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ESPS Peer-review Report
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CLASSIFICATION	LANGUAGE EVALUATION	RECOMMENDATION	CONCLUSION
[ ] Grade A (Excellent)	[Y] Grade A: Priority Publishing	Google Search:	[ ] Accept
[Y] Grade B (Very good)	[ ] Grade B: minor language polishing	[ ] Existed	[ ] High priority for publication
[ ] Grade C (Good)	[ ] Grade C: a great deal of language polishing	[ ] No records	[ ]Rejection
[ ] Grade D (Fair)	[ ] Grade D: rejected	BPG Search:	[Y] Minor revision
[ ] Grade E (Poor)		[ ] Existed	[ ] Major revision
		[ ] No records	

COMMENTS TO AUTHORS

I enjoyed this comphrensive and generally well presented paper exploring the links between vascualr thromboembolism and IBD. The authors have covered and lot of ground andhave presented their arguments and reviews clearly. There do not seem to be any major issues in the content, although the emphasis throughout could perhaps be a bit less didactic and explain where genuine uncertaintly lies. Specific points: 1. Thoughout the paper there is a lot of emphasis on odds ratios and relative risk, whilst this is fine for epidemiological studies, this is difficult for the clinical to translate into risk for the patient. What are the absolute risks of DVT for hospilatised/ambulant IBD patient? 2. Relative risks are only useful when the comparision group is well defined. It seems clear that IBD does increase the risk of TE comapared to controls but how does the risk compare to other, perhaps more comparable inflammatory conditons (divertivulitis or pancreatitis perhaps?). The authors have provided some data on the comparison with ceoliac disease but in terms of inflammatory burdon, these are very different diseases. 3. The authors have provided a fairly convincing link between vascular thrombosis and exacerbation of pathogenesis in IBD, except they have have much skimmed over the negative results obtained in the trials of anticoagulation for the treatment of active IBD, these argues against thrombosis being integral to the pathogensis iof IBD and deserve further discussion. 4. Whilst many guidelines are quotes discussing prophylactive anticoagulation in IBD inpatients: what is the evidence that this actually works? 5. On page 14, there is a whole paragrpah expanding on the possuble pathway of IBD pathogenesis: whilst this is interesting conjecture, the many statements in this section do need appropriate referencing. This is in contrast to the overrefenceing in other areas. For instance, it does seem unnecessary to cite 6 different references about prophylaxis in IBD patients

(also page 14). 6. The authors cites that managment of TE in IBD is challenging (page 14) and the present no data showing this is any more challenging than managing TE in any other group of patients and in fact present a completely standard managment pathway. 7. Page 16,line 11. Data are plural, it should be there are no data 8. Page 16 - The discussion on the merits of standard versus higher doses of heparins for prophyaxis is important but difficult to interpret given the different dose regimens of the different LMW heparins and this dose of 4000 iu/day needs to be placed in the context of the different avaialble drugs. 9. Page 17 - it seems very draconian to say that managment of cardiovascular risk factors always requires consultaation with a specialist: this is standard internal medicine or primary care and surely should be within the remit of most competent physicans and including the IBD physicians. This commemnt may well relate more to specific health care systems but is not generalisable.. Similalrly, I am sure than in many health care systems, thromboembolism in IBD patients is managed absolutely safely and appropriate without any recourse to either haemoatoogy or interventional radiology. Again this is a health service design (and/ or payment?) related issue and in no way can this be mandatory (page 17). 10. The authors do not once mention vitamin K antagonsist therapy specifically. Are there any data suggesting that these drugs are less reiable in patients with small bowel disease and /or diarrhoea? 11. Although I suspect data are very limited, a paper like this should really mention the new novel orally acting anticoagulants: are these approved and safe for the managment of TE in IBD? There is a theorectical increase risk in GI bleeding, if so how safe are these drugs? Should they be avoided at the moment? 12. The discussion on arterial thromboembiolism mentions mesenteric ischaemia: this is a very vague term. Do the authors mean acute me