Perubahan biomarka neuroinflamasi, stres oksidatif dan produk degradasi darah pada perdarahan intraserebral spontan = Changes of neuroinflamation, oxidative stress and blood degradation products CSF biomarkers in spontaneous intracerebral hemorrhage.

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Abstrak

Cedera otak pada perdarahan intraserebral spontan (PIS) terdiri dari cedera primer kerusakan struktural karena proses mekanis dan cedera sekunder akibat respons patofisiologis subklinis mencakup inflamasi, stress oksidatif dan sitotoksik terhadap komponen serta produk degradasi darah. Proses subklinis PIS yang sedang berlangsung tersebut masih belum terpantau secara lengkap, sehingga penelitian ini ditujukan untuk mengidentifikasi perjalanan proses subklinis cedera otak sekunder perdarahan intraserebral spontan dan pengaruhnya terhadap perubahan luaran respons klinis kasus (LRK) PIS pasca-intervensi bedah saraf. Penelitian ini menggunakan desain observasional prospektif, mulai Agustus 2016-April 2018 terhadap 20 subjek yang baru pertama kali mengalami perdarahan intraserebral spontan yang disertai perdarahan intraventricular dan menjalani intervensi bedah saraf external ventricular drainage (EVD). Data tercatat mencakup skor Full Outline of UnResponsiveness (SF), TIK, dan kadar hari ke-1 dan hari ke-7 Tumor Necrosis Factor alpha (TNFα), Superoxide Dismutase (SOD) dan zat besi dalam LSS. Analisis bivariat menggunakan uji Ttak berpasangan atau uji Mann-Whitney. Data skala kategorik diuji dengan Chisquare atau Fisher's exact test, dan untuk data kategorik berpasangan dengan uji McNemar.
TIK pasca-intervensi semua subjek menurun secara gradual menjadi normal dan ada lima subjek yang tidak mengalami perbaikan LRK SF hari 1-7. Semua subjek kelompok 'tanpa perbaikan' mempunyai kadar TNFα LSS hari ke-1 tinggi, sebaliknya yang kadarnya normal mengalami perbaikan LRK (P=0,003). Selisih nilai peningkatan TNFα hari 1-7 juga lebih besar bermakna pada yang 'tanpa perbaikan' (P=0,005). Kadar SOD LSS hari ke-1 dan perubahannya tidak terbukti berbeda bermakna antara kedua kelompok. Pengamatan klinis memperlihatkan 80% subjek 'perbaikan', mempunyai kadar zat besi LSS hari ke-1 normal dalam status saturasi transferin < 50%. Semua subjek yang mempunyai kadar zat besi hari ke-1 tinggi dalam status saturasi transferin ≥ 50% mengalami LRK 'tanpa perbaikan'. Terdapat perbedaan bermakna dari selisih peningkatan status saturasi transferin antara kedua kelompok subjek. (P=0,05). Penelitian ini menyimpulkan bahwa subjek PIS dengan kadar TNFα LSS hari ke-1 tinggi dan/atau zat besi LSS tinggi dalam status saturasi transferin ≥ 50%, mempunyai LRK 'tanpa perbaikan'. Semakin besar peningkatan kadar TNFα LSS pada hari ke-7 dan/atau kadar zat besi yang disertai peningkatan saturasi transferrin, mempunyai LRK 'tanpa perbaikan'. Kadar SOD hari ke-1 dan perubahan kadar hari 1-7 belum dapat dimanfaatkan sebagai penanda prognosis dan proses subklinis PIS.
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Brain injury of spontaneous intracerebral hemorrhage caused by primary injury of structural damage due to mechanical processes and secondary injuries resulting from subclinical pathophysiological responses of inflammation, oxidative stress and cytotoxicity to components and blood degradation products including iron. The subclinical pathophysiology processes still cannot be monitored explicitly. This study is aimed at identifying the course of ICH subclinical secondary brain injury process and finding its relations with the

days 1-7 trend of clinical response outcomes after neurosurgical intervention. This study is a prospective observational designed study done from August 2016 until April 2018. Twenty subjects were diagnosed as spontaneous intracerebral hemorrhage and underwent neurosurgical intervention external ventricular drainage (EVD). Recorded data consist of everyday Full Outline of UnResponsiveness (FOUR) score, intracranial pressure, and cerebro-spinal fluid (CSF) Tumor Necrosis Factor alpha (TNFα), Superoxide Dismutase (SOD), iron and transferrin saturation at day-1 and day-7. Bivariate analysis performed with unpaired T-test or Mann-Whitney test. Unpaired categorical scale data tested by Chi-square or Fisher's exact test, and McNemar test for paired categorical data.
All 'unimproved' subjects had high levels of day-1 CSF TNFα, whereas all subjects with normal TNFα have clinical improvement response (P=0.003). Subsequently those subjects had significantly greater increasing levels (P=0.005). No significant difference of CSF SOD between of 'unimproved' and 'improved' group. Clinical observation clearly showed that 80% of 'improved' subjects have normal day-1 iron levels in controlled by transferrin saturation < 50%. There will be no improvement of those high iron levels with transferrin saturation ≥ 50%. A significant difference results were also noted of increasing transferrin saturation status (P=0,05). This study concluded that SICH with high level of day-1 CSF TNFα and/or high CSF iron with transferrin saturation ≥ 50%, would have an 'unimproved' trend of clinical response outcome. Greater increasing level of those biomarkers in days 1-7, tend to have an unimproved outcome. CSF SOD could not to be use as a significant clinical prognostic and process biomarker.