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Infection with SARS-CoV-2 as potential achalasia trigger

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Achalasia is a chronic esophageal motility disorder with an estimated annual incidence of 1-2 cases per 100,000 people[1], characterized by incomplete relaxation of the lower esophageal sphincter and abnormal peristaltism. The condition results in dysphagia, regurgitation and chest pain complaints. The etiology of idiopathic achalasia remains unknown. From a pathophysiological perspective, it is caused by loss of inhibitory neurons at the esophageal myenteric plexus. Some viruses, including herpes simplex, human papillomavirus and measles virus, have been reported as triggers as viral infection may induce an aberrant immune response, which under appropriate genetic and environmental conditions would lead to loss of esophageal neurons[2].

We report the case of 66-year-old male with an uneventful history who was admitted for progressive dysphagia leading to complete solid and liquid intolerance over two months. He reported the condition started after mild infection with SARS-CoV-2, which might be causally related or a coincidental finding. Initially, a gastroduodenal barium study suggested achalasia as then did oral endoscopy, which found no mucosal lesions in a esophagus lacking motility and with retained food remnants. The diagnosis was eventually confirmed by high-resolution manometry, which found aperistaltic waves with panesophageal pressurization compatible with type-II achalasia (Figure 1). Lower esophageal sphincter motility could not be assessed because of inability of the probe to pass through. Therapeutic measures first included endoscopic treatment such as botulinum toxin injection and pneumatic balloon injection to avert greater invasiveness while expecting potential regression. Since the patient relapsed after both these techniques, and his performance status was good, he eventually underwent Heller's myotomy surgery with a good clinical outcome.

Within the hypothetical viral etiology of idiopathic achalasia, SARS-CoV-2 might be thought of as a potential triggering agent. Other case reports in the literature seemingly support this[3,4]. In addition, this virus has been reported as a potential

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cause of worsening of immune-mediated gastrointestinal disorders, including gastroparesis[5]. According to our experience, we suggest that, while the viral infection is transient, once neurons are lost the resulting motor disorder is hardly reversible, hence the therapy option should be aimed at achieving the greatest clinical effectiveness according to each patient's health status.

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Figure 1. Postero-anterior and lateral chest x-rays following a gastroduodenal barium study, which show absence of passage through the cardia, a tapered narrowing of the distal end, and proximal dilation. In the lower figure, high-resolution manometry reveals aperistaltic waves with panesophageal pressurization, consistent with type-II achalasia.

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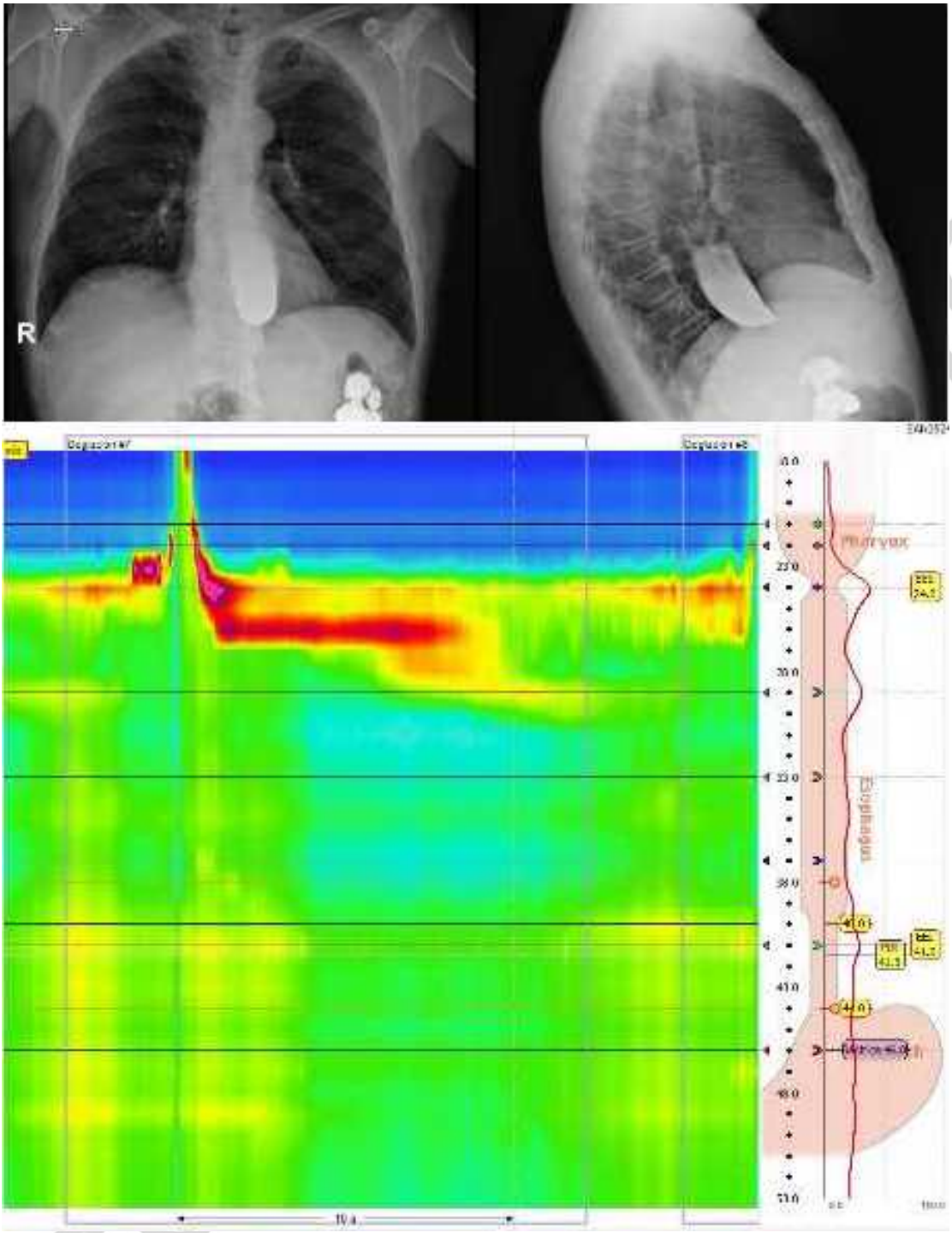

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