LETTERS TO THE EDITOR

Meningoencephalitis after measles-mumps-rubella vaccine

SIR,—Altogether 124 health districts in this country have now achieved 90% coverage for measles-mumps-rubella (MMR) vaccine.¹ Against this background, there have been several reports of meningoencephalitis temporally associated with the administration of MMR.² The adverse publicity such cases generate and their potential seriously to harm a vaccination programme must not be underestimated. We would like to describe one suspected case which, despite subsequent exoneration of the vaccine, had an immediate impact on MMR coverage.

An 11 year old boy was admitted to hospital with a 72 hour history of vomiting and headache. He had received MMR vaccine 10 days before the onset of symptoms. On arrival he was afebrile and semiconscious but immediate investigations (including microscopy of the cerebrospinal fluid) were unremarkable. He was started on acyclovir and dexamethasone but died 48 hours after admission. The cause of death was thought to be vaccine induced mumps encephalitis and this was quickly disseminated on the district medical and nursing network.

Immediately after the child's death the local director of public health convened an action committee. A press statement was released to the effect that there was no evidence to implicate the vaccine as the cause of death. Investigations subsequently revealed serological evidence of past mumps infection. No mumps virus was demonstrated on direct polymerase chain reaction of brain tissue. The only virus isolated was an echovirus type 25 from nasopharyngeal secretions. The committee therefore produced a further press statement confirming that there was no link between the vaccine and this child's death.

Professional and parental anxiety led to a sharp fall in MMR vaccine uptake that has only been restored after six months of considerable efforts by district staff. This case illustrates the importance of thorough investigation of a suspected vaccine reaction and ensuring a coordinated district response. We advise caution in labelling such an incident as vaccine related until conclusive evidence is obtained.

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1 Handford SG, Begg NT. COVER (cover of vaccination evaluated rapidly): 17. *Communicable Disease Review* 1991;1:R38.

2 Anonymous. Mumps meningitis and MMR vaccination [Editorial]. *Lancet* 1989;ii:1015-6.

A new syndrome of congenital hypoparathyroidism, severe growth failure, and dysmorphic features

SIR,—We were interested to read the paper by Dr Sanjad and colleagues¹ as we previously described an identical group of patients in this journal.²

(1) The patients in our paper were from several parts of the Middle East and were not exclusively from Kuwait. This would suggest that the gene frequency is widely distributed throughout that region, and is not confined purely to Saudi Arabia as suggested by Sanjad *et al* in their paper.

(2) No comment was made relating to the skeletal surveys of these patients and we would be interested to hear whether the skeletal abnormalities described in our paper were present in these children.

(3) Although the patients in the paper by Sanjad *et al* were quoted as having normal T cell function, we note that four of their patients died during the first two years of life, possibly because of intercurrent infections.

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1 Sanjad SA, Sakati NA, Abu-Osba YK, Kaddoura R, Milner RDG. A new syndrome of congenital hypoparathyroidism, severe growth failure, and dysmorphic features. *Arch Dis Child* 1991;66:193-6.

2 Richardson RJ, Kirk JMW. Short stature, mental retardation, and hypoparathyroidism: a new syndrome. *Arch Dis Child* 1990;65:1113-7.

Dr Sanjad and colleagues comment:

We appreciate Drs Richardson and Kirk's interest in our paper on congenital hypoparathyroidism, recently published in the journal.¹ In our original description of the syndrome in 1988 we described the clinical and laboratory findings in five infants with severe intrauterine and postnatal growth retardation, congenital hypoparathyroidism, and dysmorphic features.²

(1) We do not deny that the gene for this syndrome may be widely distributed throughout the Arabian peninsula but were intrigued by the fact that almost 60% of the patients in our series originate from the western province of Saudi Arabia.

(2) Skeletal surveys were performed in eight of our 12 patients. They revealed delayed bone age and varying degrees of demineralisation but no medullary stenosis of the long bones as described by Richardson and Kirk.

(3) Cellular immunity, measured in five patients (and in another two, subsequent to our publication), was found to be intact—both quantitatively and functionally as detailed in our paper. While two of four deaths among our patients were attributed to infections, factors other than cellular or humoral immune deficiencies would have to be invoked. Again, this is in contrast to Richardson and Kirk's experience where four patients were found to have reduced number of T lymphocytes.

In summary, the dysmorphic features and severe growth retardation in Richardson and Kirk's patients are indeed similar to the ones we have described. The skeletal and immune abnormalities in their patients probably

represent additional variants to the same syndrome.

1 Sanjad SA, Sakati NA, Abu-Osba YK, Kaddoura R, Milner RDG. A new syndrome of congenital hypoparathyroidism, severe growth failure, and dysmorphic features. *Arch Dis Child* 1991;66:193-6.

2 Sanjad S, Sakati N, Abu-Osba Y. Congenital hypoparathyroidism with dysmorphic features—a new syndrome. *Pediatr Res* 1988;23:271A.

Gonadal function after allogenic bone marrow transplantation for thalassaemia

SIR,—De Sanctis *et al* in their paper on gonadal function after allogenic bone marrow transplantation for thalassaemia observed gonadal damage in 80% of their girls with raised gonadotrophins and unmeasurable oestradiol concentrations.¹ In contrast all the boys had gonadotrophin concentrations within the normal range with variable gonadotrophin responses to an intravenous dose of gonadotrophin releasing hormone. They concluded that the chemotherapy used as a preparative regimen for bone marrow transplantation in prepubertal life led to a higher incidence of gonadal toxicity in girls than boys.

We disagree with the conclusions of the authors. There is a body of evidence to suggest that the germinal epithelium of the testis is more vulnerable to cytotoxic damage from either chemotherapy or radiation than the Leydig cells of the testis or the ovary.² Furthermore although it may be difficult to diagnose gonadal damage in prepubertal life, the age range of the two sexes at the time of the study was similar.

We would suggest that the results presented by De Sanctis *et al* do indicate a sexual dimorphism in thalassaemic children, not in terms of their gonadal response to chemotherapy damage, but rather in the persistence of gonadotrophin deficiency after iron deposition in the hypothalamic-pituitary axis. The question to ask is why the thalassaemic boys failed to mount an appropriate gonadotrophin response to gonadal damage in the presence of similar ferritin concentrations to those seen in the girls, whose gonadotrophin responded appropriately.

Incidentally, the testosterone concentrations quoted both for the thalassaemic boys and the controls are incorrect. The normal adult male range for testosterone is 10-30 nmol/l.

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1 De Sanctis V, Galimberti M, Lucarelli G, Polchi P, Ruggiero L, Vullo C. Gonadal function after allogenic bone marrow transplantation for thalassaemia. *Arch Dis Child* 1991;66:517-20.

2 Shalet SM. Gonadal function following radiation and cytotoxic chemotherapy in childhood. *Advances in internal medicine and pediatrics* Berlin and Heidelberg: Springer-Verlag 1989; 58:1-21.

Drs De Sanctis and Galimberti and Professor Lucarelli comment:

We thank Drs Ogilvy-Stuart and Shalet for their interest in our paper and for their criticism.