Author's reply

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Sir

We thank the authors for their interest in our article and offer the following clarifications to their queries:

1. In the first place, our aim was to determine the impact of curcumin on FCA-induced arthritis when it is used in combination with methotrexate, and secondly whether this combination could reduce hematological toxicity or not? Therefore, if we segregate the above-said purposes of the study, it may lead to deviation from the main theme of the study; which is exactly what the reader has done. As a therapeutic strategy, methotrexate – a disease modifying antirheumatic therapy – is always incorporated in a dose of 2 mg/kg. Therefore, our study maintained one group treated with the standard dose of methotrexate (2 mg/kg). As prolonged treatment is desirable in rheumatoid arthritis, it has been observed that compliance was low or was discontinued not because it was incapable of suppressing RA, but because of the ensuing adverse effects. We were interested to know whether reduction in dose in the presence of curcumin could offer the same protective measures as seen with methotrexate at 2 mg/kg dose; and if yes, whether this dose could reduce adverse effects. Based on this, we compared the hematological toxicity observed with methotrexate plus curcumin with methotrexate (2 mg/kg) alone. Therefore, in our opinion, comparison of hematological toxicity between the two groups was appropriate.
2. The aim of our experiment was primarily to reduce the dose of methotrexate in the management of rheumatoid arthritis as the dose of 2 mg/kg often leads to noncompliance owing to the adverse effects. Therefore, option 2 of the reader's suggestion does not suit our study. The proposition of keeping one more group with a dose of 1 mg/kg is a valid suggestion which will be taken up in future studies. We would like to bring to the notice of the reader that the combination with curcumin produced significant reduction in paw edema with minimal damage to the hematological system. Therefore, we have not only contributed this response in our discussion section to curcumin but also attributed it to the reduction in the dose of methotrexate. The aim was not purely to reduce hematological toxicity, but alsoto search for an adjuvant which can support the action of methotrexate so that compliance can be improved for a disorder such as RA which requires long-term treatment.
3. Curcumin is not soluble in saline and cannot be used in any of the solvents mentioned above as they are least preferred for parenteral administration. Curcumin is not soluble in gum acacia as well, but can be suspended in gum acacia. We have dispersed curcumin in a saline solution (0.9% NaCl) which, in addition, contained 0.3% sodium carboxy methyl cellulose, and we have termed it as saline control.
4. We have followed standard procedures in our study and they have been further standardized in our laboratory to conform the dose required and duration for complete induction of arthritis. As our treatment was initiated from the ninth day, we considered day 9 values as baseline data. Further, the aim of our study was to assess the curative action of methotrexate plus curcumin and not the preventive response. If we were focusing on the preventive response, it would be absolutely essential for us to reflect the day 0 values. As day 9 was the first day of treatment, large variations cannot be expected within a few hours after initiating the treatment.

We hope that these clarifications have addressed the queries adequately.

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Uncontrolled penile erection and increased sexual desire with intravenous moxifloxacin

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Sir,

We report an adverse reaction of uncontrolled penile erection and increased sexual desire due to moxifloxacin. To the best of our knowledge, this adverse effect has not been reported in the literature.

A 31-year-old male patient presented to our clinic with complaints of high fever, headache, myalgia, arthralgia, and

purulent postnasal drip. Past medical history was unremarkable. There was a family history of hypertension. The respiratory system including chest x-ray was normal. Acute sinusitis was diagnosed and patient was prescribedmoxifloxacin 400 mg/day intravenously. The patient presented with complaints of increased sexualdesire and uncontrolled penile erection at 12 h after the treatment. His fever was normal after 48 h. Intravenous moxifloxacin was replaced by to oral dosageon the third day. Complaints of uncontrolled penile erection and increased sexual desire of the patient were improved at the end of the fifth day. Oral moxifloxacin was continued to the 10th day. No symptom was observed on follow-up of the patient.

Moxifloxacin is a quinolone antibiotic. Quinolones are well tolerated with safety profiles similar to those of other antimicrobial agents. Some adverse effects as tendinitis and CNS-related effects are more common with quinolones than other antimicrobial agents.[1]

Brown reported a 31-year-old patient with prostatitis and premature ejaculation. He emphasized a therapeutic effect of ciprofloxacin on premature ejaculation (prolongation of ejaculation from 1–2 to 4–6 min) which was reversible.[2]

CNS side effects after administration of quinolones have been reported at the rate of 1–2%. The most common symptoms involve dizziness, somnolence, and headache. The less commonly reported symptoms have involved agitation, confusion, delirium, abnormal vision, and organic psychosis.[3] Abnormal dreams, depersonalization, depression (potentially culminating in self-endangering behavior, and emotional lability have been reported at the rate of less than 0.1%.[4] Moxifloxacin is a quinolone with potential for side effects associated with CNS.[1] Klossek *et al*. reported that CNS events such as dizziness and vertigo after moxifloxacin are observed more than five times more often than trovafloxacin.[5]

The cerebral areas especially the limbic system and hypothalamus are responsible for sexual function. However, it has been thought that anterior hypothalamic region and medial preoptic nucleus manage sexual behavior in men. It is known that dopamine is the most important neurotransmitter in sexual desire, fantasy, and motivation. Testosterone is also responsible for sexual desire.[6] To the best of our knowledge, differing side effects between intravenous or oral forms of moxifloxacin have not been described yet. In this particular case, causality assessment using the Naranjo scale,[7] showed that intravenous moxifloxacin was probably the causal drug for this adverse event.

Intravenous and oral moxifloxacin may differ in type and severity of adverse effects. Although rare, moxifloxacin may cause CNS adverse events that are reversible. Patients receiving intravenous moxifloxacinshould be closely followed-up and observed for these adverse events.

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Steven–Johnson syndrome may NOT be due to ayurvedic drugs

1

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Sir,

A recently published article in IJP[1] (Steven-Johnson syndrome due to Ayurvedic drugs) has a number of flaws. The medicine quoted by the authors fulfills the criteria for "misbranded" drug rather than anything else. Authors say that the pills resembled small balls and were white in color. Usually, Ayurvedic pills are herbal extracts and no dosage forms in