محتوای زیر، بخشهایی از مقاله کاربر است که ویراستاری نیتیو شده است. جملات و کلمات قرمز رنگ، تغییراتیست که صورت گرفته است تا معنا و مفهوم روشن گردد.

Abstract

Background: Inflammation in myelinated portion of nervous system is the mainstay of multiple sclerosis (MS). Elevation of inflammatory markers such as procalcitonin, erythrocyte sedimentation rate (ESR) and high-sensitivity C-reactive protein (hs-CRP) is suspected to occur in MS patients compared to healthy control. However, their prognostic role and their relationship with the severity of MS clinical symptoms and MRI evidences are remained unnoticed in the literature. Hence, we aimed to evaluate the serum level of inflammatory markers in the acute attack of MS patients and disclose the potential prognostic role of these inflammatory markers.

Introduction

Multiple sclerosis (MS), is a demyelinating chronic autoimmune disease which causes irreversible axonal damages and disability, and thus impairing the CNS functions. MS has burdened the healthcare system and is affecting more than 2.5 million people worldwide [1-3]. The major cause of neurological impairment among female young adults in their 2nd to 4th decade of life is attributed to multiple sclerosis [4-6]. However, the pathogenesis of MS is still under debate, but inflammatory reactions affecting myelin components of the nervous system is believed to be the main cause of the disease. Variety of immune cells are participating in MS inflammatory process such as T cells, B cells, macrophages and dendritic cells, and endothelial cells and inflammatory cytokines are also contributing [5, 7]. In fact, T cells are activated as an autoimmune activity against myelin basic protein (MBP) and myelin oligodendrocyte glycoprotein (MOG) develops. After crossing the blood-brain barrier (BBB), the inflammatory cells through generation of inflammatory cytokines will damage myelin and oligodendrocytes. Hence, this inflammatory response leads to deterioration of myelin thickness in the brain and spinal cord, which subsequently destroys the CNS [5, 7, 8].