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Are our endoscopy patients at risk for pyogenic liver abscess?

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Abstract

This is an editorial comment on a recent publication reporting an increased rate of pyogenic liver abscesses (PLAs) after upper gastrointestinal panendoscopy. Its aim is to critically highlight the findings, limitations and

potential clinical implications of this study. Issues of the mucosal barrier, the microbial flora, administration of antibiotics and underlying diseases are discussed. The probability of PLAs after endoscopies is not exactly known and the length of the "incubation period" remains unclear, but a possible causality should already suffice to make us think how to avoid them. Especially in patients with risk factors such as diabetes mellitus, end-stage renal disease, liver cirrhosis, biliary tract infection, and malignancies, the potential risk for PLAs should be considered. Unnecessary insufflation during endoscopy (causing mucosal stretching and microscopic tears) as well as mucosal damage (by direct abrasion with the scope) should be avoided in order to limit the invasiveness of the procedure as much as possible. And, in everyday routine, it should be kept in mind that in patients after endoscopy, especially in those with a breach of the mucosal barrier and significant comorbidities, PLAs can potentially develop and require timely administration of antibiotics as well as further diagnostic and therapeutic steps.

Key words: Endoscopy; Pyogenic liver abscess; Mucosal barrier; Gastrointestinal microbial flora; Comorbidities

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Core tip: An increased rate of pyogenic liver abscesses after upper gastrointestinal endoscopy has been reported in a recent publication, leaving clinicians in some kind of predicament. Are we really exposing our endoscopy patients to a considerable danger? In an invited editorial comment on this study, the background, limitations and potential clinical implications of the findings are discussed.

Cerwenka H, Schemmer P. Are our endoscopy patients at risk for pyogenic liver abscess? *World J Gastroenterol* 2017; 23(30): 5457-5459 Available from: URL: <http://www.wjgnet.com>

INTRODUCTION

In the study published by Tsai *et al*^[1] on 2135 patients with a first diagnosis of pyogenic liver abscess (PLA) and 10675 patients without PLA selected as reference controls, a higher rate of PLAs was found in those who had recently undergone an upper gastrointestinal panendoscopy. The authors concluded that clinical physicians should not ignore this. In spite of some limitations, especially related to the use of retrospective registry data, this study leaves us in some kind of predicament. Are we really exposing our endoscopy patients to a considerable danger?

MUCOSAL BARRIER

Theoretically, every endoscopy may cause a breach of the mucosal barrier and post-endoscopy bacteremia has indeed been reported^[2-4]. However, PLA is known not to develop very easily and to affect predominantly patients with comorbidities and a weakened immune system^[5,6]. In addition to the patient's condition, the extent of mucosal damage could also play a role. Are microscopic leaks relevant in immunocompetent persons? The study by Tsai *et al*^[1] does not comprise the diagnoses made at the endoscopies. It would be important to know how many patients had pathologies leading to an obvious disruption of the continuity of the mucosal barrier like ulcers or erosive inflammation. Another point of easy entrance for bacteria causing PLA (in this case *via* the biliary route) would be the papilla of Vater after endoscopic papillotomy. Patients with recent ERCP were excluded in this study, but the authors provide no information on papillotomies made longer ago.

MICROBIAL FLORA

The mucosal surface of the gastrointestinal tract is colonized by 400 different bacterial species and subspecies^[7]. In general, pathogens are part of a transient flora emerging under abnormal conditions, but some autochthonous germs may also become pathogenic under certain circumstances. The stomach, the duodenum and the jejunum contain only low numbers of microorganisms (103 to 104 bacteria/mL), notably acid-tolerant lactobacilli and streptococci, whereas the colon is the main site of microbial colonization^[7]. It is, therefore, surprising, that, in this study, the risk of developing PLAs was higher for patients with upper gastrointestinal endoscopies than for those with colonoscopies. Likewise, a higher and not a lower rate of PLAs would have been expected

in those with interventional endoscopic procedures associated with a more severe mucosal damage. As for the colonoscopies, the authors argue that the colon is further away from the portal venous and lymphatic circulations to the liver than the esophagus, stomach, and duodenum, and that it has a potent mesenteric lymphatic defense system^[1]. Nevertheless, it is well known, that underlying pathologies in the whole gastrointestinal tract can be found in patients with manifest cryptogenic PLAs^[8,9]. In terms of the interventional procedures, a possible explanation given, but not proven, by the authors is that more patients in these groups may have had antimicrobial treatment^[1].

ANTIBIOTICS

In fact, the use of antibiotics is an essential point. Although they are not given routinely as a prophylaxis for endoscopy patients, some of the study patients will have been on current antibiotic regimens for various reasons. In general, PLAs respond well to the administration of antibiotics and the microbial spectrum of these abscesses is covered by many of them. For a clearer picture on the pathogenesis of the PLAs and of the role of antibiotics in this study, it would also be necessary to know the results of microbial cultures of the aspirates from the abscess cavities as well as details on the use of antimicrobial substances.

COINCIDENCES

Another issue requiring clarification is if some of the endoscopy patients already had concomitant incipient PLAs not yet diagnosed at the time of the examination. The presenting symptoms of PLAs may be vague and indeed, unspecific pain associated with undiagnosed PLA could even have been the motive for performing the endoscopy. In one of the study patients, the PLA was diagnosed as early as one day after the panendoscopy^[1]. Here a coincidence seems most likely. The length of the "incubation time" for PLA after panendoscopy remains an unanswered question.

CONSEQUENCES

In conclusion, we do not exactly know the probability of PLAs after endoscopies, but a possible causality should already suffice to make us think how to avoid them. What can be done? It will not be advisable to renounce at endoscopies in patients requiring them for therapeutic, diagnostic or prophylactic purposes nor will it be reasonable to make these patients afraid. It will also still be indicated to do endoscopies in order to identify underlying diseases in patients with manifest cryptogenic PLAs^[10] without fearing to aggravate the abscesses. However, especially in patients with risk factors such as diabetes mellitus, end-stage renal disease, liver cirrhosis, biliary tract infection, and

malignancies, the potential risk for PLAs should be considered. Strict disinfection processes are a matter of course. Unnecessary insufflation during endoscopy (causing mucosal stretching and microscopic tears) as well as mucosal damage (by direct abrasion with the scope) should be avoided in order to limit the invasiveness of the procedure as much as possible. And, in everyday routine, it should be kept in mind that in patients after endoscopy, especially in those with a breach of the mucosal barrier and significant comorbidities, PLAs can potentially develop and require timely administration of antibiotics as well as further diagnostic and therapeutic steps.

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