Analisis struktur histologis dan molekuler plasenta bayi prematur dengan hipoksia = Histological and molecular analysis of the premature placental hypoxia

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Abstrak

Latar belakang: Prematuritas merupakan salah satu kelainan yang masih menjadi masalah global. Kejadian prematuritas tidak hanya terjadi di negara berkembang tetapi juga di negara maju. Beberapa kondisi ibu hamil dapat memicu keadaan hipoksia dalam rahim sehingga menyebabkan kelahiran prematur. Keadaan plasenta menggambarkan kesejahteraan janin intra uteri. Kondisi hipoksia seluler memicu ekspresi HIF-1 yang menjadi faktor transkripsi bagi CA9 sebagai penanda hipoksia. Penelitian ini bertujuan menganalisis pengaruh hipoksia terhadap plasenta prematur.
Metode: Sampel menggunakan plasenta prematur yang hipoksia (H) dan nonhipoksia (N) sebagai kontrol. Parameter yang dinilai adalah struktur histologis plasenta (Hematoksilin-Eosin), regulator hipoksia HIF-1 (imunohistokimia), dan penanda hipoksia CA9 (ELISA).
Hasil: Penilaian struktur histologis menunjukkan adanya perbedaan jumlah pembuluh darah fetus antara kedua kelompok secara bermakna, dimana pada kelompok hipoksia jumlah pembuluh darah fetus lebih banyak dibandingkan kelompok non-hipoksia. Distribusi intensitas ekspresi HIF-1 kedua kelompok juga berbeda bermakna. Rerata kadar CA9 kedua kelompok tidak berbeda bermakna, namun terdapat kecenderungan rerata kadar CA9 kelompok hipoksia lebih tinggi 28% dibandingkan yang non-hipoksia.
Kesimpulan: Pengaruh hipoksia terhadap plasenta prematur pada tingkat molekuler berupa stabilitas protein HIF-1 yang menyebabkan peningkatan jumlah pembuluh darah fetus dan terjadi kecenderungan peningkatan sintesis protein CA9.
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Background: Prematurity is a disorder that is still a global problem. Incidence of prematurity is a problem in developing and also in developed countries. Certain condition accompanying pregnancies may trigger uterine hypoxia, causing premature birth. The placental condition is related with the intra-uterine fetal condition. Cellular hypoxic condition caused by systemic chronic hypoxia, lead to stabilization of HIF-1 protein, a transcription factor of CA9. This study aimed to analyze the effect of hypoxia on the premature placenta.
Methods: Samples from hypoxic premature placenta (H) and non-hypoxic premature placenta (N) were collected. Parameters assessed were histological structure of the placenta (Hematoxylin-Eosin), expression of HIF-1 (immunohistochemistry) and the level of CA9 (ELISA).
Results: Assessment of histological structure showed the number of fetal blood vessels were differed significantly between the two group, wherein the hypoxia group was more than the non-hypoxia. The distributions of HIF-1 expression between the two groups were also differed significantly. The average level of CA9 between two groups were not significant, but there is a tendency of higher level of CA9 in the hypoxia group (28% higher compared to the non-hypoxia group).
Conclusion: It is concluded that the effect of the hypoxia on premature placenta in this study occured at molecular level and lead to HIF-1 protein stability that causes an increase of the number of fetal blood

vessel and synthesis of CA9 protein.