Staphylococcus aureus infected embolic stroke upregulates Orm1 and Cxcl2 in a rat model of septic stroke pathology - DTU Orbit (03/10/2019)

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Objective: Ischaemic brain lesions and brain abscesses are frequent in both human and animal cases of septic embolic stroke. However, existing models of brain infection do not reflect central aspects of septic embolic stroke. Our aim was to compare septic and non-septic embolic stroke in order to identify gene expressions, inflammatory mediators and brain damage in a rat model. Methods: We created precisely located focal brain infarcts in a rat model of Staphylococcus aureus infected embolic stroke. To cause septic embolic stroke we used a fibrin-rich embolus with bacteria, while every rat in the control group received a non-infected embolus. 64 rats were randomized to receive sham-surgery, sterile embolic stroke or septic embolic stroke. All groups were compared for brain pathology, mortality, gene expressions and inflammatory mediators using histology and reverse transcription quantitative real-time PCR. Results: Although infarct volumes did not differ, septic embolic stroke caused higher mortality than sterile embolic stroke (p = 0.002). Brain abscesses were observed only in the septic group. Approximately 400–500 fold increases were observed for Orm1 and Cxcl2 respectively (1.00E-08 < p < 1.92E-07) in the septic group compared to the sterile group, and these were the most dramatically regulated genes in septic embolic stroke compared to sterile embolic stroke. Conclusions: Septic embolic stroke caused brain abscesses, increased mortality and upregulated Orm1 and Cxcl2 gene expressions compared to non-infected embolic stroke. The dramatic Orm1 increase observed in the septic group is unprecedented and suggests a significant biological role of Orm1 during septic neuroinflammation.

**General information**
Publication status: Published
Organisations: National Veterinary Institute, Innate Immunology, University of Copenhagen
Corresponding author: Astrup, L. B.
Pages: 399-412
Publication date: 2019
Peer-reviewed: Yes

**Publication information**
Journal: Neurological Research
Volume: 41
Issue number: 5
ISSN (Print): 0161-6412
Ratings:
BFI (2019): BFI-level 1
Web of Science (2019): Indexed yes
Original language: English
Keywords: abscess, animal model, Embolic stroke, gene expressions, neuroinflammation, septic
DOIs:
10.1080/01616412.2019.1573455
Source: Scopus
Source ID: 85060914084
Research output: Contribution to journal › Journal article – Annual report year: 2019 › Research › peer-review