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Perubahan ekspresi protein penaut endotel kapiler pada hipoksia plasenta upaya untuk memahami perubahan permeabilitas endotel kapiler pada bayi prematur dengan perdarahan intraventrikel = Protein junction expression changes of capillary endothelium due to placental hypoxia an effort to understand endothelial permeablity in premature infants with intraventricular hemorrhage

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Abstrak

Pendahuluan: Hipoksia plasenta pada bayi prematur menyebabkan stres oksidatif yang merusak protein penaut endotel kapiler plasenta. Kerusakan pada kapiler plasenta diharapkan dapat menggambarkan perubahan permeabiltas kapiler otak yang menyebabkan perdarahan intraventrikel.
Metode: Penelitian observasional potong lintang terhadap 58 sampel plasenta bayi prematur. Hipoksia dinilai dari saturasi vena umbilikal, respon jaringan terhadap hipoksia dari kadar HIF-1, stres oksidatif dari kadar malondialdehid (MDA) dan glutation (GSH). Integritas lapisan endotel dinilai dengan histomorfologi, ekspresi protein N-kaderin dan okludin dengan imunohistokimia, Glial fibrillary acidic protein (GFAP) sebagai petanda kerusakan perivaskular astrosit dan perdarahan intraventrikel dinilai dengan ultrasonografi kepala.
Hasil: Kadar HIF-1 lebih tinggi tidak bermakna pada kelompok hipoksia dibandingkan kelompok non hipoksia (uji t tidak berpasangan, p = 0,122); Kadar MDA jaringan plasenta lebih tinggi tidak bermakna sedangkan GSH lebih rendah tidak bermakna (Mann Whitney, p = 0,414 dan p = 0,810). Gambaran histomorfologi lebih tidak utuh tidak bermakna (Chi-square, p = 0,066), sedangkan ekspresi N-kaderin dan okludin lebih rendah bermakna (Chi-square, p = 0,001). Kadar GFAP serum lebih tinggi bermakna (Mann Whitney, p = 0,05). Korelasi tidak bermakna antara ekspresi N-kaderin dan okludin dengan perdarahan intraventrikel (Spearman?s rho, p = 0,869 dan p = 0,341).
Kesimpulan: Hipoksia pada plasenta menyebabkan perubahan ekspresi protein penaut endotel kapiler plasenta, secara molekuler sudah menyebabkan perubahan permeabilitas lapisan endotel kapiler plasenta tetapi secara struktural belum. Perubahan ekspresi protein penaut endotel kapiler plasenta tidak berhubungan dengan perdarahan intraventrikel.
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Introduction: Plasental hypoxia in premature infants causes oxidative stress which inflicts damage to endothelial protein junction of placental capillary. It is expected that damaged of placental capillary can demonstate permeability changes in brain capillary that can cause intraventricular hemorrage.
Method:.a cross sectional observational study conducted on 58 placenta of premature infants. Hypoxia is determined by umbilical venous saturation. Tissue response to hypoxia determined by the level of HIF-1, stress oxidative by the level of malondialdehide (MDA) and glutation (GSH). Endothelial layer integtrity by histomorfologi overview, N-cadherine and occludin by immunohistochemistry. Glial fibrillary acidic protein (GFAP) as perivascular astrocyte disruption marker and intraventricular hemorrhage carried by head ultrasound.
Result: The levels of HIF-1 was not significantly higher in hypoxia group compared to non hypoxia group

(unpaired t test, p = 0,122); The level of placental MDA was not significantly hingher while GSH was not significantly lower (Mann Whitney, p = 0,414 and p = 0,810). Histomorpological overview was not significantly not intact (Chi-square, p = 0,066), while the expression of N-cadherine and occludin were significantly lower (Chi-square = 0,001). There was not significant correlation between protein junction expression with intravenrticular hemorrhage (Spearman?s rho, p = 0,869 and p = 0,341).
Conclution: Hypoxia causes lower expression of N-cadherin and occludin, moleculary it cause placental endothelial capillary permeability but structurally it does not. Protein expression changes does not correlate with intraventricular hemorrhage.