Sir,
I read the article of Garg, et al.[1] with great interest. As a co-author of our work[2] cited in their article, I would like to make it clear that the microfilariae detected in the pleural biopsy material of our case were of *Wuchereria bancrofti* and not *Mansonella perstans* as quoted in the text by the authors. I was surprised to note that the closed pleural biopsy was not carried out as there was a chance to establish the filarial etiology within the pleura. Microfilariae reside in the arterioles of pulmonary system during daytime and appear in peripheral blood and other body fluids only in the night time during the peak biting time of mosquito vectors. The traditional diagnostic method of filariosis is to demonstrate microfilariae microscopically in the peripheral blood (capillary finger prick or thick venous blood smears) drawn in the night or presence of circulating filarial antigen.[3] Filariosis is a major health problem in India and microfilariae have been detected along with other diseases such as tuberculosis, non-Hodgkin’s disease, etc.[4] I am curious to know the scientific basis regarding the number of times a clinical specimen like pleural fluid can be tested and methodology adapted by the authors who successfully demonstrated microfilariae on all four occasions to conclude that the pleural effusion was due to filariosis only.

### Filarial pleural effusion

Sir,

I read the article of Garg, et al.[1] with great interest. As a co-author of our work[2] cited in their article, I would like to make it clear that the microfilariae detected in the pleural biopsy material of our case were of *Wuchereria bancrofti* and not *Mansonella perstans* as quoted in the text by the authors. I was surprised to note that the closed pleural biopsy was not carried out as there was a chance to establish the filarial etiology within the pleura. Microfilariae reside in the arterioles of pulmonary system during daytime and appear in peripheral blood and other body fluids only in the night time during the peak biting time of mosquito vectors. The traditional diagnostic method of filariosis is to demonstrate microfilariae microscopically in the peripheral blood (capillary finger prick or thick venous blood smears) drawn in the night or presence of circulating filarial antigen.[3] Filariosis is a major health problem in India and microfilariae have been detected along with other diseases such as tuberculosis, non-Hodgkin’s disease, etc.[4] I am curious to know the scientific basis regarding the number of times a clinical specimen like pleural fluid can be tested and methodology adapted by the authors who successfully demonstrated microfilariae on all four occasions to conclude that the pleural effusion was due to filariosis only.
Author's reply

Sir,

This is in reference to the letter by an esteemed reader. I appreciate the interest shown by him regarding our case. We regret that the case reported by him was wrongly quoted in our article as caused by *Mansonella perstans* and apologize for the inadvertent error. The only reported case of pleural effusion due to *M. perstans* was of Kahn in 1983.[1]

We did not consider pleural biopsy as the diagnosis was firmly established by repeated demonstration of microfilariae in pleural fluid, and the patient denied consent for pleural biopsy. The literature shows that pleural biopsy does not always demonstrate microfilariae.[2,3]

The method used for demonstration of microfilariae in pleural fluid was hematoxylin and eosin stain on smears prepared from the sediment of pleural fluid after centrifuging at 2000 rpm for 15 min. Pleural fluid samples were collected at night time or in early morning.

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What dose of anti-snake venom should be given in severe neuroparalytic snake bite?

Sir,

Indian cobra (*Naja naja*) and Common Indian krait (*Bungarus caeruleus*) are two important species of elapid snakes found in India and are responsible for most of the cases of neurotoxic snake bite. Respiratory failure is the most important cause of morbidity and mortality in victims of neurotoxic snake bite.[1,2]

Cobratoxin and *α*-bungarotoxin act postsynaptically by binding to acetylcholine receptors on the motor end plate while *β*-bungarotoxin and crotoxin act presynaptically and prevent release of acetylcholine at the neuromuscular junction.[3,4]

Timely administration of anti-snake venom (ASV) along with cardiorespiratory support is the only effective treatment available for neurotoxic snake bite.[3,4]

ASV is the most effective when administered early enough to neutralize venom in the circulation before it reaches the target site. However, there is no universal consensus on the optimal dose and protocol of ASV administration. Higher doses of ASV had been used earlier with the hope of early recovery.[5]

Other investigators have found no significant difference on survival outcome and duration of ventilation while comparing high dose ASV regimens with low dose ASV regimens.[6]

Fifty-eight patients with severe neurotoxic snake bite with respiratory failure were admitted to MICU during the study period. Of this there were 41 males and 17 females. The

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References

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