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Outcome evaluation of clarithromycin, metronidazole and lansoprazole regimens in *Helicobacter pylori* positive or negative children with resistant otitis media with effusion

Dear Editors,

I came across a very interesting study published in your prestigious journal titled 'Outcome evaluation of clarithromycin, metronidazole and lansoprazole regimens in *Helicobacter pylori* positive or negative children with resistant otitis media with effusion' by Mel-Hennawi *et al*.[1] The authors have conducted a very meticulous study and I would like to appreciate their effort; however, I have some concerns regarding the methodology and conclusion, which I would like to express through your esteemed journal.

The role of *H pylori* in the pathogenesis of otitis media with effusion (OME) is a matter of debate, with studies both in favour of it[2] and against it.[3] The aspirated fluid from the middle ear has been used for the detection of *H pylori* in most of the studies as it is confirmatory for the presence of the bacteria in the middle ear. However, in the present study only the stool antigen has been used. The detection of *H pylori* in stool samples, although cheap and non-invasive, does not confirm the presence of the bacteria in the middle ear. In addition, the prevalence of *H pylori* in children ranges from 15 to 70 per cent;[4] therefore, the detection of *H pylori* in stool samples is non-specific and may not be significant.

A few studies have explored the role of gastroesophageal reflux in the pathogenesis of otitis media.[5] In such a scenario, it is possible that the children in the present study could have benefitted primarily from a reduction in the gastroesophageal reflux by lansoprazole. This needs to be proven in future prospective trials. If proven, then clarithromycin and metronidazole can be omitted, thereby reducing the cost of treatment and avoiding the side effects of these antibiotics.

A further concern is the rising incidence of development of clarithromycin resistance in *H pylori*. Therefore, misuse of clarithromycin should be prevented at all costs, and its use should be based on clear scientific evidence. The evidence for the use of clarithromycin in OME is still unclear. Future randomised, controlled trials with larger sample populations are warranted before a conclusion can be drawn.

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Authors' reply

Dear Editors,

We are so glad to hear of the interest in our work titled 'Outcome evaluation of clarithromycin, metronidazole and lansoprazole regimens in *Helicobacter pylori* positive or negative children with resistant otitis media with effusion'. In addition, we appreciate the focused interest from Dr Satvinder Singh Bakshi on our published papers and the enquiry comments.

I would like to reply to the comments made in the above-mentioned letter.

First comment

This was 'The role of *H pylori* in the pathogenesis of otitis media with effusion (OME) is a matter of debate, with studies both in favour of it and against it'. Dr Bakshi mentioned that some studies were against the role of *H pylori* in OME and used a study by Sudhoff e*t al*.[1] to support this. Sudhoff and colleagues found little evidence for the existence of *H pylori* associated OME, but did not deny its role, nor were they against it (as Dr Bakshi pertained in his letter). Moreover, Sudhoff *et al*. concluded by stating that further research is needed in order to delineate the presence of *H pylori* and its role in the pathogenesis of OME.[1]

In a recent study by Saki *et al*. heavy colonisation of *H pylori* was detected by polymerase chain reaction in adenoid tissue and the middle ear for OME cases.[2] The authors concluded that *H pylori* had a role in the pathogenesis of OME and that the condition was resistant to medical treatment. In addition, Bai *et al*., in a 2012 study conducted in China on OME patients, confirmed that the middle-ear effusion was *H pylori* positive, as established by both culture and urease tests.[3] Their findings suggest that *H pylori* is strongly involved in the aetiology of OME. Yilmaz *et al*. found significantly increased colonisation by *H pylori* of the middle ear, and tonsillar and adenoid tissue in patients with OME using culture and polymerase chain reaction analyses, and mentioned *H pylori* involvement in the pathogenesis of OME.[4]

There are many published papers on the involvement of *H pylori* in the pathogenesis of OME, that use many different investigative methods (culture, polymerase chain reaction, urease test, etc.), which support our work and results, with no published literature to support the comment against the role of *H pylori*.

Second comment

The author commented that 'in the present study only the stool antigen has been used'. As part of our study, a pilot

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