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Asthenoazoospermia in patients receiving anti-tumour necrosis factor α agents

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Tumour necrosis factor α (TNFα) is known to affect gonadal cell viability and function by several pathways: inhibiting germ cell apoptosis[1]; promoting Sertoli cell androgen receptor expression and activity[2]; and inhibiting Leydig testicular cell steroidogenesis at the transcriptional level.[3] Recently, infliximab (that is, chimeric monoclonal anti-TNFα antibody) has been shown to block the survival promoting effect of TNFα on seminiferous epithelium in vitro.[4] These data prompted us to search for any alteration in gonadal function or spermatogenesis, or both, in patients undergoing anti-TNFα treatment.

At present, we have investigated three male patients (median age 40 years, range 35–51), all with ankylosing spondylitis (median disease duration 14 years, range 12–22), according to modified New York criteria,[5] and treated with infliximab only (5 mg/kg/6 weeks, after the induction period for 8, 13, and 24 months, respectively).

We detected no alterations in serum follicle stimulating hormone, luteinising hormone, prolactin, and testosterone levels. Nevertheless, we found asthenoazoospermia in two of the patients, who were both previously fathers of sons. We are unable to confirm that asthenoazoospermia depends only on the drug. Nevertheless, the above mentioned evidence for the effect of TNFα on gonadal function leads us to think that this is the case.

Further studies are needed to confirm or refute the influence of anti-TNFα antibody on spermatogenesis. It would be useful to know of such an effect when planning anti-TNFα treatment in male patients.

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