Letters to the Editor

Transient Blindness in Pregnancy-induced Hypertension

Sir,

With reference to the three case reports by Drs H. N. Ranganath, B. I. Elhag and A. M. Malibary (Saudi Med J 1991; 12: 441), I would like to report two similar cases which I had the opportunity to manage.

The first case was during my residency period under Professor S. E. Ali in Sylhet Medical College Hospital, Bangladesh, in November 1978. The patient was a 27-year-old woman, second gravidae primagravee, with a history of previous stillbirth following severe pre-eclampsia, 1 year previously.

She was admitted with a 34-week pregnancy complaining of severe headache, vertigo and blurring of vision. She was anaemic with gross oedema and puffy face. Her blood pressure was recorded as 200/100 mmHg. The urine had Alb 3+, and the heart and lungs were normal. The patient was not in labour; the fetal heart was normal and there was a non-engaged cephalic presentation.

The patient was kept under conservative treatment with oral anti-hypertensive and anti-anaeyloptic therapy plus diuretics and vitamin supplements. On the third day of admission she developed severe headache with a high blood pressure of 200/110 mmHg and complained of sudden loss of vision. An ophthalmologist was called in and his examination revealed FC-nil, PR, PI + ve, media-cleear, and attenuated arterioles.

Premature delivery was decided upon and an immediate Caesarean section was performed which produced a healthy baby with Apgar score 9-10, weight 2.5 kg. The mother's recovery was uneventful; on the third postoperative day her visual condition had improved and on the sixth postoperative day she was discharged with a healthy baby and normal vision (V/A = 6/6 unaided).

The second case I saw was during my stay in Iran, in Ayatullah Kashani General Hospital, Baft, Kerman, in June 1991. The patient was a 17-year-old primagravee, with a 36-week pregnancy, severe headache, blurring of vision and gross oedema, puffy face and a high blood pressure recorded as 160/100 mmHg. Her urine Alb was 3+, Hb%: 8g/dl. Her heart and lungs were normal. In the observation ward she complained of severe headache and loss of vision and soon after developed an eclamptic convulsion. She was stabilized with iv. methylioda 250 mg, MgSO4 7H2O in a drip, 4 mg in the first 20 min, then 2 mg in the next 1 hour, and diuretics.

Immediate Caesarean section was performed which produced a living healthy baby with Apgar score 6-8, weight 3.4 kg. The mother's postoperative recovery was uneventful; on the second postoperative day her visual condition improved markedly and on the fifth postoperative day she was discharged with normal vision.

These two cases of severe pre-eclampsia and fulminating eclampsia with transient blindness were managed by immediate Caesarean delivery. Hypertensive retinopathy in acute and severe hypertension is said to be in pre-eclampsia or in fulminating eclampsia usually appears as localized segmental or focal spasm or generalized narrowing of the retinal arterioles. It may occur in all the vessels or in an early stage only in a single vessel resulting in ischaemic retinopathy with optic neuropathy and an insufficient choroidal vascular supply.

These ischaemic attacks of temporary, complete, or relatively complete cessation of blood flow are possibly associated with microembolism of the vessels which deprives the ganglion cells and bipolar cells of their blood supply from the arterioles and their capillaries, and the photoreceptor elements from the choroidal vascular bed. Less often, bilateral ischaemic injuries to the optic visual radiations or occipital cortex produce complete or partial cortical blindness.

These are acute emergency complications rarely encountered in general obstetric practice and they may be fatal, or may result in permanent loss of vision with spontaneous retinal detachment. Early recognition and an instant decision regarding proper management are the only measures which can ensure total reversal and complete recovery.

Pre-eclampsia does not necessarily cause chronic arteriolar changes.

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Saudi Medical Journal 1993; 14(2): 167

References

Dr Ranganath was invited to comment but did not respond to the invitation [Editor].

Aorto-Duodenal Fistula: Secondary to Spinal Tuberculosis

Sir,

Aorto-duodenal fistula is one of the rare causes of major gastrointestinal bleeding with a very high mortality of 50-60%. All cases are either spontaneous due to ruptured aortic aneurysm or postoperative due to aortic prosthesis insertion.

We had a patient who developed aorto-duodenal fistula secondary to tuberculosis of her dorsal spine. This rare aetiology of aorto-duodenal fistula has, to the best of our knowledge, never been reported before.

The patient was a 70-year-old woman who had been undergoing treatment for proven spinal tuberculosis for nearly 5 months before she developed exsanguinating gastrointestinal bleeding. Laparotomy revealed matted paraortic lymph nodes that had invaded the wall of the aorta leading to aorto-duodenal fistula involving the third part of the duodenum. The aorta was otherwise normal with no aneurysm.

Unfortunately we lost the patient because the bleeding was massive and the diagnosis unexpected.

We advise therefore that in patients with tuberculosis, presenting with acute gastrointestinal bleeding, the possibility of aorto-duodenal fistula should be kept in mind and unless
The Need for Revising the MRCP Regulations

Sir,

I have read with interest the short communication by David A. Pyke, Registrar, Royal College of Physicians (Saudi Med J 1991; 12(5): 436–437) regarding the need for revising the MRCP regulations and would like to raise the following points.

1. The current MRCP regulations are particularly detrimental to overseas doctors. The standard should be international and not UK-oriented.
2. There are no centres for MRCP and other examinations in Asian countries like Bangladesh, India, Pakistan and Iran. No consideration has been given to the training capacity of these countries.
3. For overseas doctors rural health services are ignored.
4. There is no appropriate and standard system of scholarships for MRCP and other courses.
5. Like the ECFMG in the USA, a sponsorship system should be introduced.
6. As an international organization the college has no standard rules and regulations like WHO, UNICEF, OK, OAU.

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Saudi Medical Journal 1993; 14(2): 168

Sir,

In answer to the six points above:

1. As I explained in my previous letter (Saudi Med J 1991; 12(5): 436–437), the MRCP(UK) examination is, as it says, a United Kingdom examination. The policy of the Colleges is to encourage other countries to set up their own postgraduate medical examinations.
2. There are no centres for Part 1 MRCP(UK) in India etc. because the authorities do not want the examination there.
3. This is answered by paragraph 1 above.
4. It is hardly reasonable to expect the UK Colleges to find scholarships for overseas candidates to come to the UK to take courses and/or the MRCP. Overseas countries may, and often do, fund their own nationals to come to this country for postgraduate training and diplomas.

Blood Groups and Rh (D) Status in Saudi Newborns

Sir,

I read with interest the letter by David Stevens (Saudi Med J 1992; 1:78) on α-thalassaemia, blood groups and Saudi tribes. It was a useful contribution to our knowledge of gene frequency of A, B, O and Rh (D) in the various tribes of Saudi Arabia serving in the National Guard. I present our experience on the incidence of A, B, O, and Rh (D) in the neonates born at Gurayat General Hospital (GGH), located in the north-western corner of Saudi Arabia. This study was carried out over a period of 8 months from August 1990 to March 1991. During this period all the newborns delivered in the GGH had their cord blood checked routinely for A, B, O and Rh (D) status.

During this period, 1551 babies were delivered and 1349 (87%) were Saudis and 202 (13%) were non-Saudis. The blood group and Rh (D) status of Saudi newborns were as follows: A = 20%, B = 29%, 0 = 47%, AB = 4%, Rh (D) + ve = 93.5% (Table 1). This can be compared with another study conducted by Abdel Gader and his colleagues in Riyadh,1 in a population size of 1235 (age range 11–60 years). Like Stevens we did not analyse the incidence of different blood group and Rh (D) antigens according to the various tribes. It is possible that there are some genetic differences between the various tribes of Saudi Arabia. The population of Gurayat region are mostly of the Sharari tribe, the rest are Rowelli and a few are from the Al-Anzili tribe. In Table 1 the blood group of expatriate neonates were also compared, but their number was very small compared with Saudi neonates. The majority of these expatriates were of Arab origin from Jordan, Palestine and Egypt. For comparison, the blood groups of a Caucasian population2 are also included in the table. It is good news that the incidence of Rh(D)−ve in Saudi neonates is low, 6.5% compared with a 15% prevalence in a USA population, so the incidence of Rh haemolytic disease in USA in newborns would be higher if the mothers were not injected promptly after birth with anti Rh (D) immunoglobulin. This report highlights the incidence of blood groups in a population of Saudi Arabia.

<table>
<thead>
<tr>
<th>Nationality and (Place of study)</th>
<th>Sample size and (characteristics)</th>
<th>A No. (%)</th>
<th>B No. (%)</th>
<th>O No. (%)</th>
<th>AB No. (%)</th>
<th>Rh (D) +ve No. (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Saudi (Gurayat)</td>
<td>1349 (Neonates)</td>
<td>271 (20)</td>
<td>384 (29)</td>
<td>637 (47)</td>
<td>57 (4)</td>
<td>1262 (93.5)</td>
</tr>
<tr>
<td>Non-saudi (Gurayat)</td>
<td>202 (Neonates)</td>
<td>64 (32)</td>
<td>48 (24)</td>
<td>74 (36)</td>
<td>16 (8)</td>
<td>188 (93)</td>
</tr>
<tr>
<td>Saudi (Riyadh)</td>
<td>1435 (11–60 years)</td>
<td>– (27)</td>
<td>– (20)</td>
<td>– (49)</td>
<td>– (4)</td>
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<tr>
<td>USA</td>
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<td>– (27)</td>
<td>– (20)</td>
<td>– (49)</td>
<td>– (4)</td>
<td>– (85)</td>
</tr>
</tbody>
</table>

Table 1

A, B, O blood group and Rh (D) gene frequency in different population