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Is There Still a Role for Proactive Therapeutic Drug Monitoring (TDM) in Inflammatory Bowel Disease (IBD): A Review of the Literature

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Introduction

The management of biologic medications in inflammatory bowel disease (IBD) is complex due to the inter- and intra-individual variability in pharmacokinetics and pharmacodynamics. There exist important differences in drug uptake and metabolism depending on a variety of factors including dosing intervals, route of administration, gender, body weight, albumin levels, inflammation, immunogenicity, genetic variation and other concurrent therapies.[1] Males and individuals with higher body weight exhibit increased drug clearance, and certain biologics are more immunogenic than others. Moreover, the presence of a high inflammatory state, as demonstrated by elevated CRP levels and low albumin levels, also increase drug clearance and are associated with worse clinical outcomes.[2,3]

Therapeutic drug monitoring (TDM) can be useful in titrating certain biologic medications in IBD patients. By measuring drug levels and screening for antibody formation, TDM allows physicians to evaluate and optimize response to medications. Using these values, physicians can determine whether patients are sub-optimally dosed and can benefit from a reinduction or dose escalation, or whether these patients have begun developing immune responses to these medications.[4-7]

Reactive and Proactive TDM

There are 2 strategies for the use of TDM in clinical practice. The first strategy is reactive, whereby TDM is used in patients with active clinical, biochemical and endoscopic inflammation. This strategy allows physicians to understand whether active inflammation can be attributed to sub-therapeutic drug levels, anti-drug antibodies, or a pharmacodynamic treatment failure, where patients have optimal drug concentrations. There is a general consensus that a reactive TDM strategy is useful in patients on anti-tumor necrosis factor (anti-TNF) medications and that regular proactive monitoring of disease activity is the standard of care in IBD patients starting biologic medications[8] (**Figure 1a**). The American Gastroenterological Association's (AGA) clinical guidelines recommend reactive TDM in IBD patients on anti-TNF medications, though this recommendation is based off low-quality evidence.[9] Suggested therapeutic TDM thresholds in clinical practice (expert opinion) are outlined in **Table 1**, but clinical judgement should be exercised when using these thresholds.

The utility of a reactive strategy for patients on non-anti-TNF biologics is less clear. However, there is data demonstrating an exposure response relationship in patients on ustekinumab (UST)[10] and vedolizumab (VDZ) [10,11] and that dose escalation may be helpful for patients with a loss of response to help recapture remission.[12,13] Given

Drug	Suggested Trough Concentration (*ug/mL*)
Infliximab	≥10–15
Adalimumab	≥15
Golimumab	≥3

Table 1. Therapeutic TDM levels in anti-TNF agents; *courtesy of Davide De Marco, MD and Waqqas Afif, MD, M.Sc. (Epi), FRCPC.*

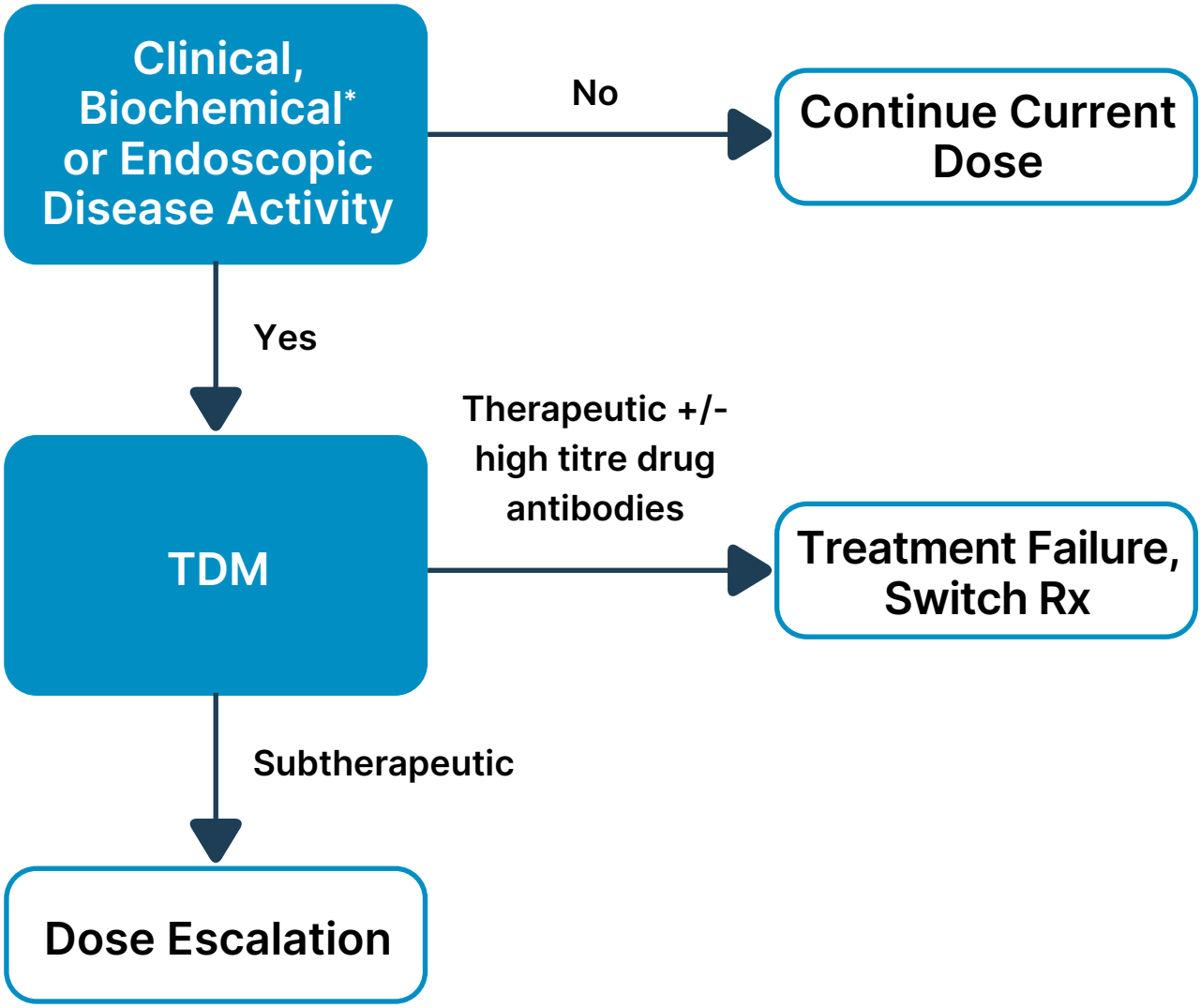

Figure 1a. Reactive TDM strategy; *courtesy of Davide De Marco, MD and Waqqas Afif, MD, M.Sc. (Epi), FRCPC.*

*Fcal q3–4, months in first year of Rx followed by yearly.

the low rates of immunogenicity of UST, VDZ, and newer Interleukin (IL)-23 biologics (<5%) and the absence of clear cut-offs where dose escalation would not be useful, the utility of reactive TDM for UST and VDZ remains unclear. Although TDM assays for UST and VDZ are readily available, their use in routine clinical care is likely not indicated given the available data. TDM is likely not necessary for oral small molecules such as Janus Kinase inhibitors (tofacitinib and upadicitinib) and the sphingosine-1-phosphatase receptor modulators (ozanimod and etrasimod). These oral molecules have stable pharmacokinetics and no immunogenicity, which makes their dose effect more predictable.[14-21]

On the other hand, a proactive strategy employs TDM during induction or maintenance, irrespective of the presence of symptoms or objective inflammation, to help guide decisions on drug dosing. It has been proposed that this strategy may allow drug serum concentrations to be optimized which may prevent suboptimal drug concentrations and antibody formation. It has also been proposed that this strategy may help prevent the development of active inflammation in patients who are in remission.[22,23] Most of the data for a proactive TDM approach pertains to patients on anti-TNF medications, which will be the main topic of discussion for this review.

Proactive TDM with Anti-TNF Medications

Anti-TNFs, such as infliximab (IFX), adalimumab (ADAL) and golimumab, are commonly used agents for the induction and maintenance of clinical remission in both CD and UC.[24-27] While these medications achieve 52-week clinical remission rates of 35–40% (26, 28, 29), 30% of patients are primary non-responders and an additional 50% will eventually experience a loss of response.[11,30] There is a well-documented exposure-response relationship amongst IBD patients on anti-TNF therapies.[31-35] ACT I/II trials in UC and the ACCENT I trials in CD respectively demonstrated post-induction IFX trough levels of >5.1 ug/mL and >3.4 ug/mL at 14 weeks, respectively, as predictive of prolonged clinical response.[29,34,36]

Anti-TNF therapy alone without concomitant use of an immunomodulator (monotherapy) is associated with an increased risk of immunogenicity. A recent study by Battat et al, which included 63,176 patients, found that 23.6% of patients who were treated with IFX and 19.6% of those treated with ADAL developed anti-drug antibodies.[37] Additionally, the PANTS study which included 955 IFX patients and 295 ADAL patients, found that suboptimal drug concentrations at week 14 predicted immunogenicity.[4] Given this data a proactive TDM approach can be considered to prevent treatment failure in the context of biologic monotherapy. This approach allows for dose optimization via increasing the dose or frequency during or immediately after induction.

The TAXIT randomized control trial (RCT), which included 263 IBD patients with stable responses to maintenance infliximab therapy, randomized patients to dose adjustments based on clinical features or on TDM levels (target trough 3–7 ug/mL). While no statistically significant difference was observed in achieving remission based on the 2 treatment strategies, a statistically significant decrease was noted in disease relapse among patients with concentration-based dosing compared to clinical dosing (7% vs 17% p=.018).[38] The TAILORIX RCT, which included 122 biologic naïve CD patients, showed no statistically significant difference between clinically driven dose escalation when compared to TDM driven dose escalation. However, it is important to note that most patients were not able to achieve therapeutic drug concentrations (sustained IFX level >3ug/mL in only 47% and 46% of the intensification groups and 60% in the control group).[39] Conversely, the PAILOT RCT, a randomized control trial of 78 children with CD found that patients who were randomized to the proactive group were more likely to achieve corticosteroid remission at 72 weeks compared to the reactive group (82% vs 48%, p=.002).[40]

A meta-analysis of 9 RCTs on proactive TDM found that there was no difference in the risk of failing to maintain clinical remission in patients who underwent proactive TDM when compared to conventional management (38% vs 42%, risk ratio [RR] 0.96; confidence interval [CI] 0.81–1.13).[23] Similarly, a meta-analysis by Sethi et al.,[22] consisting of 26 studies –9 of which were RCTs–sought to better understand the role of proactive TDM compared to standard of care (SOC) or reactive TDM. Amongst these studies, 8 explored proactive TDM for clinical remission or response. The proactive arm included 704 patients, and the SOC included 632 patients. No significant difference was noted between the two arms (RR 1.07, 95% CI 0.97–1.18, p=0.19).[22] However, sub analyses of studies comparing 793 proactive patients to 525 reactive patients, revealed that the proactive group was less likely to experience treatment failure (RR 0.46, 95% CI 0.21–0.98) and had lower hospitalization rates (RR 0.33, 95% CI 0.21–0.54). Conversely, there were no statistically significant differences between proactive and reactive TDM in the need for surgical interventions (RR 0.54, 95% CI 0.17–1.77, p=0.31). Ongoing prospective RCT's are further investigating the role of proactive TDM testing in patients with IBD.

Proactive TDM with Non Anti-TNF Medications

There is limited data available on the use of proactive TDM in patients receiving newer biologic medications such as UST, VDZ and newer IL-23 medications. A retrospective analysis of 436 Crohn's patients showed that induction and post-induction levels did not correspond to biochemical normalization.[41] Conversely, a recent single centre cohort of 94 IBD patients found those who underwent at least 1 proactive TDM were more likely to achieve drug persistence on multivariate analysis (hazard ratio [HR] 14.3, p<0.001).[42] The recently published ENTERPRET study showed that for patients with

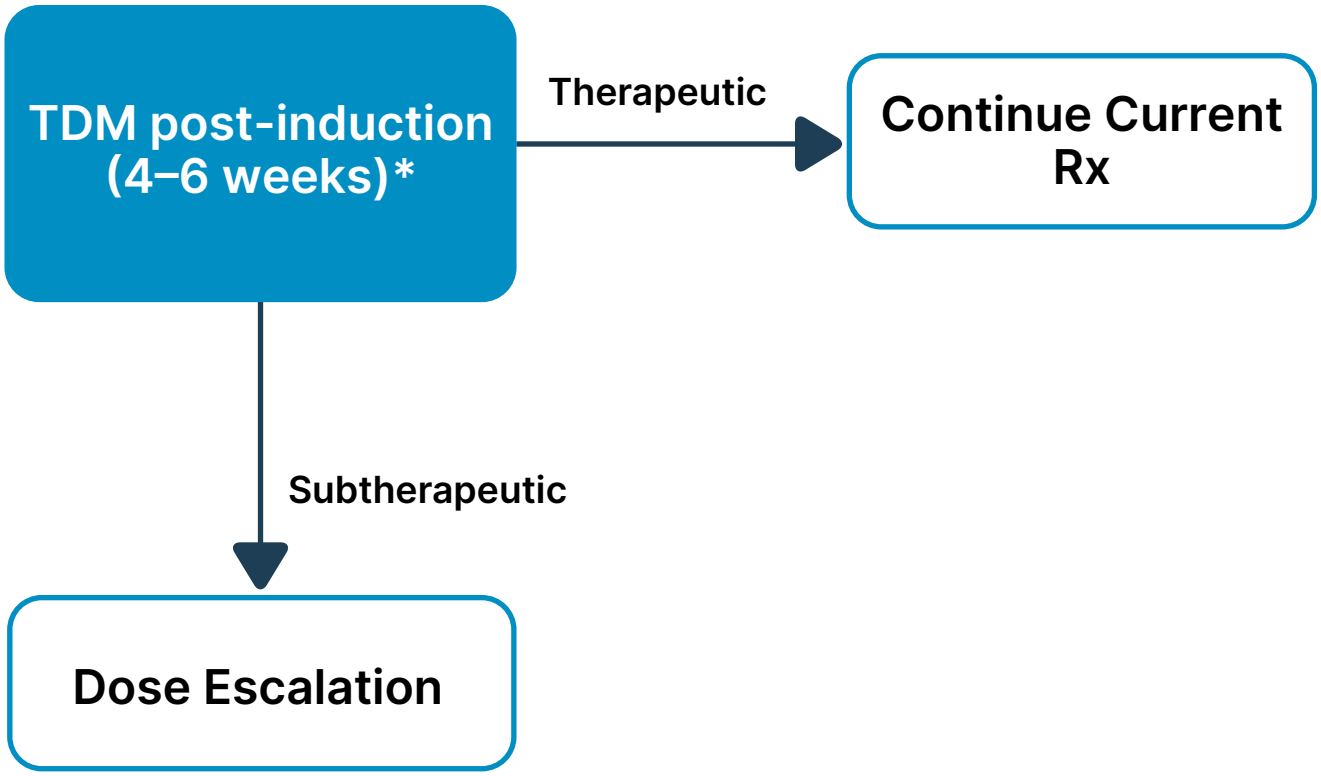

Figure 1b. Proactive TDM strategy; *courtesy of Davide De Marco, MD and Waqqas Afif, MD, M.Sc. (Epi), FRCPC.*

*Preferred in patients with low albumin, HLA-DQA1*05, large inflammatory burden, or multiple failed biologics.

early non-response and high drug clearance (low drug concentrations), dose optimization was of no benefit.[43] For UST, a recent single centre retrospective cohort study of 83 patients on SC UST found that those who underwent a single proactive TDM had higher drug persistence and fewer IBD-related hospitalizations.[44] Given these limited data, proactive VDZ and UST TDM cannot be recommended in routine clinical care.

Proactive TDM in the Clinical Setting

Based on the current evidence, routine proactive monitoring with anti-TNF medications cannot be recommended. But proactive monitoring is important in patients with increased clearance and/or an increased risk of immunogenicity (e.g., those with low albumin, high inflammatory burden, HLA-DQA1*05 haplotype, or anti-TNF exposed) to ensure adequate drug concentrations and treatment success[5,6,11,45] (**Figure 1b**). Proactive TDM may play an important role in these patient populations, as higher trough levels have been shown to decrease non-immunogenic treatment failure and may also lower the risk of neutralizing antibody formation.[46] We would recommend a proactive TDM assay in these patients be done at week 6 for IFX targeting levels of >10 ug/ml and at week 4 for ADAL, targeting concentrations >5 ug/ml.[39,40] Finally, while not within the scope of this review, clinicians can consider using proactive TDM with anti-TNF medications in the setting of dose de-escalation and when considering withdrawal of concurrent immunosuppression with methotrexate or thiopurines.

Conclusion

In conclusion, the use of TDM in the management of IBD provides a valuable tool for optimizing biologic therapy, specifically with anti-TNF medications. Reactive TDM is well-supported in guiding clinical decision-making during disease flares. While proactive TDM cannot be routinely recommended, it shows potential benefits in reducing immunogenicity and maintaining drug persistence in high risk patient populations on anti-TNF medications. The routine use of reactive or proactive TDM for non-anti-TNF biologics or small molecules is not supported at this time.

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