

intrauterine pregnancies but has little or no effect on the incidence of ectopic pregnancies, and therefore the total number of such pregnancies observed is roughly equal to what would be expected if no contraception had been used.

In the past three years we have treated 715 cases with the Yuzpe regimen and had 17 failures, including the present case of an ectopic pregnancy. This rate is compatible with the calculated risk of 10% for ectopic pregnancies.⁴

We believe that this is the first reported case of an ectopic pregnancy after the Yuzpe regimen. We suggest that patients requesting post-coital treatment should be counselled about this rare but serious complication and warned of the need for prompt examination if they develop severe pelvic pain. Until more evidence is available about the presence or absence of a causative relation between postcoital hormonal treatment and ectopic pregnancies, the history of previous tubal pregnancy would be a strong contraindication to postcoital treatment. A careful vaginal examination at the follow up visit is important, as vaginal bleeding associated with an ectopic pregnancy can be so easily mistaken for a period, as in our patient.

¹ Yuzpe A, Percival Smith R, Rademaker AW. A multicentre clinical investigation employing ethinyloestradiol combined with dL-norgestrel as a post-coital contraceptive agent. *Fertil Steril* 1982;**37**:508-13.

² Sparrow MJ. Oestrogen interception: the morning-after pill. *NZ Med J* 1974;**79**:862-4.

³ Smythe AR, Underwood PB. Ectopic pregnancy after post-coital diethylstilbestrol. *Am J Obstet Gynecol* 1975;**121**:284-5.

⁴ Morris JM, Van Wagenen G. Interception: the use of post-ovulatory estrogens to prevent implantation. *Am J Obstet Gynecol* 1973;**115**:101-6.

⁵ Yuzpe AA. Post-coital contraception—new considerations in oral contraception. *Proceedings of an international symposium, Catholic University-Leuven, Leuven, Belgium, Sept 24-25 1981*. Biomedical Information Corporation Publications, 1982.

(Accepted 1 July 1983)

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Superior sagittal sinus thrombosis and essential thrombocythaemia

Both bleeding and thrombosis are complications of essential thrombocythaemia.¹ Common sites of thrombosis are the splenic vein¹ and the small vessels of the foot,² and the sagittal sinuses are rarely affected. Neurological symptoms such as amaurosis fugax, recurrent vertigo, and recurrent hemiparesis may occasionally be associated with thrombosis due to essential thrombocythaemia.³⁻⁴ We describe two patients with essential thrombocythaemia who presented with persistent headaches and chronic papilloedema. One patient was shown to have thrombosis of the superior sagittal sinus, and the second probably had a similar cerebral venous thrombosis.

Case reports

CASE 1

A 28 year old woman teacher presented to this hospital in 1980 complaining of severe headaches during the past six months made worse by coughing and bending. She had had two miscarriages, one in April 1979 at 16 weeks' gestation and the other in April 1980 at 24 weeks' gestation. She had stopped taking the contraceptive pill in April 1978.

On examination she was alert but had severe bilateral papilloedema. Visual acuity was 6/6 bilaterally and the visual fields were normal. There were no other abnormal findings. A gammascan and computed tomography of the head yielded normal results. Lumbar puncture showed a cerebrospinal fluid pressure of 3.15 cmH₂O, but further examination of the cerebrospinal fluid gave normal results. Left carotid arteriography showed superior sagittal sinus thrombosis with occlusion of the sinus at the vertex. Haemoglobin concentration was 13.9 g/dl and white cell count was $9.1 \times 10^9/l$ with a normal differential, but the platelet count was $748 \times 10^9/l$ and subsequently varied between this level and $934 \times 10^9/l$. The erythrocyte sedimentation rate was 15 mm in the first hour. Total cellularity of the bone marrow was normal but there were increased numbers of megakaryocytes. There was no cause for secondary thrombocytosis, and essential thrombo-

cythaemia was diagnosed. Platelet aggregation in response to adenosine diphosphate, adrenaline, and collagen was normal. She underwent bilateral decompression of the optic nerve in October 1980 and was treated with hydroxyurea. The platelet count fell to within the normal range and her symptoms resolved, but mild papilloedema persisted.

CASE 2

In 1977 a 38 year old Iranian woman presented with visual obscurations. Examination showed bilateral papilloedema but no other abnormal physical signs. She had never taken the contraceptive pill. Cerebrospinal fluid tests, a gammascan, and serial computed tomography of the head over three years gave normal results. Benign intracranial hypertension was diagnosed. She was found to have thrombocytosis and was referred to this hospital. Her platelet count was $1326 \times 10^9/l$. Haemoglobin concentration was 13.1 g/dl and white cell count $9.5 \times 10^9/l$ (6% myelocytes, 1% metamyelocytes, 50% neutrophils, 35% lymphocytes, 3% eosinophils, 5% monocytes). Total cellularity of the bone marrow was normal, but increased numbers of megakaryocytes were present. Essential thrombocythaemia was diagnosed. Platelet aggregation in response to adenosine diphosphate, adrenaline, and collagen was normal. She was treated initially with pipobroman and subsequently with one dose of radioactive phosphorus. The thrombocytosis and her symptoms resolved. Mild papilloedema persisted for six years but otherwise she remained well.

Comment

Spontaneous thrombosis of the superior sagittal sinus is rare⁵ and may present as isolated papilloedema. The classic predisposing factors such as sepsis, malignancy, polycythaemia rubra vera, pregnancy, and the puerperium are often absent. This condition was demonstrated in one patient in this report and was strongly suspected in the other; both patients had essential thrombocythaemia. Anticoagulants were not used in either case because of the associated thrombocytosis.

¹ Gunz FW. Haemorrhagic thrombocythaemia: a critical review. *Blood* 1960;**15**:706-23.

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³ Levine J, Swanson PD. Idiopathic thrombocytosis. *Neurology* 1968;**18**:711-3.

⁴ Preston FE, Martin JF, Stewart RM, Davies-Jones GAB. Thrombocytosis, circulating platelet aggregates, and neurological dysfunction. *Br Med J* 1979;**ii**:1561-3.

⁵ Humphrey PRD, Clarke CRA, Greenwood RG. Cerebral venous thrombosis. In: Harrison JG, Dyken ML, eds. *Cerebral vascular disease*. Sevenoaks, Kent: Butterworths, 1983.

(Accepted 12 July 1983)

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Low protein diets in uraemia

Patients with chronic renal failure usually receive dietary advice as a palliative measure to reduce uraemic symptoms, but some start dialysis without having reduced their protein intake. In both experimental animals and man early protein restriction slows the rate of progression of renal failure.^{1,2} We reviewed our experience of low protein diets to alleviate uraemic symptoms in patients approaching end stage renal failure referred to this unit for maintenance dialysis.

Patients, methods, and results

Over the past five years (1977-82) 176 patients were accepted for dialysis; 20 continued to be managed conservatively and 68 received no dietary treatment because of late referral. Of the remainder, 12 had evidence of protein malnutrition—that is, low serum albumin concentrations or low ratios of serum urea to creatinine concentrations—and did not receive advice to reduce protein intake. Ninety six patients were placed on diets containing 35-45 g protein (about 0.6 g/kg body weight) and 19-23 mmol