Letters to the Editor

Rupture of the Female Urethra

Sir

I was interested in Zein and Sidani's Case Report of rupture of the urethra in a 12-year-old girl, in the January edition of the Saudi Medical Journal (1992; 13: 63-64). Certainly, I would confirm their experience and that of others as regards the rarity of this injury.

In my department in Wellington, New Zealand there are two recorded cases which occurred in the last 25 years, one of which came under my care in 1975. She was a 3-year-old child who was accidentally run over when her father unknowingly backed his truck over her. She sustained a fractured pelvis along with complete avulsion of the urethra and transection of the vagina. The vagina was repaired forthwith by simple suture, while urethral continuity was re-established with tubed split skin splinted with a Foley Catheter. Her postoperative recovery was without event. She was followed regularly thereafter with 3-monthly urethral calibrations for a year, after which the intervals were lengthened. Urinary control was uncertain for 3 years but rectified itself only to recur again for 18 months some 4 years later. It was mainly stress-related but it settled.

She is now aged 17 years and when last seen 2 years ago she had a good urinary stream, virtually normal control, although violent physical exercise was occasionally associated with dampness. Her vagina is completely normal in length and distensibility with no visible signs of an earlier transection.

The other patient was a young girl also involved in road trauma and treated by primary repair. Unfortunately her family left the district and she was lost to follow-up.

My feeling, based as it is on the experience of one patient only, is that approximation of the ends of the ruptured urethra using the simplest technique possible should be the first priority. Subsequent treatment may well be necessary and will be determined by whether stricture formation and/or incontinence follows. With luck, neither may occur.

I congratulate the authors on an interesting and instructive contribution.

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Saudi Medical Journal 1993; 14(1): 83

Sir

This letter is in response to Dr Urquhart-Hay. The difference between the two cases that Dr Urquhart-Hay presented and our case is that his cases presented to him firstly and he was able to manage them primarily. However, our case presented after being operated on in a peripheral hospital. I tend to agree with Dr Urquhart-Hay that primary repair can be attempted; however, if associated bowel injury is present, a higher incidence of stricture and fistula formation might take place. I also reemphasize that this injury is a very rare entity and experience from one centre is inadequate so a collective collection of case reports of management of urethral injuries should be reported so more experience can be gained. We thank Dr Urquhart-Hay for his contribution and hope that other doctors will report their experience so we can learn more about this entity.

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Interferon-gamma Control in Relation to Infection

Sir

Since I wrote my article,1 Intracellular Survival on Microorganisms, there has been further clarification of the role of interferon-gamma in host defence. It would therefore save interested readers much trouble if I could make a few points.

Interferon-gamma (γ-IFN) can originate from CD4 T helper cells, from CD8 cytotoxic T cells or from natural killer cells.2 In particular it is now clear that the helper cells from which IFN-gamma comes are Th-1 cells, which are inflammatory or delayed hypersensitivity T cells. The alternative cell type is a Th-2 T cell that produces the cytokines IL-4, IL-5 or IL-6 which cause B cell production of antibodies. Th-1 and Th-2 cells can now be identified in man3 as well as in the mouse.

In leishmaniasis a good Th-1 cell response with production of cytokines IL-2 and IFN-gamma will lead to a healing response in which activated macrophages play an offensive role. Conversely in situations in which Th-2 cells predominate, antibody formation will be adequate but cell-mediated immunity (CMI) will be defective. As I discussed before,4 one may have to give interferon-gamma in order to boost the (CMI). Also in schistosomiasis the early response is usually a Th-2 cell response, since it is promoted by antigens from the eggs.4 Only if there has been previous vaccination (which boosts IFN-gamma output), or much later in the illness, do Th-1 cells start to predominate and mount an eradicative delayed hypersensitivity reaction.

It is now established that it is the release of interleukin 105 by Th-2 cells that leads to the down-regulation of the Th-1 response. So, for example, it has been shown that IL-10 blocks the ability of IFN-gamma to inhibit the intracellular replication of Tryp. cruzi in mouse peritoneal macrophages.6 Furthermore interleukin 4 from the Th-2 cells suppresses production of IFN-gamma,7 and transforming growth factor TGFBeta8 de-activates macrophage responses.

All this is to say that there are fine controls over all the several cells in the immune system. Normally 'macrophage activating factors' such as IFN-gamma, tumour necrosis factor alpha or granulocyte-macrophage colony stimulating factor (GM-CSF) should so boost macrophage reactivities that they are able to kill intracellular organisms. Yet unfortunately the microorganisms have often learned how to subvert the immune defence system.

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References