

Kalprotektin dan alfa-1 antitripsin tinja sebagai petanda inflamasi usus dan prediktor terjadinya kolestasis terkait sepsis neonatorum = Faecal calprotectin and alpha 1 antitrypsin as intestinal inflammation marker and predictor sepsis associated cholestasis

Firda Fairuza, author

Deskripsi Lengkap: <https://lib.ui.ac.id/detail?id=20389039&lokasi=lokal>

Abstrak

Latar belakang. Kolestasis terkait sepsis (KTS) masih merupakan permasalahan medis di negara berkembang disebabkan tingginya morbiditas, mortalitas dan lama rawat. Inflamasi usus akibat disfungsi sawar usus diduga berperan dalam KTS sehingga perlu dibuktikan perannya terhadap terjadinya KTS. Inflamasi dan permeabilitas mukosa usus dapat dinilai melalui kadar kalprotektin dan alfa-1 antitripsin (AAT) pada tinja.

Tujuan. Untuk mengetahui hubungan antara terjadinya KTS pada sepsis neonatorum dengan inflamasi dan gangguan permeabilitas usus yang dinilai dengan peningkatan kadar kalprotektin dan -1-antitripsin dalam tinja. Metode. Studi kohort prospektif di ruang rawat inap Perinatologi dan Neonatal Intensive Care Unit Departemen Ilmu Kesehatan Anak Rumah Sakit Cipto Mangunkusumo periode Juni 2012- Oktober 2013. Delapan puluh neonatus diambil secara consecutive sampling dari 271 subjek proven sepsis yang dirawat pada periode studi ini, terbagi menjadi 2 kelompok (KTS dan sepsis tidak kolestasis) masing-masing 40 subjek. Dilakukan pemeriksaan kadar kalprotektin dan AAT tinja.

Hasil penelitian. Tidak ditemukan perbedaan antara KTS dan sepsis tidak kolestasis dalam ekskresi kalprotektin tinja [KTS vs. sepsis tidak kolestasis, median (rentang) 104,4 (25 sampai 358,5) vs. 103,5 (5,4 sampai 351) g/g; $p = 0,637$] dan alfa-1 antitripsin tinja [median (rentang) 28 (2 sampai 96) vs. 28 (2 sampai 120) mg/dL; $p = 0,476$]. Tidak ditemukan peningkatan bermakna kadar kalprotektin tinja dengan nilai $p = 0,63$ (IK 95% 0,4 sampai 3,6) dan kadar AAT tinja dengan nilai $p = 0,152$ (IK 95% 0,4 sampai 3,3).

Simpulan. Kadar kalprotektin dan alfa-1 antitripsin tinja tidak terbukti dapat memprediksi kejadian KTS pada sepsis neonatorum. Tidak ada bukti proses inflamasi usus yang terjadi pada KTS melalui peningkatan permeabilitas paraselular usus. Perlu dilakukan penelitian lebih lanjut mengenai patogenesis inflamasi usus yang terjadi melalui peningkatan permeabilitas trans-selular dan kerusakan enterosit usus pada KTS.

<hr>

Background. Sepsis-associated cholestasis (SAC) remain a medical problem in developing countries due to high morbidity, mortality and length of hospital. Intestinal inflammation as the causes of intestinal barrier dysfunction are suspected play a role in SAC, so it is necessary to prove its contribution to SAC. Intestinal inflammation and increased permeability were assessed through faecal calprotectin and alpha-1 antitrypsin (AAT) concentrations.

Objective. To determine the association between SAC in sepsis neonatorum with intestinal inflammation and permeability were assessed through increased faecal calprotectin and AAT levels.

Methods. This was cohort prospective study at Perinatologi and Neonatal Intensive Care Unit Department of Child Health Cipto Mangunkusumo Hospital during June 2012 to October 2013. Eighty neonates were obtained by consecutive sampling, of which 271 proven sepsis hospitalized in this period, divided 2 groups (SAC and non cholestasis sepsis) respectively 40 subjects. Faecal calprotectin and AAT concentrations was

measured.

Results. There was no significant association between SAC and faecal calprotectin excretion [SAC vs. non cholestasis sepsis, median (range) 104.4 (25 to 358,5) vs. 103.5 (5.4 to 351) g/g; $p = 0.637$] and faecal AAT [median (range) 28 (2 to 96) vs. 28 (2 to 120) mg/dL; $p = 0.476$]. Increased faecal calprotectin (CI 95% 0.4 to 3.6; $p = 0,63$) and AAT (CI 95% 0.4 to 3.3; $p=0.152$) did not differ significantly between the two groups.

Conclusions. Faecal calprotectin and alpha-1 antitrypsin concentrations is not associated with SAC in sepsis neonatorum. There is no evidence of intestinal inflammation causes increased paracellular intestinal permeability in SAC. Further research is needed on the pathogenesis of intestinal inflammation in SAC which may result in increased intestinal permeability by transcellular and enterocyte damage.