

A tablet of clopidogrel remaining in the lower esophagus after primary percutaneous coronary intervention for acute myocardial infarction

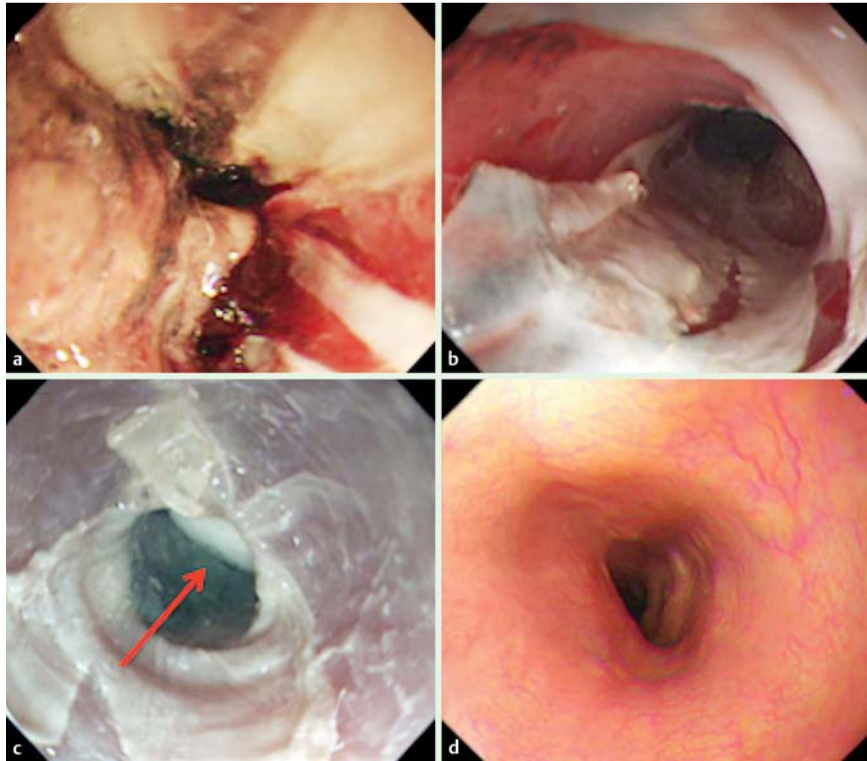


Fig. 1 a, b Gastroscopic images obtained after primary percutaneous coronary intervention (pPCI) in a 70-year-old woman with ST-segment elevation myocardial infarction (STEMI) show a thin, white coat of esophageal erosion. c A clopidogrel tablet (red arrow) remains in the esophagus. d Gastroscopic image obtained 1 week after pPCI shows healing of the epithelial membrane.

A 70-year-old woman with ST-segment elevation myocardial infarction (STEMI) underwent primary percutaneous coronary intervention (pPCI). She did not have a past history of either gastrointestinal disease or pemphigoid. Before the pPCI, she received dual antiplatelet therapy (a total of six tablets taken as a loading dose without much water) and maintained a supine position for several hours. The patient experienced back pain during the pPCI procedure, and blood testing showed a hemoglobin level of 10.0 g/dL. During gastroscopy after the pPCI, a thin white coat of esophageal erosion was noted (● Fig. 1 a, ● Fig. 1 b), and a clopidogrel tablet was seen remaining in the lower esophagus (● Fig. 1 c). The fiberoptic scope could not be advanced to the stomach because the erosion extended to the lower esophagus with narrowing. Neither a hematoma at the vascular access site nor

a retroperitoneal hematoma was observed by computed tomography, and no malignancy was diagnosed.

The antiplatelet therapy was discontinued, and the patient was treated with an intravenous high calorie diet and proton pump inhibitor for 1 week. A second gastroscopy showed healing of the esophageal epithelial membrane (● Fig. 1 d) and no evidence of gastric ulcer. No stent thrombosis occurred during the recovery period, and she resumed her daily intake of 100 mg of aspirin.

It is not clear if the drug itself caused the widespread exfoliation of the lower esophageal mucosa seen in this patient. Another possibility is that the exfoliation resulted from ischemic mucosal damage or “stress” associated with STEMI, and the drug exacerbated the problem to some extent. Because clopidogrel is a prodrug and exerts its antiplatelet effect after ab-

sorption, the clopidogrel tablet itself probably stimulated the esophageal mucosa in the present case [1–3].

To avoid such undesirable situations, a patient with STEMI who is undergoing pPCI should take an adequate amount of water with the pills and should maintain a right lateral decubitus position for a few minutes in the emergency room before going to the catheter laboratory.

Endoscopy_UCTN_Code_CCL_1AB_2AC_3AD

Competing interests: None

Tomomi Koizumi, Shigeyuki Nishimura

Department of Cardiovascular Medicine, Saitama International Medical Center, Saitama Medical University, Hidaka, Japan

Acknowledgement

The authors wish to thank Heidi N. Bonneau, RN, MS, CCA, and Hideaki Kaneda, MD, PhD, for their expert review of this report.

References

- 1 Zografos GN, Georgiadou D, Thomas D et al. Drug-induced esophagitis. *Dis Esophagus* 2009; 22: 633–637
- 2 Cummin AR, Hangartner JR. Oesophago-atrial fistula: a side effect of tetracycline? *J R Soc Med* 1990; 83: 745–746
- 3 Yamaoka K, Takenawa H, Tajiri K et al. A case of esophageal perforation due to a pill-induced ulcer successfully treated with conservative measures. *Am J Gastroenterol* 1996; 91: 1044–1045

Bibliography

DOI <http://dx.doi.org/10.1055/s-0034-1393231>
Endoscopy 2015; 47: E508
© Georg Thieme Verlag KG
Stuttgart · New York
ISSN 0013-726X

Corresponding author

Tomomi Koizumi, MD
Division of Cardiovascular Medicine
Saitama International Medical Center
Saitama Medical University
1397-1 Yamane
Hidaka
Saitama 350-1298
Japan
Fax: +81- 42-984-4741
tkoizumi@saitama-med.ac.jp