presentation of campylobacter infection in immunodeficient patients may result in inappropriate treatment.

- ¹ Asherson GL, Webster ADG. *Diagnosis and treatment of immunodeficiency diseases*. Oxford: Blackwell Scientific Publications, 1980.
- ² Pönkä A, Tilvis R, Kosunen U. Prolonged campylobacter gastroenteritis in a patient with hypogammaglobulinaemia. *Acta Med Scand* 1983;213:159-60.
- ³ Ahnen DJ, Brown WR. Campylobacter enteritis in immune-deficient patients. *Ann Intern Med* 1982;96:187-8.
- ⁴ Goldman H, Antonioli DA. Mucosal biopsy of the rectum, colon and distal ileum. *Hum Pathol* 1982;13:981-1012.

(Accepted 2 May 1984)

Departments of Haematology and Gastroenterology, Whittington Hospital, London N19 5NF

E S GREEN, MA, MRCP, senior house officer
N E PARKER, MRCP, MRCPATH, consultant haematologist
A R GELLERT, MB, MRCP, registrar
E R BECK, FRCP, consultant physician

Correspondence to: Dr E S Green, Department of Haematology, Royal Free Hospital, London NW3 2QG.

Skull radiography in epilepsy, dementia, and non-specific neurological symptoms

Despite the development and widespread use of computed tomography skull radiographs continue to be ordered routinely, often as a prelude to computed tomography. Recent reports of studies to evaluate skull radiographs in trauma¹ and psychiatric illness² contained only limited references to the unique diagnostic information provided by skull radiographs compared with computed tomograms. Our study was designed to determine whether skull radiography could provide any important diagnostic information not available from computed tomograms in patients presenting with epilepsy, dementia, or non-specific neurological symptoms.

Patients, methods, and results

During 12 months 367 patients were referred for computed tomography. They had a presumptive diagnosis of epilepsy or dementia or were suffering from headache, vertigo, or blackouts without any focal neurological signs or papilloedema. Those patients who had not had a series of skull radiographs within the preceding week underwent a full four film skull series on the same day as the computed tomography. Computed tomography was performed with an EMI 1010 head scanner. The abnormalities seen in the scans were compared with those detected in the skull radiographs.

Skull radiographs showed abnormalities not seen on computed tomography in 14 of the 367 patients. These consisted of five skull fractures, five cases of sinus disease, and one case each of hyperostosis induced by tumour, signs of raised intracranial pressure, enlarged middle meningeal markings, and craniostenosis. In every case the abnormal skull radiograph finding was seen in the lateral skull radiograph alone.

Comment

The diagnostic yield achieved by computed tomography in patients presenting with dementia, epilepsy, and non-specific neurological symptoms without signs has been reported previously.³ In only 14 patients did the skull radiograph show diagnostic features not shown by computed tomography. Although the enlarged middle meningeal vascular markings and hyperostosis induced by tumour helped confirm the diagnosis of meningioma in two cases, the skull radiograph findings did not actively cause the management of any of the patients to be changed.

Our finding that all the abnormalities were evident in the lateral skull radiograph accords with the findings of Bull and Zilkha.⁴ They recommended that in the absence of physical signs a lateral view of the skull plus an anteroposterior projection to show the position of the pineal gland (if it is shown to be calcified in the lateral projection) is all that is initially required.

Computed tomography performed with modern third and fourth generation scanners is preceded by a computerised radiograph generated by the scanner. The quality of this computerised radiograph is such that in a recent study of computed tomography in trauma it detected 20 of 21 fractures of the skull.⁵

The results of this study indicate that once the decision has been made to investigate patients with epilepsy, dementia, and non-specific symptoms without neurological signs by computed tomography a skull series should not initially be performed. If it is decided that computed tomography is not initially indicated, or facilities for computed tomography are not easily available, a lateral skull radiograph, plus an anteroposterior radiograph if the pineal gland is calcified, is all that is required.

- ¹ Royal College of Radiologists. A study of the utilisation of skull radiography in 9 accident-and-emergency units in the UK. *Lancet* 1980;ii:1234-7.
- ² Rastogi SC, Barraclough BM. Skull radiology in patients with psychiatric illness. *Br Med J* 1983;287:1259.
- ³ Tress BM, Hare WSC. CT in dementia, epilepsy and non-specific neurological symptoms without signs: is there a place for radionuclide scanning? *Med J Aust* 1979;1:453-4.
- ⁴ Bull JWD, Zilkha KJ. Rationalising requests for x-ray films in neurology. *Br Med J* 1968;iv:569-90.
- ⁵ Cromwell LD, Mack LA, Loop JW. CT Scoutview for skull fractures: substitute for skull films? *AJNR* 1982;3:421-3.

(Accepted 17 May 1984)

University of Melbourne Department of Radiology, Royal Melbourne Hospital, Victoria 3050, Australia

BRIAN M TRESS, FRCP, FRACR, first assistant

Spontaneous separation of fused labia minora in prepubertal girls

Labial adhesions or fused labia minora are a common problem in prepubertal girls. They are harmless but must be differentiated from anatomical abnormalities. Commonly they are asymptomatic, though they may be associated with apparent urinary symptoms. Patients are referred to surgical clinics usually because their parents fear an abnormal development of the external genitalia. Occasionally the adhesions are an incidental finding during the course of routine examination. Topical oestrogen cream has been advocated as effective treatment.^{1 2 3} Alternatively, separation under general anaesthetic, except in very young infants, has been recommended.^{4 5} Treatment has been urged for these children in case more dense adherence develops. This study was undertaken to determine whether any intervention is necessary.

Patients, methods, and results

Over three years 10 girls aged from 10 to 22 months were seen in the out-patient department by one surgical firm and subsequently followed up. Two other girls were lost to follow up. The diagnosis of labial adhesions was the primary reason for attendance in nine; in the tenth diagnosis was an incidental finding at examination. In each case the parents and, when appropriate, the patient were reassured and no further action was taken. The patients were then re-examined at 6, 12, and, if necessary, 18 months.

After six months five of the 10 showed complete and spontaneous separation, four partial separation, and one no change. By 12 months nine cases had resolved, and by 18 months all 10 had resolved completely. Adhesion did not subsequently recur in any of the patients.

Comment

Treatment for fused labia minora with local oestrogen cream has an incidence of success of about 90%^{1 2} and was recently stated to be the treatment of choice.³ Side effects, however, have been reported. Aribarg found that reversible vulval pigmentation developed in all patients, one also developing vulval erythema, and adhesion recurred in one girl.² Capraro and Greenberg noticed a small incidence of breast enlargement and tenderness.¹ We have seen a child in whom adhesions recurred after the application of oestrogen cream had been stopped. Moreover, this treatment requires good compliance by patient and parent and may be resented by the child.

Surgical separation is long established⁴ and still has its advocates.⁵ Unless, however, patients undergo surgery when they are only a few months old, they have to be anaesthetised and may suffer undue distress. The stress of admitting a child to hospital and the potential risk of a general anaesthetic led us to undertake this study. Furthermore, the incidence of recurrent adhesions has been reported to be as high as 20%.⁴

Conservative management in our series led to an incidence of spontaneous separation of 100%, albeit over 18 months, though half

had resolved by six months. Provided adequate explanation of the condition is given to the parents, this approach is readily accepted and avoids the potential complications of the other methods of treatment currently recommended.

We thank the Chris Fund for financing secretarial help.

- ¹ Capraro VJ, Greenberg H. Adhesions of labia minora, a study of 50 patients. *Obstet Gynecol* 1972;**39**:65-9.
² Aribarg A. Topical oestrogen therapy for labial adhesions in children. *Br J Obstet Gynaecol* 1975;**82**:424-5.
³ Lewis GT. Fused labia minora. *Br Med J* 1983;**287**:886.
⁴ Nowlin P, Adams JR, Nalle BC. Vulvar fusion. *J Urol* 1949;**62**:75-9.
⁵ Barnes SE. Labial lesions in small girls. *Br Med J* 1983;**287**:1725.

(Accepted 1 June 1984)

Sub-Department of Paediatric Surgery, Children's Hospital, Sheffield S10 2TH

S D JENKINSON, MB, CHB, senior house officer
 A E MACKINNON, MB, FRCS, consultant surgeon

Correspondence to: Mr A E MacKinnon.

Jarisch-Herxheimer reaction in falciparum malaria?

During the treatment of falciparum malaria with Fansidar (pyrimethamine and sulphadoxine) a secondary rise in temperature is sometimes seen. We have recently studied such a case in detail.

Case report

A 20 year old man was admitted to hospital two weeks after returning to Denmark from Tanzania. Blood slides on admission showed infection with *Plasmodium falciparum*. He was given three tablets of Fansidar and his temperature declined from 40.3°C to 37.0°C in 16 hours (see figure). Ten hours later his temperature began to rise again. This was associated with malaise, headache, backpain, nausea, vomiting, and tachycardia. Suspecting failure of treatment, as no other focus of infection was found, we gave another three tablets of Fansidar. Fresh blood films showed that the maximal parasitaemia of 428 infected erythrocytes per 50 leucocytes had disappeared several hours before the new temperature rise, leaving the patient free of parasites for at least eight hours before the fever rose to its peak. During the second period of fever the total leucocyte count was $4.4 \times 10^9/l$. Differential count showed a left shift in the granulocytes, lymphopenia, and absolute eosinopenia.

On questioning the patient it emerged that in Tanzania he had suspected that he had malaria and treated himself with Fansidar. His symptoms had then disappeared in 36 hours and he had had no secondary reaction. He had never used Fansidar as a prophylactic agent but always chloroquine 500 mg a week. He was not known to be allergic to any medicine, including sulphonamides.

Comment

After the start of treatment this patient initially became well and his fever declined. The parasitaemia was cleared, from a maximum of about 1% infected erythrocytes in the 18th hour to almost none in the 38th hour. Nevertheless, simultaneously he again developed acute symptoms, with fever and a rise in the heart rate. Such a picture is similar to the Jarisch-Herxheimer reaction, which is best known after the treatment of syphilis but which has also been reported after the treatment of other spirochaetal infections, some bacterial infections, and trypanosomiasis.¹ The typical features of this are fever, chills, headache, malaise, myalgia, arthralgia, nausea, vomiting, and exacerbation of skin lesions²; a rise in the heart rate; and changes in blood pressure. During the reaction the typical haematological finding is leucocytosis with lymphopenia, eosinopenia, and a shift to the left in the granulocytes.³ In malaria, in contrast, neutropenia is the commonest finding.

The reaction in our patient after treating him for malaria resembled the classic Jarisch-Herxheimer reaction, firstly in its clinical features, secondly, in the changes in the temperature and pulse rate, and lastly in the changes in the leucocyte count. In patients with neurosyphilis treated with penicillin the reaction lasts for about 32 hours, in primary syphilis for 12 hours when penicillin is given intravenously² and for

Course of temperature and parasitaemia during treatment of falciparum malaria treated with sulfadoxin/pyrimetamin (Fansidar). Arrows show when patient received Fansidar (3 tablets each time).

24 hours when mercury is given.⁴ This compares closely with the 48 hours seen in this patient. As in syphilis it is not known why some people develop this reaction and others do not; immunological factors may have a role, as might endotoxins as well.⁵

- ¹ Bryceson ADM. Clinical pathology of the Jarisch-Herxheimer reaction. *J Infect Dis* 1976;**133**:696-704.
² Aronson IK, Soltani K. The enigma of the pathogenesis of the Jarisch-Herxheimer reaction. *Br J Vener Dis* 1976;**52**:313-5.
³ Skog E, Gudjonsson H. On the allergic origin of the Jarisch-Herxheimer reaction. *Acta Dermato-venereol (Stockh)* 1966;**46**:136-43.
⁴ Herxheimer K, Krause P. Ueber eine bei Syphilitischen vorkommende Quecksilberreaction. *Dtsch Med Wochenschr* 1902;**28**:895-7.
⁵ Grond K, Fritz J, Sandhofer M. Zur immunologischen Dynamik der Jarisch-Herxheimer-Reaktion. *Z Hautkr* 1979;**54**:191-6.

(Accepted 24 April 1984)

Department of Internal Medicine, Svendborg Hospital, DK-5700, Svendborg, Denmark

POUL-ERIK KOFOED, MD, physician

Correspondence to: Kløvervaenget 26D-21, DK-5000 Odense C, Denmark.