36	40	No change
13	M	49	31	95	No change

*Treatment stopped because of dyspepsia.
†Erythema multiforme developed.

unequivocally from taking the drug. The response was usually rapid, with lessened discomfort from the skin and a decrease in erythema.

Benoxaprofen produces a high incidence of cutaneous side effects,³ which are usually minor and include photosensitivity to long-wave ultraviolet light. Initially we thought that benoxaprofen was exerting its effect by producing photosensitivity, but this hypothesis was untenable in view of the equal improvement in areas not exposed to light. It therefore seems likely that benoxaprofen acted by influencing the migration of inflammatory cells, particularly polymorphonuclear leucocytes, probably by inhibiting the production of chemotactic substances. A characteristic and consistent feature of the histology of psoriasis is a massive infiltration of polymorphonuclear neutrophilic leucocytes, often with the formation of Monro abscesses, which in the most severe cases are clinically evident as pustules. Whether or not these polymorphonuclear neutrophilic leucocytes are normal has been the subject of dispute for some years. What is known is that they have an active lipoxygenase system that produces 5-hydroperoxyeicosate-trienoic acid and derivatives, which have been christened "leukotrienes." One of these, leukotriene B₄, is one of the most potent chemotactic substances yet identified and rivals the C5a component of complement in its activity.⁴ We suggest that benoxaprofen acted on psoriasis in the same way that it does in inflammatory conditions—by inhibiting the migration of leucocytes, in this case into the skin. It is known that polymorphonuclear neutrophilic leucocytes contain hydrolases that may stimulate epidermal cell proliferation⁵ and this supports the hypothesis that an abnormal response by polymorphonuclear neutrophilic leucocytes to a triggering factor might be the initiating event in psoriatic lesions.

There may be a basic deficiency in the cyclo-oxygenase enzyme system in psoriasis. This leads to an overproduction of leukotrienes, inappropriate accumulation of polymorphonuclear neutrophilic leucocytes, and a secondary increase in epidermal cell proliferation.

Our findings show that benoxaprofen might have had an important place in the treatment of severe psoriasis but perhaps more importantly indicate the direction for research to produce drugs with a more consistent effect and to determine the underlying defect.

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Muscle cramps during treatment with nifedipine

Nifedipine, a calcium antagonist with strong vasodilating properties, is widely used in the treatment of angina and has recently been shown to be effective in some cases of cardiac failure and systemic hypertension.¹ A low incidence of side effects has been reported.¹ We describe three cases of severe muscle spasms in which the drug had to be discontinued owing to intolerable side effects.

Case reports

Case 1—A 67-year-old man with stable angina pectoris, hypertension, and old myocardial infarction was treated with nifedipine 30 mg/day. Four months after starting the drug he complained of severe muscle cramps in both legs and later also in the hands. Nifedipine was discontinued and after 24 hours the pain disappeared. Rechallenge with nifedipine after informed consent provoked the same symptoms after 48 hours. The drug was discontinued and six months later no muscle cramps had occurred.