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ESPS PEER REVIEW REPORT

Name of journal: World Journal of Gastroenterology
ESPS manuscript NO: 14670
Title: Microscopic Enteritis: The Bucharest Consensus
Reviewer code: 00008491
Science editor: Yuan Qi
Date sent for review: 2014-10-27 11:47
Date reviewed: 2014-11-18 05:23

CLASSIFICATION	LANGUAGE EVALUATION	RECOMMENDATION	CONCLUSION
[ ] Grade A: Excellent	[Y] Grade A: Priority publishing	Google Search:	[ ] Accept
[ ] Grade B: Very good	[ ] Grade B: Minor language polishing	[ ] Existing	[ ] High priority for publication
[Y] Grade C: Good	[ ] Grade C: A great deal of language polishing	[ ] No records	
[ ] Grade D: Fair		BPG Search:	[ ] Rejection
[ ] Grade E: Poor	[ ] Grade D: Rejected	[ ] Existing	[ ] Minor revision
		[ ] No records	[Y] Major revision

COMMENTS TO AUTHORS

GENERAL COMMENT The paper is signed by a very authoritative panel and it is an interesting work which attempts to define the histology picture of the microscopic enteritis (ME) and the clinical conditions which can be associated to it. However, I have some concerns; the main of these is that in this work the ME is considered as an "independent" clinical condition. In particular, in the paragraph regarding the "Diagnosis", the Authors seem to suggest a series of investigations to clarify the cause of ME. In the clinical practice, on the contrary, the histology evaluation of the duodenum (and the eventual finding of ME) is part of the investigations performed in the suspect of several diseases. In this way, on my opinion, the text is difficult to read and to understand. A similar comment is valid for the "Treatment". I would suggest to rewrite the paper, considering the different diseases which can cause ME, as the Authors made until page 9. Other useful considerations now suggested in the following para (Diagnosis and Treatment) could be included in the respective previous para (CD, NCGS, Infection, Drugs, etc) SPECIFIC POINTS - Page 4 4th para: "It has become clear that "non-specific" referred to multiple aetiological conditions, other than gluten sensitivity". I think that the Authors refer to Celiac Disease and not "gluten sensitivity". In general, to avoid confusion I would suggest to use the term "celiac disease" instead of "gluten sensitivity", throughout the text. - Page 5 "Definition". "ME is a histopathological condition that affects the small bowel and causes microscopic and sub microscopic changes....." I would suggest to change "cause" with "is characterised by" - Label of the figures is inaccurate

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ESPS PEER REVIEW REPORT

Name of journal: World Journal of Gastroenterology
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CLASSIFICATION	LANGUAGE EVALUATION	RECOMMENDATION	CONCLUSION
[ ] Grade A: Excellent	[ ] Grade A: Priority publishing	Google Search:	[ ] Accept
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[ ] Grade D: Fair	[ ] Grade D: Rejected	BPG Search:	[ ] Rejection
[ ] Grade E: Poor		[ ] Existing	[Y] Minor revision
		[ ] No records	[ ] Major revision

COMMENTS TO AUTHORS

Dear Authors, Good paper, interesting . comments are in uppercase inserted located after your text (please see comments inserted in your document in attachment) ….. The nature and timing of reactions following oral or epicutaneous gluten challenge is crucial for diagnosis. NCGS is a re-discovered condition applicable to patients who fail to satisfy diagnostic criteria for CD or gluten/wheat allergy but benefit from GFD (52, 53). BUT THE BENEFIT FROM GFD MAY BE NOT DUE A SO-CALLED NCGS BUT TO THE REDUCTION OF 'FODMAP' (FERMENTISCIBLE OLIGOSACCHARIDE, DISACCHARIDE, MONOSACCHARIDE AND POLYOLS)++ Furthermore H. pylori has been highlighted as the most frequent aetiology in patients with LD and abdominal pain (58, 59). Santoloria et al found small bowel bacterial overgrowth caused LD in 22% of patients (58-60). see table 2 WE'D LIKE TO KNOWN WHAT IS THE FREQUENCY OF ME RECOVERY AFTER HELICOBACTER PYLORI ERADICATION. The increased bacterial burden on the small intestine may cause ME by depleting of essential nutrients necessary for normal mucosal function and the subsequent generation of toxic metabolic products. Tropical sprue (61), post-infective malabsorption and parasite infections including giardiasis and threadworm represent less prevalent infectious causes of ME (2, 62, 63). WE'D LIKE TO KNOWN AS WELL WHAT IS THE FREQUENCY OF ME RECOVERY AFTER TREATMENT OF THESE INFECTIONS. Drug therapy ….More recently Olmesartan has been implicated in a severe sprue like enteropathy (65, 66). WE'D LIKE TO KNOWN THE CAUSALITY OF OLMESARTAN, OR OTHER SARTAN, IN THE

NEXT SECTIONS, THE CAUSALITY OF ACE INHITORS IS POINTED OUT: IS THERE A CONFUSION ? MAY I ADD SARTANS ARE ANGIOTENSIN II RECEPTOR ANTAGONIST AND NOT ACE INHIBITOR ? AND THE SAME QUESTION: WHAT IS THE FREQUENCY OF ME RECOVERY AFTER NSAID STOP?. Systemic inflammatory conditions Systemic inflammatory conditions implicated in ME include sarcoidosis and inflammatory bowel disease (3). Vidali et al identified microscopic duodenitis in 26.6% of patients with ulcerative colitis and significantly increased CD3+ and CD8+ IELs and lamina propria mononuclear cells compared with disease free controls (67). THE AUTHORS DO NOT COMMENT THESE STRANGE DATA: ENTERITIS ASSOCIATED WITH ULCéRATIVE COLITIS, WITHOUT REFERENCE ABOUT CRONH'S DISEASE! Diagnosis The term ME can be proposed in cases of Marsh 0-II mucosal changes with clinical, serological, genetic and histological data unsupportive for a specific aetiology (agreement: 100%) CONFUSING SECTION : WITH THE PREVIOUS AND FOLLOWING SECTIONS, WE CAN UNDERSTAND THAT ME CAN BE OBSERVED DURING MISCELLANEOUS CLINICAL DISORDERS (THE 'UNDERLYING AETIOLOGY') LEADING TO THE NEED OF AETIOLOGICAL INVESTIGATIONS. HERE, AT THE OPPOSITE, THESE CAUSES EXCLUDE THE DIAGNOSIS OF ME. OF COURSE, THIS DOES NOT CHANGE THE NEED OF THESE INVESTIGATIONS, BUT ONLY THE DEFINITION OF ME. Current and recently ceased medications should be reviewed, including NSAIDs ACE inhibitors and 'over the counter' medications. SEE ABOVE COMMENT ON ACEI AND SARTANS …. The effect of current or previous dietary modifications may provide useful diagnostic information. Symptomatic improvement with gluten or lactose free diet suggests underlying gluten intolerance. ? but very cautiously: see comment on FODMAP reduction. Liver function tests may demonstrate elevated transaminases suggesting a chronic inflammatory disease such as primary biliary cirrhosis or sclerosing cholangitis potentially with concomitant inflammatory bowel disease. ? NO. CHRONIC INFLAMMATORY DISEASE SUCH AS PRIMARY BILIARY CIRRHOSIS OR SCLEROSING CHOLANGITIS POTENTIALLY WITH CONCOMITANT INFLAMMATORY BOWEL DISEASE MAY BE SUGGESTED BY BIOLOGICAL CHOLESTASIS NOT BY ELEVATED TRANSAMINASES, WHICH ARE SUGGESTIVE OF CD. ….Vande-Voort et al found that 38% of patients with LD and non-HLA DQ2 or DQ8 genotype improved with initiation of GFD (79). In contras