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Symptomatic inflammatory bowel disease in remission – it could be irritable bowel syndrome

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The prevalence of irritable bowel syndrome (IBS) varies considerably according to the population under study and the definition used. However, a figure of 10–15% would seem to be a reasonable estimate[1]. IBS certainly does not appear to prevent the development of other diseases, thus it might be expected that 10–15% of patients with any disease would therefore suffer symptoms consistent with this diagnosis. Therefore, 10–15% of patients with conditions such as angina, arthritis or inflammatory bowel disease will suffer symptoms of IBS, although it is possible that, if the primary disease is sufficiently severe, it might mask the presence of IBS. The fact that any individual, irrespective of what is wrong with him/her, can suffer from IBS confirms that the approach where IBS is considered as a diagnosis of exclusion is entirely illogical.

Although post-dysenteric IBS (PD-IBS) was first described many years ago, this condition has recently been the centre of much attention. It appears to follow any form of dysenteric illness and is more common in females, after prolonged illness or when there is pre-existing psychological comorbidity. The subject has recently been comprehensively reviewed by Spiller[2]. Mucosal biopsies from the colon of patients with IBS are typically normal, but recently it has been shown that subtle inflammatory changes may be present, particularly in those with PD-IBS[3–5]. In addition, there has been a report suggesting that anti-inflammatory cytokines might be somewhat deficient in some IBS patients[6], and all this has led to the concept that inflammation may be important in the pathogenesis of some cases of IBS, especially when it follows gastrointestinal infection[7]. Whether the syndrome following infection is identical to other forms of IBS, or represents an IB(like)S form, of disorder remains to be determined[8].

Patients with inflammatory bowel disease (IBD) not uncommonly continue to complain of abdominal symptoms, despite apparently being in clinical remission. The symptoms are often reminiscent of IBS and pain is sometimes more of a problem than the disorder of bowel habit. Endoscopy is usually normal or

shows only minimal change; likewise other markers of activity such as erythrocyte sedimentation rate, C-reactive protein and white cell scanning are also normal or equivocal. Characteristically, these patients do not respond to steroids but these drugs are often continued with the result that patients become Cushingoid without any symptomatic benefit. All these factors raise the possibility that, under certain circumstances, IBD might be able to provoke a syndrome similar to that observed in PD-IBS.

Although the pathophysiology of IBS is incompletely understood, changes in visceral sensitivity[9] and motor activity[10] of the gastrointestinal tract are thought to be important. Thus it would be of interest to know more about these two phenomena in patients with IBD.

There seems little doubt that patients with active colitis have increased rectal sensitivity[11,12] and it was originally thought that patients with quiescent colitis appeared to have normal sensitivity[11,12]. However, there has been a recent report suggesting that patients with mild activity, somewhat surprisingly, have reduced sensitivity[13]. It is also interesting to note that, in animals, changes in visceral sensitivity associated with inflammation have been shown to persist once the inflammatory process has settled down[14]. Lastly, we have shown that experimentally induced diarrhoea can result in changes in rectal sensitivity in healthy female subjects[15], raising the possibility that the diarrhoea associated with IBD might lead to sensitization, irrespective of the inflammatory process. Motor activity has also been assessed in patients with quiescent ulcerative colitis and those with Crohn's disease, and appears to be affected. Furthermore, the changes do not necessarily appear to be confined to the inflamed segment of bowel[16–19]. Work in animals has revealed a similar situation to that observed with visceral sensitivity in that motor changes induced by inflammation appear to persist following its resolution[20,21].

It would therefore seem reasonable to assume that, based on epidemiological evidence, at least 10–15% of patients with IBD will suffer concurrent IBS. Furthermore, one could speculate that additional patients might suffer from a syndrome similar to that seen in PD-IBS. This would lead to the prediction that, if IBS symptoms were sought in an IBD population, one might expect a prevalence even higher than that observed in the general population.

Many years ago we looked at the prevalence of IBS in patients with ulcerative colitis in remission and found it to be 33%[22]. More recently, Simren and colleagues have undertaken a similar evaluation and found an identical figure[23]. In addition, they also looked at the prevalence of IBS symptomatology in Crohn's disease patients in remission and reported a figure of 57%[23]. It would therefore appear that the prediction that excessive numbers of IBD patients might suffer symptoms consistent with a diagnosis of IBS is correct, and this results from the combination of patients with pre-existing IBS and those in whom the problem has been initiated by inflammation, if indeed the two conditions are different.

In conclusion, IBS should be seriously considered as the cause of continuing symptoms in patients with IBD in remission. The recognition of this possibility is critical to management as it can lead to the avoidance of the inappropriate use of steroids, especially as these drugs have been shown to be unhelpful in PD-IBS[24]. We have previously shown that the presence of bloating, lethargy,

backache, nausea and urinary symptoms can help to substantiate the diagnosis of IBS[25]. Thus, the presence of these additional symptoms in an IBD patient who is in apparent mucosal remission may make the physician feel more confident in making the diagnosis of IBS.

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