myeloproliferative disease, strongly suggest that it was caused by the underlying condition. Furthermore, to the best of our knowledge, PRV presenting as disabling glossitis has not previously been reported.

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A case of recurrent eclampsia

Sir,
Eclampsia occurring in successive pregnancies, especially after the second, is a relatively rare condition.^{1,2} We describe here a case who presented with recurrent eclampsia in all her five pregnancies.

A 25 year old gravida 5 came to us at 34 weeks gestation with over 20 generalized tonic–clonic convulsions. She had complained of headache preceding the fits; there was no previous history of fever, vomiting, epigastric pain or blurring of vision. She had not had any antenatal check ups. The woman was not an epileptic but had developed similar tonic–clonic convulsions intranatally in each of her four previous pregnancies. The first child was still-born at term. The second and fourth children, both daughters, 6 years and 1 year old now are growing well. The third child born at 32 weeks gestation died after 10 days. She had a normal delivery each time following augmentation of labour and recovered rapidly after delivery, becoming normotensive without medication at discharge. She had not come for antenatal visits in any of the pregnancies and did not report for follow-up despite adequate counselling. The entire family was illiterate.

At admission, she was semicomatose, the pulse rate 120/minute, the blood pressure 210/130 mmHg, tempera-

ture 102°F, there was no peripheral oedema. The uterus was 34 weeks in size, the presentation cephalic and the head not engaged. The uterus was contracting mildly, once every 10 minutes, lasting 20 seconds and the cervical score was 5.

An intravenous diazepam infusion, phenytoin sodium (100 mg every 8 hours) and nifedipine (10 mg sublingually immediately and every 6 hours), was started along with life support measures. Low rupture of membranes was carried out and oxytocin augmentation started. The woman delivered a live 1.6 kg female baby within 2 hours vaginally (Apgar 6/10 at 1 minute and 8/10 at 5 minutes). The baby is now growing well. The PCV was 34%, urinary albumin present in traces, serum urea creatinine uric acid and liver enzymes were within the normal range, as were the fibrinogen and fibrin degradation product levels. The platelet count was ($120 \times 10^9/l$), the ocular fundi were normal.

The patient recovered well following delivery. There were no further convulsions and her blood pressure was 130/80 mmHg by the fourth postpartum day without medication. She was discharged on the seventh postpartum day, but despite intensive counselling was again not willing for any form of contraception and failed to report for any subsequent follow-up.

Chesley *et al.*¹ have reported that 2% of primiparous eclamptics have recurrent eclampsia in the second pregnancy and Sibai and coworkers³ have also reported a 1.4% incidence of eclampsia in the second pregnancies of such women. We, however, did not come across any report of eclampsia occurring in each of five pregnancies of any women. Home visits with more counselling at home appear necessary for this patient to prevent any future recurrence.

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