REVIEW

Mumps orchitis

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SUMMARY

Although the incidence of mumps orchitis has dramatically declined since the introduction of the childhood vaccination programme, a sharp increase in reported cases of both mumps and mumps orchitis has been seen recently in the UK. There are great concerns about mumps outbreaks and the associated risk of infertility; it remains an important clinical condition. Immunization is the best policy to avoid this viral disease.

INTRODUCTION

The incidence of mumps orchitis has declined dramatically since the introduction of the childhood vaccination programme. Over the last few years mumps orchitis has rarely been seen in our institution: however, more recently, 11 patients with mumps orchitis were admitted to our unit between March and September 2005.

This sharp increase was also noticed elsewhere in the UK; 25 cases of mumps orchitis were reported by the Urology Department of the Royal Liverpool University Hospital between September 2004 and April 2005.[1]

METHODS

We conducted a MEDLINE/PubMed search (search terms 'mumps, orchitis, infertility') and review of the literature with emphasis on management.

Natural history

Mumps is a contagious viral disease that often results in painful swelling of the parotid gland. About 30%–40% of patients with mumps do not develop parotitis. The virus is an RNA virus of the genus *paramyxovirus* which is spread from human reservoir by direct contact, airborne droplets, fomites contaminated by saliva and possibly by urine.

The microbiological diagnosis is by serology or virus culture. Enzyme immunoassay for mumps immunoglobulin antibodies are most commonly used for diagnosis. IgM antibodies are detectable in the first few days of the illness and are considered diagnostic. In addition, seroconversion or a fourfold increase in IgG titre are also diagnostic.[2]

The measles, mumps and rubella (MMR) vaccine was introduced in the UK in1988. In 1996, a second dose of the vaccine was routinely introduced to all children born in 1991 onwards. A large increase in both notifications and laboratory confirmed cases were observed in 2003 which has continued into 2004 and 2005 (Table 1).[3]

Ninety per cent of confirmed cases in 2004–2005 were among people aged 15 years and over. This age group had either never received any MMR vaccine because they were too old when it was introduced, or had only received a single dose. Uptake of MMR vaccination in the UK has fallen from a peak of 92% in early 1995 to a national level of 82% in 2003 (at the age of 2). In London, uptake is now less than 75%—much less in some areas—which is leading to significant risk of outbreaks.[3]

Mumps orchitis is now rarely seen in children under 10.[4] Orchitis is the most common complication of mumps in post-pubertal men, affecting about 20%–30% of cases:[5] 10%–30% are bilateral.[2] Orchitis usually occurs 1–2 weeks after parotitis.

Of affected testicles, 30%–50 % show a degree of testicular atrophy.[2] Within the first few days of infection the virus attacks the testicular glands, leading to parenchymal inflammation, separation of seminiferous tubules and perivascular interstitial lymphocyte infiltration.

Table 1 **All laboratory confirmed cases of mumps (England and Wales 1996–2005). Source: Communicable Disease Surveillance Centre**

Year	*Mumps cases*
1996	94
1997	180
1998	119
1999	372
2000	703
2001	777
2002	502
2003	1556
2004	8130
2005	43322

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The tunica albugenia forms a barrier against oedema, and the subsequent rise in intratesticular pressure leads to pressure-induced testicular atrophy.[5]

Adamopoulos *et al*.[6] studied the effects of mumps orchitis on Leydig cell function and found low testosterone levels, elevated leuteinizing hormone levels and an exaggerated pituitary response to leuteinizing hormone-releasing hormone (LHRH) stimulation in the acute phase. Whilst basal testosterone concentrations returned to normal after several months, mean basal follicle stimulating hormone and leuteinizing hormone concentrations remained significantly increased at 10 and 12 months after the acute phase.

The causal link between mumps orchitis and anti-sperm antibodies has been unclear. Although the antibodies were suspected to impair fertility, Kalaydjiev *et al*.[7] demonstrated that both the incidence and the level of serum anti-sperm antibodies among mumps orchitis patients were low, and did not support the hypothesis of an enhanced humoral immunity against spermatozoa.

Mumps orchitis rarely leads to sterility but it may contribute to subfertility. It can also can lead to oligospermia, azoospermia, and asthenospermia (defects in sperm movement). Unilateral disease can significantly, but only transiently, diminish the sperm count, mobility, and morphology. Impairment of fertility is estimated to occur in about 13% of patients,[2] while 30%–87% of patients with bilateral mumps orchitis experience infertility.[8]

TREATMENT

Treatment is supportive (bed rest, scrotal support, and the use of nonsteroidal anti-inflammatory agents). We have found two studies which supported prescribing broad-spectrum antibiotics.[8,9] They claimed that bacterial infection of the oedematous testicular tissues cannot always be ruled out.

Steroid administration helps in diminishing pain and oedema, but it does not alter the clinical course of the disease or prevent future complications. The benefits must be weighed against the self-limiting nature of mumps orchitis and the potential side effects of steroid treatment. Adamopoulos *et al*.[6] found that corticosteroids may reduce pain and oedema, but it caused the testosterone levels to decrease and follicle stimulating hormone and leuteinizing hormone levels to increase. Smith and Bishir[10] described the use of high doses of steroids in reducing pain, but they had little effect on local findings and subsequent clinical course of the disease. Bertschat *et al*.[11] in his small series found that patients on corticosteroids showed better semen analysis parameters at follow up examinations, although it was not significant.

As interferon inhibits transcriptase-induced viral replication, it would be expected that systemic interferon treatment could prevent the development of testicular atrophy and infertility. Erpenbach *et al*.[12] claimed in 1991 to have prevented testicular damage and infertility in four patients who had bilateral mumps orchitis by using systemic interferon-α 2B for seven days. Those cases with severe subfertility before treatment improved to normospermia and remained fertile during the 12–20 month follow-up period. Ku *et al*. [13] studied the effect of interferon-α 2B on 13 patients with mumps orchitis in comparison to eight infected patients who did not receive therapy. No patient in the treatment group developed testicular atrophy as opposed to three in the control group. The sperm count was improved in all patients treated with interferon, but low sperm count persisted in four from the control group. Abnormal sperm morphology persisted in both patient groups. Yeniyol *et al*.[14] studied the effects of interferon-α 2B on 18 patients with mumps orchitis. Patients were evaluated by testicular biopsy after a year of treatment. The biopsies showed total atrophy of the seminiferous tubules in 39%, 10% atrophy in 16%, and no apparent histological alterations, except an arrest in spermatogenesis, in 45%. They concluded that systemic treatment with interferon is not completely effective in preventing atrophy after mumps orchitis.

In the past, surgical management with early incision of the tunica albuginea was tried, but failed to prevent development of testicular atrophy.[15] Mumps outbreaks are now seen only sporadically. Therefore, evaluation of any treatment is difficult as it is not possible to recruit adequate number of patients for controlled studies.

When azoospermia occurs as a consequence of mumps orchitis, it does not necessarily cause complete absence of spermatozoa in the testes. Testicular biopsy often yields occasional spermatozoa. The development of intracytoplasmic sperm injection has significantly changed the treatment of male subfertility. Fertilization and pregnancy can be achieved after retrieving a few spermatozoa.

CONCLUSION

Small mumps outbreaks and their complications continue to be identified, and it remains an important clinical condition. There are great concerns about subsequent infertility in these patients. Immunization is the best policy to avoid mumps-related complications. Treatment remains conservative.

Competing interests None declared.

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