UCSCS IN TREATMENT OF T1DM AND T2DM; A META-ANALYSIS

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UCSCs IN TREATMENT OF T1DM AND T2DM; A META-ANALYSIS

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Background: Many patients struggle to achieve appropriate glucose control without side effects. Mesenchymal stem cells (MSCs) have been suggested for Diabetes Mellites (DM) treatment due to their immunomodulatory and regenerative properties. We aimed to evaluate the efficacy and safety of umbilical cord MSCs (UCMSCs) for DM management through this meta-analysis.
Methods: PubMed, Cochrane, WOS, Embase, and Scopus, were searched for randomized controlled trials (RCTs) investigating the effects of UCMSCs on DM (Types1, 2) till January 2024. Patient demographics, interventions, and outcomes, including glycated hemoglobin (HbA1c%), C-peptide levels, and insulin requirements, were extracted. Comprehensive meta-analysis software was used.
Results: Eight CTs with 334 patients (172 experimental, 162 control) were included. UMSCs treatment substantially lowered HbA1c levels (MD = –1.06, 95% CI [–1.27, –0.85], P < 0.00001), with consistent outcomes (i^2 = 0%, P = 0.43). Fasting C-peptide levels were heterogeneous but favored placebo (MD = 0.35, 95% CI [0.15, 0.56], P = 0.0007). In T1D patients, daily insulin requirements decreased considerably (MD = –0.24, 95% CI [–0.29, –0.18], P < 0.00001), with heterogeneity addressed by sensitivity analysis.
Conclusion: UMSCs therapy reduced HbA1c and insulin requirements and raised C-peptide. Multicenter clinical trials are needed to confirm UMSC therapy's long-term efficacy and safety.
Keywords: "Diabetes Mellitus," "Diabetes Type1," "Diabetes Type2," and "Umbilical Cord Mesenchymal Stem Cells," efficacy, Safety.

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NF-κB1,TNF,VEGF A&VEGF B IN A MOUSE MODEL OF SARS-CoV-2 INFECTION

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Introduction: The COVID-19 pandemic encouraged extensive research into its pathophysiology, specifically the role of biomarkers in disease progression. Although TNF, NF-κB1, VEGF-A and VEGF-B play fundamental roles in vascular development and infection response, their precise involvement in COVID-19 remains unclear. We aimed to evaluate and synthesize the TNF, NF-κB1, VEGF-A and VEGF-B gene expression patterns in a mouse model of SARS-CoV-2 infection to understand their involvement in disease pathogenesis.
Methods: Gene datasets available on the open-source Gene Expression Omnibus (GEO) platform were extracted from eleven specific datasets: GSE68220, GSE51387, GSE49262, GSE51386, GSE50000, GSE40824, GSE33266, GSE50878, GSE40840, GSE49263, and GSE40827. We used R 4.3.2 software in this analysis.
Results: A Substantial changes in the expression of VEGFA, VEGFB, TNF-, and NF-κB1 genes were noticed. Upregulation of TNF- and NF-κB1 genes implies a strong inflammatory response, consistent with their established involvement in inflammation. Conversely, VEGFA and VEGFB showed a pattern of downregulation, suggesting alterations in the vascular and endothelial functions.
Conclusion: During SARS-CoV infection, substantial changes in TNF, NF-κB1, VEGFA, and VEGFB gene expression were observed, indicating their interconnected roles in disease pathogenesis. These findings improve our understanding of the molecular basis of COVID-19 vascular complications and can guide future research and therapies.
Keywords: COVID-19, gene expression, VEGFA, VEGFB, and TNF, NF-κB1, Murine Model.

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ACTIVE VS. CONVALESCENT KAWASAKI DISEASE: COMPREHENSIVE MOLECULAR INSIGHTS

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Background: Kawasaki disease is a troublesome acquired condition characterized by systemic blood vessel inflammation and predominantly affects children below 5 years of age. It is diagnosed on clinical grounds, forming a syndrome; therefore, it is prone to misdiagnosis. Heart muscle damage and coronary artery abnormalities are fatal complications of Kawasaki disease; therefore, early diagnosis is crucial to halt this process. There are no discriminatory tests for differentiating Kawasaki disease from viral or bacterial infections. Furthermore, the molecular mechanism is not fully understood, posing an obstacle to the development of novel therapeutic targets.
Objective: We aimed to discern the genetic patterns and molecular mechanisms associated with Kawasaki disease (KD) by employing comprehensive gene expression analysis.
Methods: RNA sequences and microarray genomic datasets were obtained from NCBI Gene Expression Omnibus (GEO) datasets. Four