of the alimentary tract was intermittent or more localised than we explored, similar to the localised tender segments in the oesophagus described by Edwards.[29] Alternatively, pain may arise in other areas such as the pancreas or biliary tract or in some cases from a non-alimentary source.

Referral of pain to extra-abdominal sites has now been shown from the small as well as the large bowel. A wider appreciation of the protean presentation of functional abdominal pain and the existence of potential tender areas throughout the whole gut should expedite the recognition of non-organic abdominal pain and prevent unnecessary investigations and even exploratory laparotomy.

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SHORT REPORTS

"Scalded mouth" caused by angiotensin-converting-enzyme inhibitors

Captopril, an angiotensin-converting-enzyme inhibitor, is used to treat resistant hypertension.[1] Side effects include rash, proteinuria, leukopenia, ageusia, angio-oedema, and aphthous and tongue ulcers; its sulphydryl moiety may be an aetiological factor.[1] Enalapril, a new converting-enzyme inhibitor, does not contain a sulphydryl group.[2] We describe three patients who reported a scalded sensation of the oral mucosa during treatment with captopril or enalapril.

Case reports

Case 1—A 53-year-old hypertensive woman receiving hydrochlorothiazide 50 mg daily was prescribed captopril 12·5 mg thrice daily in addition; she took no other medication. After two weeks the dosage of captopril was increased to 25 mg thrice daily. Six days later she complained of a burning sensation on the upper surface of her tongue, comparable to having been "scalded by a hot liquid," though she had no recollection of such an occurrence. Examination of her tongue and mouth was unremarkable. She reported no alteration in taste. With her consent the regimen was continued, the symptom persisted for five days and then dissipated despite two additional weeks of captopril treatment, without subsequent recurrence.

Case 2—During a research study a 54-year-old man with essential hypertension received enalapril 20 mg twice daily. Seven days later he said that his tongue and mouth felt as though they had been "scalded by coffee or pizza," though he could not recall such an occurrence. Examination of the mouth showed only periodontal disease and questionable hyperaemia; neurological evaluation including taste perception was normal. He took no other medications. He continued treatment with enalapril for four additional days without resolution. The symptom abated, however, when enalapril was stopped, and he was treated with hydrochlorothiazide 50 mg/day. Two weeks later enalapril was restarted in a dose of 20 mg daily, the diuretic being continued. On the twelfth day of treatment he again complained of the "scalded-mouth" sensation, which was similar to but milder than the first episode. This continued for the next two days but abated when enalapril was stopped at the end of the trial. Similar symptomatology did not recur.

Case 3—A 64-year-old hypertensive woman taking propranolol 160 mg and hydrochlorothiazide 50 mg daily was prescribed captopril 25 mg thrice daily in addition. Other medications included nitroglycerine and isosorbide dinitrate. After two weeks the captopril dosage was increased to 50 mg thrice daily. Nine weeks later she complained of a burning sensation in her throat, palate, and tongue. Examination of the mouth was unremarkable. After two additional weeks of treatment with captopril the symptoms persisted and mild injection at the margins of the soft palate was noted; there was no alteration in taste. Because of continued discomfort captopril was stopped with a distinct improvement during the next week; slight discomfort, however, localised to the tip of her tongue, persisted.

Comment

Although disorders of taste as well as aphthous and tongue ulcers have been reported during captopril treatment,[3-5] the symptom described by our patients has not, to our knowledge, been previously associated with use of either captopril or enalapril. The mechanism of this side effect is unclear. Neither fever nor eosinophilia accompanied the patients' complaints. No concomitant laboratory abnormalities were noted, and all the patients had normal renal function. Since the side effect was noted with both captopril and enalapril the aetiology of this scalded-mouth reaction appears to be independent of the sulphydryl moiety of captopril. The hyperaesthesia of the oral mucosa abated during continued treatment with captopril in case 1, recurred

on rechallenge with enalapril in case 2, and was troublesome enough to require captopril treatment to be stopped in case 3. Doctors treating patients with converting-enzyme inhibitors should be aware of the potential occurrence of this side effect.

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Prenatal serological diagnosis of intrauterine cytomegalovirus infection

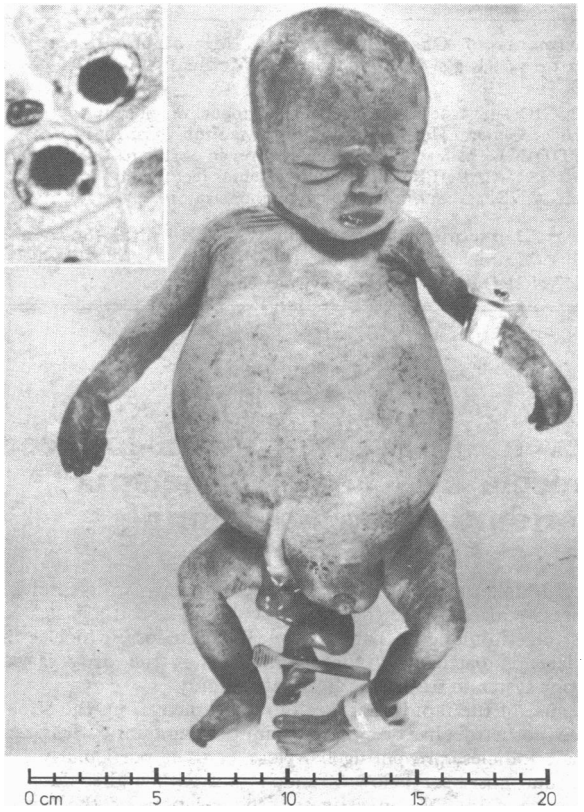
Cytomegalovirus commonly causes intrauterine infection. Finding a primary or reactivated cytomegalovirus infection in the mother by isolating the virus from the throat, urine, cervix, or blood, or by maternal serology, does not necessarily indicate that the fetus will be infected. Fetal infection appears to occur in no more than 25-50% of even primary maternal infections. The presence of intrauterine infection has been established by culture of cytomegalovirus from amniotic fluid,[1] but such isolation may be time consuming. We report a case that shows that the availability of a sensitive radioimmunoassay for the detection of cytomegalovirus-specific IgM[2] and the ability to obtain fetal cord serum[3] mean that intrauterine serological diagnosis is possible.

Case report

A 28-year-old rhesus-positive woman without atypical antibodies was referred at 25 weeks' gestation after ultrasonography showed a hydropic fetus. She had felt well throughout the pregnancy, denied taking any medication, and reported no relevant personal or family history. At 16 weeks' gestation an ultrasound scan had confirmed her dates and shown no major structural abnormalities. Examination was unremarkable, with fundal height being consistent with dates. A further ultrasound scan confirmed a single fetus with gross ascites, but no other abnormalities were observed. Fetoscopy was carried out and 8 ml pure fetal blood and 260 ml ascitic fluid collected. Fetal serum showed appreciable hypoalbuminaemia of 6 g/l (normal 21 $\pm$ SD 2 g/l (unpublished)). A fetal blood film showed severe erythroblastosis.

A follow-up ultrasound scan showed re-collection of the ascites. Intravascular fetal albumin infusion was planned but precluded by spontaneous premature rupture of the membranes at 27 weeks' gestation. A conservative policy was adopted but labour ensued and a 1480 g severely depressed male infant delivered. Resuscitative attempts were stopped after 20 minutes. Initial serology for cytomegalovirus on maternal sera showed an antibody titre of 32 when a complement fixation test was performed on sera collected at 21 weeks' and 25 weeks' gestation. No cytomegalovirus-specific IgM was detected in the fetal serum using indirect immunofluorescence (Electronucleonics Laboratories Inc, Maryland, USA).

Necropsy showed non-icteric hydrops fetalis (figure). Macroscopically the only abnormal findings were a large liver with a finely nodular and mottled surface and massive ascites causing gross elevation of the diaphragm with consequent pulmonary hypoplasia. Microscopy showed cytomegaly and intranuclear "owl's eye" inclusions in many organs, particularly the kidney (figure). The appearance was typical of cytomegalic inclusion disease. The liver showed extreme active erythropoiesis. Cytomegalovirus was isolated from urine obtained at necropsy.



Male fetus of 1480 g showing gross hydrops fetalis. Inset: kidney showing cytomegalic cells with inclusion bodies. Haematoxylin and eosin. $\times$2500 (original magnification).

These findings prompted a re-examination of stored maternal and fetal cord sera. The total IgM content of the fetal serum was 0·11 g/l. This is within the normal range for a fetus of this gestation as stated by Alford *et al.*[4] Radioimmunoassay for cytomegalovirus IgM showed that the fetal serum had a titre of 4000. This is within the range seen in neonatal or cord serum from confirmed cases of symptomatic congenital cytomegalovirus.[5] Examination of the maternal serum for cytomegalovirus-specific IgM by radioimmunoassay showed a titre of 800 in the serum taken at 21 weeks' gestation, but such IgM was absent at 25 weeks. These results were compatible with a primary infection occurring early in the pregnancy.

Comment

Cytomegalovirus is a recognised cause of hydrops fetalis, and intrauterine cytomegalovirus infection was confirmed in this case by histology and isolation of the virus. Initial serology for cytomegalovirus specific IgM on fetal cord serum obtained at 25 weeks' gestation was negative using an indirect fluorescence assay. When a sensitive radioimmunoassay was used, cytomegalovirus-specific IgM was detected with ease, showing the increased sensitivity of this procedure for diagnosing congenital cytomegalovirus.[5] We believe this to be the first case in which virus-specific IgM has been shown in fetal serum obtained in utero.

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Requests for reprints should be addressed to CHR.

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