A case of Clindamycin-induced aphagia

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To the editor

An 80-year old male was transferred by his general practitioner because of sudden total aphagia. In his medical history we note a laryngectomy and thyroidectomy followed by adjuvant radiotherapy for a squamous-cell carcinoma of the larynx and vocal cords with invasion of the supraglottis, subglottis, thyroid cartilage, surrounding muscles and right thyroid lobe (pT4aNxG2L1V0Pn0R0).

Three days ago, he woke up with an inflamed and painful right hallux. The next day he presented to his general practitioner who suspected acute gout, for which she prescribed colchicine. Because of an ingrown toenail, his general practitioner added clindamycin so as not to miss an erysipelas. The pain in his toe quickly subsided, but an hour after ingesting his second pill of clindamycin he developed sudden painless swelling in his throat with complete aphagia to both solids and liquids.

Esophagogastroduodenoscopy showed a severe circular ulceration with swelling of the esophageal mucosa causing a near total stenosis, non-passable by endoscopy (figures 1-2). He was hospitalized to receive supportive therapy with intravenous fluids and parenteral nutrition, and was started on a proton-pump inhibitor. He remarked a slow decline of the globus sensation and after three to four days, he could once again slowly drink minimal amounts of fluids. One week later he underwent a new esophagogastroduodenoscopy which showed a decreased swelling of the esophagus and a partial healing of the circular ulcerations. As there still was an important stenosis of the esophagus, he underwent an esophageal dilatation to 11mm. The next day he was able to swallow adapted solid food, after which he was discharged from our hospital. After two sessions of balloon dilatation the patient could resume a normal food intake.

Medication-induced esophagitis is a rare although underestimated adverse reaction to certain drugs, and is thought to affect around 3.9/100 000 people per year. Antibiotics account for more than 60% of all reported cases. (1) Predisposing factors typically involve prolonged contact with the esophageal mucosa: taking medication in a recumbent position (e.g. in bed), without fluid, having a decreased production of saliva, pre-existing esophageal disorders. (2) The exact physiopathological mechanism of clindamycin-induced esophagitis is still under speculation. Whereas the most common culprits of medication-induced esophagitis, doxycycline and tetracycline, produce a pH below 3.0 when dissolved (which is thought to directly harm the esophageal mucosa), clindamycin does not alter the pH. (3)

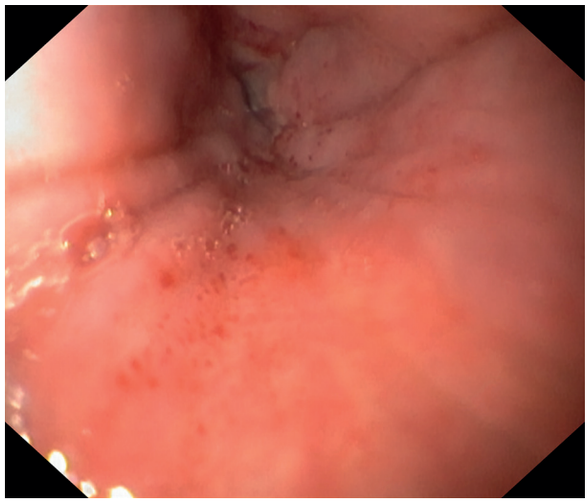

Fig. 1. — Esophagogastroduodenoscopy, viewing from the top of the esophagus.

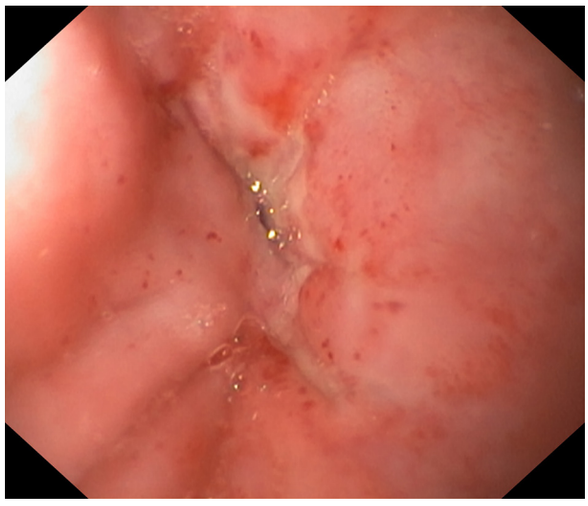

Fig. 2. — Esophagogastroduodenoscopy, the near-total stenosis up front.

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In literature, practically all cases of clindamycin-induced esophagitis heal without intervention within a few days. In our case however, clindamycin seems to have caused a severe reaction with circular ulceration and swelling of the esophageal wall leading to a painless globus sensation and severe aphagia. To our knowledge, this is the first described case of esophageal swelling leading to complete aphagia caused by any medication.

We suppose that his medical history of laryngectomy and thyroidectomy followed by adjuvant radiotherapy caused a pre-existing stricture of the esophagus. This presumed stricture would have caused a considerable prolonged contact of clindamycin with the esophageal mucosa. This would explain the severe and exudative reaction. Afterwards our patient confirmed that he already had more difficulty swallowing during the previous years.

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