## CORRESPONDENCE

## JC Virus, *Helicobacter pylori*, and Oesophageal Achalasia: Preliminary Results from a Retrospective Case-Control Study

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JC virus (JCV) is a member of the polyomavirus family. It infects humans worldwide, and 90 % of the population carry antibodies to the virus by adult life [1]. The initial infection is asymptomatic, but it may become persistent. JCV DNA is frequently present in the upper and lower gastrointestinal tract of healthy adults [2, 3].

Several studies have suggested the involvement of certain DNA viruses in chronic gastrointestinal motility disorders such as chronic intestinal pseudo-obstruction [4–7]. Although a close causal relationship between neurotropic viruses and enteric neuromuscular lesion is not easily demonstrated, the identification of neurotropic viruses and/or molecular components associated with them may help to clarify their role in the highly severe gastrointestinal disease.

A1 **Electronic supplementary material** The online version of this A2 article (doi:10.1007/s10620-012-2485-9) contains supplementary

A3 material, which is available to authorized users.

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The aim of our study was to assess the prevalence of JC virus in a cohort of patients with oesophageal achalasia, comparing it with a cohort of healthy subjects, in order to evaluate whether JC virus could be play a role in this disease; we evaluated also the relationship between JC virus and *Helicobacter pylori* (HP).

In the cohort of patients with achalasia, 12 out of 15 (80 %) had a positive JC-PCR, while in the cohort of healthy controls, ten out of 15 (66.7 %) had a positive JC-PCR (chi-square 0.68, odds ratio 2, p = 0.409). In the cohort of patients with achalasia, 13 out of 15 (86.7 %) had a positive hystologic HP test, while in the cohort of healthy controls, eight out of 15 (53.3 %) had a positive hystologic HP test. Curiously, in the entire cohort of patients, JC virus infection proved to be protective against HP infection (chi-square 11.35, odds ratio 0.09, p = 0.0008). The prevalence of HP and JC virus infection in the entire cohort is shown in Table 1.

Because of the known neuropathic capability of JC virus, and its frequent presence in the upper gastrointestinal tract, as well as in the gut, we proposed that JCV might be detectable in tissues of patients with oesophageal achalasia, and possibly be involved in the pathogenesis of this disease. However, in this study the difference between the prevalence of JC virus in achalasia patients and in healthy controls was not statistically significant, probably due to the small number of cases and control subjects.

Few data are available about the relationship between JC virus and HP. Selgrad and coworkers, in their case—control study, showed that JC virus is present in HP induced gastritis and gastric cancer [9]. By contrast, the findings of our case—control study show that JC virus could be a protective factor against HP infection, though the biological and pathophysiological reasons for this microbiological interference are not yet known.

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**Table 1** Distribution of JC virus and HP infection in the entire cohort of patients

Parameter	HP positive	HP negative
JC virus positive	5	15
JC virus negative	6	4

HP Helicobacter pylori

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In conclusion, despite the retrospective nature of our study and the small number of patients involved, we suggest that actually the basis to consider JC virus as an etiologic factor in pathogenesis of oesophageal achalasia is not significant. Curiously, JC virus could be a protective factor against HP infection, though the reasons of this antagonism are not fully elucidated. Further prospective case—control studies, with a larger number of patients, are needed to clarify the possible etiopathogenetic role of JC virus in oesophageal achalasia and the relationship between JC virus and HP.

The methods for the letter are available in supplementary material.

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