LETTER TO THE EDITOR

PEPTIC ULCER AND SMOKING

Dear Editor,

The Article by N. A. Strugnell on reducing mortality from perforation of peptic ulcer in Nepal needs to be highlighted for several reasons.¹

Firstly, it has been shown that applying measures like mortality studies and especially auditing will help us to find out faults in our management system and corrective steps can be taken to help effective care of the patients. The need for this is not only found in tertiary centers but also in remote hospital where patient care has to be delivered with minimum resources.

Peptic ulcer disease is now approached as an infectious disease, in which elimination of the causative agent cures the condition.² Today, H. pylori is considered as a causative factor for the majority of duodenal and gastric ulcers encountered in routine clinical practice.

Clinically relevant eradication regimens should have cure rates of at least 80b percent and the benefit of treatment is durable.²

One seroprevalence study done in Nepal has shown that:

1. Prevalence of infection significantly increased with age, and
2. There was no difference in H. pylori positivity between individuals with and without upper abdominal symptoms in both villages where study was conducted.³

The conclusion was that there was significant regional difference in the seroprevalence of H. pylori within Nepal, which showed lower prevalence in an isolated rural village.

In the context of the above study and increasing resistance of H. pylori to antibiotics and the high cost of the regimens, I wonder if the protocol mentioned by N. A. Strugnell is applicable in the villages of Nepal. However we need further seroprevalence studies.

The prognosis of ulcer perforation today is determined by patient's age, the site of perforation and the delay in treatment.⁵

The last factor is the only one which can be modulated by good clinical practice. Delay in treatment for more than 12 and 24 hrs increases mortality by threefold and ninefold respectively.⁴

Poorer survival in perforated ulcer survivors is predominantly due to smoking related diseases such as lung cancer or benign respiratory and cardiovascular diseases.⁵

To conclude, with no major role for H. pylori in perforation of peptic ulcer established, prevention of ulcer perforation can be achieved mainly through smoking prevention. It is high time that the authorities deliver this message to general public through conduction of mass education programmes and combine this with existing programmes for prevention of smoking.

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