Germany) viscous gel in 10 of 51 (20%) patients. Intravenous tramadol median dose 100 mg/day was administered for breakthrough or persistent severe pain in five of 51 (10%) patients.

The side effects of TB were observed in seven of 51 (14%) patients. Their occurrence, however, led to discontinuation of the TB treatment in an attempt to not worsen the patients' subjective quality of life (i.e., in the case of nausea despite antiemetic medication) or to avoid somnolence and dizziness. No serious adverse event was observed or any technical problems with the patch.

In conclusion, the OM pain intensity did not progress in most patients within the first three to five days of the treatment with TB 35 μg/hour or 52.5 μg/hour. Probably, the TB served as an effective prevention from pain progression at the time of OM development and peak activity phase. Significant decrease of pain intensity to "mild pain" level (VAS score <4) was observed within five to seven days of the TB use. Despite this treatment, however, there still remained some individuals (five of 51, 10%) who appeared “refractory” and experienced severe pain (VAS score ≥7).

In our experience, buprenorphine was at least partially effective in pain control in most of the patients and was well tolerated. The limitations that must be taken into account are the slow onset of analgesic action and that prompt on-demand adjustment of the dose is not possible (OM pain usually gets worse during swallowing, chewing, or speaking). If considered for OM treatment, TB appears suitable for patients at high risk for severe and long-duration OM, and the “in advance” application of TB should be preferred. Definite recommendation, however, cannot be made yet and large, prospective, and well-designed observations are needed for that.

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References

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Laryngospasm is an involuntary contraction of the intrinsic muscles that control the vocal

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cords or the extrinsic muscles of the larynx. Involuntary contractions result in either a partial or a complete closure of the upper airway, obstructing airflow into and out of the lungs. Risk factors for laryngospasm include general anesthesia, upper respiratory tract infection, asthma, gastroesophageal reflux (GER), and exposure to environmental tobacco smoke. So far, we know of no reported cases of laryngospasm resulting from Helicobacter pylori infection. Here, we report what we believe to be the first case of H. pylori-induced laryngospasm for a patient in climacteric.

**Case**

A 53-year-old woman presented with a one-week history of abrupt awakenings associated with gasping and coughing. She was H. pylori positive, diagnosed by gastric histology and the C-urea breath test. She also was diagnosed with sleep-related GER by an esophageal pH probe. Other laryngospasm-related causes were excluded, including general anesthesia, upper respiratory tract infection, and asthma.

She was adequately treated with a proton pump inhibitor, but the symptoms were not significantly improved. The patient then received a one-week course of triple combination therapy (amoxicillin 1000 mg, clarithromycin 500 mg, and omeprazole 20 mg twice daily). The treatment efficacy of H. pylori eradication was assessed using the C-urea breath test two months after treatment. The intensity and frequency of attacks of laryngospasm were assayed for 12 months after finishing treatment. After eradication, the symptoms completely disappeared in the patient.

**Comment**

In reported cases, an association between sleep-related laryngospasm and GER has been previously suspected. Here, we also present evidence to support this association.

Four things strongly suggested that H. pylori infection caused our patient's disorder. First, the importance of H. pylori is now moving from gastric diseases toward a number of extragastrintestinal disorders, such as ischemic heart disease, ischemic cerebrovascular disease, atherosclerosis, Raynaud’s phenomenon, and skin diseases. The persistent inflammatory response related to H. pylori infection may induce vascular disorders by means of immune-mediated release of substances. Second, symptoms entirely resolved after H. pylori was eradicated. Third, persistent infection may induce chronic inflammatory and immune responses that can cause lesions that are local or distant from the site of primary infection. Finally, there are no alternative explanations.

To the best of our knowledge, this is the first report about the relation of H. pylori infection and laryngospasm symptoms. Clinicians should be aware of this possibility when prescribing for laryngospasm symptoms for patients in climacteric.

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