



# Traumatic brain injury with concomitant injury to the spleen: characteristics and mortality of a high-risk trauma cohort from the TraumaRegister DGU®

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## Abstract

**Purpose** Based on the hypothesis that systemic inflammation contributes to secondary injury after initial traumatic brain injury (TBI), this study aims to describe the effect of splenectomy on mortality in trauma patients with TBI and splenic injury.

**Methods** A retrospective cohort analysis of patients prospectively registered into the TraumaRegister DGU® (TR-DGU) with TBI ( $AIS_{\text{Head}} \geq 3$ ) combined with injury to the spleen ( $AIS_{\text{Spleen}} \geq 1$ ) was conducted. Multivariable logistic regression modeling was performed to adjust for confounding factors and to assess the independent effect of splenectomy on in-hospital mortality.

**Results** The cohort consisted of 1114 patients out of which 328 (29.4%) had undergone early splenectomy. Patients with splenectomy demonstrated a higher Injury Severity Score (median: 34 vs. 44,  $p < 0.001$ ) and lower Glasgow Coma Scale (median: 9 vs. 7,  $p = 0.014$ ) upon admission. Splenectomized patients were more frequently hypotensive upon admission (19.8% vs. 38.0%,  $p < 0.001$ ) and in need for blood transfusion (30.3% vs. 61.0%,  $p < 0.001$ ). The mortality was 20.7% in the splenectomy group and 10.3% in the remaining cohort. After adjustment for confounding factors, early splenectomy was not found to exert a significant effect on in-hospital mortality (OR 1.29 (0.67–2.50),  $p = 0.45$ ).

**Conclusion** Trauma patients with TBI and spleen injury undergoing splenectomy demonstrate a more severe injury pattern, more compromised hemodynamic status and higher in-hospital mortality than patients without splenectomy. Adjustment for confounding factors reveals that the splenectomy procedure itself is not independently associated with survival.

**Keywords** Traumatic brain injury · Splenectomy · Inflammation · Mortality · Humans

## Introduction

Traumatic brain injury (TBI) remains a major cause of mortality and disability particularly in young adults [1]. Local and systemic inflammation has been identified as an important contributor to secondary brain injury, which leads to additional downstream damage and represents a potential therapeutic target for neuroprotective approaches [2]. Peripheral immune mechanisms lead to systemic release of proinflammatory mediators and entry of immune cells into the CNS, particularly in the context of blood–brain barrier disruption [3, 4]. Peripheral immune organs like the spleen, which serves as a storage site for immune cells like monocytes [5], may be major contributors to peripheral immune responses. Indeed, a proinflammatory contribution of the spleen has already been demonstrated in the context of cerebral ischemia [6–8]. This important role of the spleen related to systemic

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