Multiple Sclerosis is a demyelinating degenerative disease that is rising in prevalence. Relapsing-Remitting Multiple Sclerosis is a type of MS in which patients experience an onset of neurological symptoms followed by complete or partial recovery. Provided is a review of the efficacy and safety of Teriflunomide for the treatment of Relapsing-Remitting Multiple Sclerosis using clinical studies, reviews, and comparative studies. The results show that Teriflunomide is one of the preferred medications prescribed for RMS because of its ability to significantly reduce relapse rates, disability progression, and its suppression of active inflammatory lesions. When considering the type and number of adverse events encountered with all treatments in addition to the efficacy Teriflunomide becomes the clear choice.

**Abstract**

The efficacy of Teriflunomide was determined through a number of clinical trials before its approval by the FDA. The Phase II trial entitled: Treatment of MS with Teriflunomide Monotherapy (2006) found that a daily 7 mg or 14 mg dose significantly reduced the number of cumulative active lesions. In addition, there was a significant reduction in the number of T1 gadolinium-enhancing lesions and T2 lesions. There was also a dose dependent trend toward fewer relapses (3). The TEMSO study was a phase III trial that found a significantly lower annualized relapse rate, reduced total lesion volume, and fewer gadolinium-enhanced lesions. However, only the 14 mg dosage group had a significantly reduced disability progression and hazard ratio (5). The phase III TOWER study found a significantly lower annualized relapse rate in both study groups. While only finding the 14 mg dose to reduce the risk of sustained accumulation of disability (4). TOPIC a phase III study found that Teriflunomide significantly increased the time to the primary endpoint (2). In Teriflunomide v Placebo with Glatiramer Acetate a phase III study it was found that the use of Teriflunomide significantly reduced the number and volume of lesions compared to Glatiramer Acetate (3). In addition, in Teriflunomide v Placebo added to Intravenous IFN beta another phase III trial found that there was an acceptable tolerability for these two medications to be used in conjunction. In addition, there were significantly fewer adverse events and a reduced number of T1 lesions in the Teriflunomide study group (3).

**Side Effects**

Teriflunomide is the Safest and Most Effective Diseases Modifying Therapy for the Treatment of Relapsing-Remitting Multiple Sclerosis

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**Efficacy**

The efficacy of Teriflunomide was determined through a number of clinical trials before its approval by the FDA. The Phase II trial entitled: Treatment of MS with Teriflunomide Monotherapy (2006) found that a daily 7 mg or 14 mg dose significantly reduced the number of cumulative active lesions. In addition, there was a significant reduction in the number of T1 gadolinium-enhancing lesions and T2 lesions. There was also a dose dependent trend toward fewer relapses (3). The TEMSO study was a phase III trial that found a significantly lower annualized relapse rate, reduced total lesion volume, and fewer gadolinium-enhanced lesions. However, only the 14 mg dosage group had a significantly reduced disability progression and hazard ratio (5). The phase III TOWER study found a significantly lower annualized relapse rate in both study groups. While only finding the 14 mg dose to reduce the risk of sustained accumulation of disability (4). TOPIC a phase III study found that Teriflunomide significantly increased the time to the primary endpoint (2). In Teriflunomide v Placebo with Glatiramer Acetate a phase III study it was found that the use of Teriflunomide significantly reduced the number and volume of lesions compared to Glatiramer Acetate (3). In addition, in Teriflunomide v Placebo added to Intravenous IFN beta another phase III trial found that there was an acceptable tolerability for these two medications to be used in conjunction. In addition, there were significantly fewer adverse events and a reduced number of T1 lesions in the Teriflunomide study group (3).

**Mechanism of Action**

- Selectively reversible inhibitor of dihydroorotate dehydrogenase
- Blocks de novo pyrimidine synthesis in rapidly proliferating cells
- Reduces activity of proliferating T and B lymphocytes
- Diminishes the inflammatory response
- Inhibits protein tyrosine kinases
- Alters cytokine production
- Modulates the expression of cell surface adhesion molecules
- This all contributes to the observed immunomodulatory effects (7)

**References**