NEUROINFLAMMATION IN AUTISM SPECTRUM DISORDER: WHAT DO CURRENT STUDIES SHOW? A NARRATIVE REVIEW

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Evidence increasingly implicates immune–brain interactions in autism spectrum disorder (ASD). This review synthesizes human and translational findings on neuroinflammation and its relevance to ASD heterogeneity and treatment prospects. Narrative review of post-mortem, cerebrospinal fluid (CSF), blood, neuroimaging, and animal literature examining glial activation, cytokine profiles, blood–brain barrier (BBB) integrity, and peripheral–central immune crosstalk in ASD. Post-mortem studies frequently report microglial and astroglial activation, altered complement signaling, and cytokine dysregulation in cortical and cerebellar regions. CSF and peripheral assays often demonstrate elevated pro-inflammatory mediators (e.g., IL-6, TNF-α), though effect sizes vary and subgroups exist. Positron emission tomography using TSPO ligands shows mixed results, reflecting methodological limits (ligand affinity polymorphisms, age/sex effects) and biological heterogeneity. Genomic and transcriptomic data suggest immune-related pathways in subsets of individuals with ASD, while maternal immune activation models recapitulate ASD-like behaviors and microglial priming, underscoring developmental timing. Emerging work links gut dysbiosis and epithelial permeability to peripheral immune activation and possible BBB effects, but causality remains unresolved. Clinically, immune signatures correlate with symptom severity in some cohorts; however, anti-inflammatory or microglia-modulating interventions (e.g., minocycline, ibudilast, omega-3s) yield inconsistent, small-sample benefits and lack robust biomarkers to guide selection. Overall, convergent evidence supports context-dependent neuroinflammation in ASD—not universal, but salient in biologically defined subtypes. Priorities include longitudinal, multimodal studies (peripheral + CSF + imaging), stratification by age/sex/comorbidity, and development of validated inflammatory endophenotypes to enable targeted trials.

IMPLEMENTATION AND FEASIBILITY OF INTEGRATED PSYCHOLOGICAL CARE IN ACUTE STROKE: AN IMPLEMENTATION PERSPECTIVE

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Beyond clinical effects, integrating psychological care into the acute/subacute stroke pathway hinges on operational feasibility—workflow fit, coverage, adherence, acceptability, and safety. We evaluated the implementation of a brief, structured psychological care pathway in routine practice. Prospective service-development cohort (April–August 2025) including consecutive adults with ischemic or hemorrhagic stroke. The intervention comprised up to six sessions emphasizing psychoeducation, coping skills, problem-solving, and support. Implementation indicators: eligibility and coverage (proportion enrolled of those admitted), adherence (sessions completed), acceptability (completion of standardized measures: EQ-5D/VAS, CANSAS, GAD-7, HAM-A, HAM-D, PHQ-9; MMSE at entry when appropriate), safety (intervention-related adverse events), and workflow integration (ability to deliver sessions during acute/subacute care). A total of 121 patients were enrolled, with cardiometabolic comorbidities common. The pathway was delivered without disrupting medical care; most patients completed ≥4 sessions and pre/post assessments, indicating good acceptability and manageable administrative load. No intervention-related adverse events were reported. Standardized tools facilitated interdisciplinary communication and needs prioritization (via CANSAS), while early in-hospital initiation improved coverage. From an implementation standpoint, brief integrated psychological care proved feasible in an acute setting and shows potential to improve patient-reported outcomes and continuity of care after discharge.