The London Position Statement of the World Congress of Gastroenterology on Biological Therapy for IBD With the European Crohn ' s and Colitis Organisation: Safety

G Van Assche, JD Lewi, GR Lichtenstein, EV Loftus, Q Ouyang, J Panes, CA Siegel, WJ Sandborn, SPL Travis, and J-F Colombel.

Am J Gastroenterol 2011;106:1594-602

Serious infections

WCOG Statement 2.1
Risk of infection as a consequence of anti-TNF therapy
Although there is unequivocal evidence of an increased risk of serious infection among patients with rheumatoid arthritis treated with anti-TNF therapy, the data are less robust in CD.
The risk may be increased in patients on combination therapy with steroids and / or immunomodulators [EL 2a]
Treatment with anti-TNF therapy is associated with an increased relative risk of opportunistic infection and this risk is further increased by combination therapy with other immunomodulators [EL 2b]

WCOG Statement 2.3
Risk of tuberculosis as a consequence of anti-TNF therapy
The risk of reactivation of tuberculosis is signifi cantly increased in patients receiving anti-TNF therapy and all patients should undergo screening for latent tuberculosis before treatment. In those who have evidence of latent tuberculosis, treatment for latent tuberculosis should begin before starting anti-TNF therapy [EL 2b]

WCOG Statement 2.4
Risk of fungal infection as a consequence of anti-TNF therapy
Invasive fungal infections such as disseminated histoplasmosis, coccidioidomycosis, pneumocystis, disseminated sporotrichosis, and cryptococcosis have been reported following treatment with anti-TNF agents. There are no useful screening measures to identify high-risk individuals [EL 4]

Risk of malignancy

WCOG Statement 2.5
Overall risk of malignancy as a consequence of anti-TNF therapy
There is no consistent evidence of an increased overall risk of malignancy in patients with IBD treated with anti-TNF therapy [EL 2a]

WCOG Statement 2.6
Risk of non-Hodgkin ' s lymphoma as a consequence of anti-TNF therapy Patients whose treatment includes anti-TNF therapy have a higher risk of non-Hodgkin ' s lymphoma compared with the general population (OR: 3.2, 95 % CI: 1.5 – 6.9) [EL 2a], but it is unclear whether the risk is attributable to anti-TNF therapy, thiopurines, or the combination of both

WCOG Statement 2.7
Risk of hepatosplenic T-cell lymphoma as a consequence of anti-TNF therapy
Hepatosplenic T-cell lymphoma in patients with IBD is very rare, but young patients treated with a combination infl iximab and thiopurines appear to be predisposed [EL 4]. It is not

known whether all anti-TNF agents confer the same risk, or whether the risk is primarily attributable to the thiopurines in combination with anti-TNF therapy

Other toxicity related to biological therapy

WCOG Statement 2.8
Risk of immunogenicity and drug induced reactions to biological therapy
All monoclonal antibodies (anti-TNF agents and natalizumab) used to treat patients with IBD induce anti-drug antibodies [EL 1b]. Antibodies to infl iximab and to natalizumab are associated with acute infusion reactions [EL 3], whereas injection site reactions to certolizumab pegol or adalimumab have not been clearly linked to anti-drug antibodies

WCOG Statement 2.9
Risk of autoimmunity following biological therapy
De novo formation of anti-nuclear antibodies in patients with IBD occurs with all anti-TNF agents [EL 2b], but the clinical relevance is as yet unclear

WCOG Statement 2.10
Miscellaneous adverse events and biological therapy
Less frequent adverse events such as demyelinating disease, worsening of congestive heart failure, or eczematous skin lesions are class effects of anti-TNF therapies [EL 4]