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Audit of process of antenatal screening for sickle cell disorders at a north London hospital

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See p 779

Antenatal screening for haemoglobin disorders (sickle cell disorders and thalassaemias) is now routine.¹ Couples who are at risk have a strong preference for prenatal diagnosis during the first trimester,² but most are identified too late for this in the presenting pregnancy.³ We studied the process of antenatal

screening and counselling for sickle cell disorders at this hospital, where 40% of births are in groups at risk. The hospital has a policy of universal antenatal and neonatal screening for haemoglobin disorders. Two community based haemoglobinopathy counsellors are notified when a pregnant carrier is identified and invite the couple within two days to attend for testing of the partner and counselling. About 80% of couples attend. Couples who are interested in prenatal diagnosis are referred to a specialist centre.

Subjects, methods, and results

Thirty one women at risk were identified among those booking for antenatal care between 1 January and 31 December 1994: 29 from laboratory records and two after the birth of an affected child. Since couples at risk have a 1 in 4 chance of having an affected child, this suggests that about six additional couples were not identified. The number of couples at risk was probably about 37. Obstetric notes were reviewed. Six women were of African Caribbean descent and 25 of African descent. Eleven were primiparous (most of whom were unaware of their risk before this pregnancy) and 20 were multiparous (most of whom had been screened and informed of their risk in a previous pregnancy). Gestation at the time of the first visit to the general practitioner, booking for antenatal care, and counselling are shown in the figure.

In the 11 primiparous women the mean gestation at the first visit to the general practitioner was 11.9 weeks, at booking 15.7 weeks, at partner's blood test result 19.2 weeks, and at counselling 19.5 weeks. Three couples did not attend counselling. All declined prenatal testing; one attended the specialist centre for counselling at 16 weeks' gestation. Seven out of 11 liveborn children had a sickle cell disorder.

In the 20 multiparous women the mean gestation at the first visit to the general practitioner was 9.2 weeks. Two of the 20 women were referred directly to the prenatal diagnosis centre for counselling and had early prenatal testing. One affected fetus was aborted. Mean gestation at booking in the remaining 19 cases

Gestation (weeks) at three points in antenatal screening for haemoglobin disorders—first visit to the general practitioner, booking for antenatal care, and counselling—in 31 women at risk of having children with sickle cell disorder. Black bars denote those who had prenatal testing

was 12.6 weeks and at counselling 15.3 weeks. Three out of four further couples referred to the specialist centre had prenatal diagnosis (at 11, 11.3, and 17.3 weeks). No fetus was diagnosed as affected, but one pregnancy miscarried. One woman declined prenatal testing because of a poor obstetric history, and she later miscarried. Two out of 17 liveborn children had a sickle cell disorder. Overall, 1 in 10 affected pregnancies was detected prenatally and terminated.

Comment

The use of prenatal diagnosis for sickle cell disorders observed here (5/37, 13.5%) is typical for London, so the data are representative though the numbers are small.³ Antenatal screening as presently practised does not meet patients' needs for early information and counselling. Instead it leads to late recognition of risk in both the index and subsequent pregnancies and a correspondingly low uptake of prenatal testing.² Only the two couples whose general practitioner referred them directly to the specialist centre had assured access to first trimester diagnosis.

Primary care teams could improve services by referring women (or couples) known to be at risk for

counselling as soon as pregnancy is reported, simultaneously screening both partners when a woman in a risk group reports a pregnancy, and opportunistically offering screening before a woman becomes pregnant.⁴ We plan to issue couples at risk with a card that emphasises the importance of early counselling in pregnancy and to build a routine audit of the screening process into counselling records.

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Drug points

Intranasal chlorhexidine resulting in anaphylactic circulatory arrest

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Acute anaphylaxis due to chlorhexidine has been reported, especially in Japan.¹⁻⁴ Most of the cases have presented with cutaneous eruptions, bronchospasm, tachycardia, and mild hypotension. We report a case of prolonged circulatory arrest that was subsequently proved to be an acute anaphylactic reaction to intranasal chlorhexidine.

A 53 year old man with acromegaly was admitted for transphenoidal resection of a pituitary adenoma. He was otherwise healthy and gave no history of drug allergy. Anaesthesia was initially uneventful. His nasal mucosa were cleaned with an aqueous solution of chlorhexidine gluconate 0.05%. Shortly after the nasal incision his blood pressure fell to 30/15 mm Hg with a heart rate of 70 beats per minute. One litre of 0.9% saline was infused rapidly, with ephedrine 20 mg and then adrenaline 0.5 mg being given intravenously. This failed to restore his blood pressure, and his systolic pressure fell to below 20 mm Hg. The operation was abandoned and cardiac massage started. Over the next 20 minutes he required a further 3 mg of adrenaline, as well as three direct current shocks for persistent ventricular fibrillation. A total of 1.5 l of 0.9% saline and 1 l of colloid solution were infused during this initial resuscitation period. He was transferred to intensive care being given an adrenaline infusion, the tube being removed 20 hours later.

At no stage was there any evidence of bronchospasm, erythema, or oedema. He suffered no neurological or cardiac damage. He remembered that a red blotchy skin rash had appeared on his face after chlorhexidine 1% dental gel had been applied to his gums during a visit to a

dental hygienist in the previous year. Allergy testing was therefore arranged. Skin prick testing to chlorhexidine gave a strongly positive result at 500 µg/ml, and a leucocyte histamine release test was strongly positive for chlorhexidine. All the other agents used during anaesthesia gave negative results.

The Committee on Safety of Medicines received 182 reported reactions to products containing chlorhexidine from 1965 to 1996 (personal communication). Most of these were cutaneous or mucosal eruptions. The committee has received only one previous report of acute circulatory failure. In Japan between 1967 and 1989 there were 15 reported cases of anaphylactic shock related to chlorhexidine, of which 13 cases followed application of the drug to mucous membranes. In 1984 the Japanese ministry of welfare recommended that the use of chlorhexidine on mucous membranes be prohibited.³ The medical data sheet for the solution of chlorhexidine used in this case (Sterets Unisept) states that contact with the brain, eyes, meninges, and the middle ear should be avoided, but there is no specific advice on avoiding contact with mucous membranes (Seton Prebbles, January 1996). As our case illustrates, allergic reactions to chlorhexidine may be life threatening. It also shows the importance of taking a detailed drug history that pays particular attention to previous reactions.

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