An 83-year-old man with severe kyphosis and parkinsonism caused by cerebral infarction was admitted to our hospital with a history of episodes of chest discomfort. Endoscopic study revealed a long segment of Barrett’s mucosa with large hiatal hernia and a deep ulcer in the Barrett’s mucosa (● Fig. 1). No malignancy was proven by endoscopic biopsy of the ulcer. The patient was not receiving any drugs or chemicals. Thus, we diagnosed the ulcer as a “Barrett’s ulcer” [1]. Computed tomography showed that the thoracic esophagus was dorsally displaced and the stomach ventrally displaced by kyphosis of the spine. Despite overnight fasting, fluid was retained in the mid-esophagus, which corresponded to the location of the Barrett’s ulcer (● Fig. 2a). A barium examination revealed reflux from the stomach to the mid-esophagus around the location of the ulcer when the patient was semi-upright and supine (● Fig. 2b,c). The patient had to sleep in the supine position during the night because his ability to turn over in bed was restricted by the parkinsonism. No evidence of Helicobacter pylori infection was found in the serological test. Evidently, highly acidic reflux fluid might be retained for a long time in the mid-esophagus, where the ulcer developed.

The ulcer healed completely after treatment with omeprazole 20 mg/day for 2 months (● Fig. 3).

The onset mechanism of Barrett’s ulcer is thought to be peptic ulceration in Barrett’s esophagus due to gastroesophageal reflux [2]. It is suggested that kyphosis increases the severity of hiatal hernia and subsequently induces gastroesophageal reflux [3]. In our hospital all three patients...
with a nonmalignant ulcer in a Barrett’s segment had either kyphosis or scoliosis. This suggests that nonmalignant Barrett’s ulcers may be associated with vertebral deformities including kyphosis in Japanese patients.

**Competing interests:** None

Endoscopy_UCTN_Code_CCL_1AB_2AC_3AZ

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Endoscopy 2010; 42: E286–E287
© Georg Thieme Verlag KG Stuttgart · New York · ISSN 0013-726X

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