PROLACTIN AS A FACTOR OF REMYELINATION IN A TOXIC CUPRIZONE-INDUCED MODEL OF MULTIPLE SCLEROSIS

E. Predtechenskaya, A. Khodakov, N. Popova, A. Romashenko
Department of Medicine, Novosibirsk State University, Russia
Elena_pred@mail.ru

Is prolactin increase an independent factor of hormonal defense during pregnancy, or is it itself a consequence of increase in placental steroids? To evaluate the protective effect of prolactin we create a murine toxic cuprizone model of demyelination. To induce pure hyperprolactinemia, male mice have been administered metoclopramide (cerucal). Metoclopramide-induced hyperprolactinemia is well documented in clinical gynecology as well as in endocrinology. The population of mice has been divided into three groups:
[1] control group (18 animals)
[2] mice that have been administered cuprizone for 6 weeks (26 animals)
[3] mice that have been subcutaneously administered metoclopramide at the same time or before they have been fed with cuprizone (24 animals)
Demyelination has been studied using MRT flurane anesthesia. The power of magnetic field has been set to 11T. We analyzed 6th and 7th slices from the the frontal part of olfactory bulb. We measured the change in the area of callosum in pixels (pu).
Results: Control group: Average value of callosum area within the group is 215pu, st. dev. 14pu. Second group: Average value of callosum area is 89pu, st. dev. 17pu. Third group: Average value is 148pu, st. dev. 29pu.
Conclusions: Prolactin offers a potent protection to brain tracts subjected to cuprizone-induced demyelination. The present model can be extrapolated to physiological hyperprolactinemia during pregnancy. We conjecture that the protective effect of prolactin is related to accelerated remyelination, histologically manifested through accelerated growth of oligodendrocytes precursor cells. We do not exclude that the direct effect of prolactin is strengthened by the effect of placental steroids.