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Omgea-3 Polyunsaturated Fatty Acids attunuates Contrast-Induced Blood-Brain-Barrier Injury in Uremic Mice

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Objectives : Patients with chronic kidney disease (CKD) often require contrast media (CM) examinations due to underlying conditions. While CM can impact brain cells, the blood-brain barrier (BBB) typically safeguards against such damage in vivo. However, CKD-associated uremia can disrupt the BBB, increasing the risk of CM-induced brain-cell damage. ω-3 polyunsaturated fatty acids (PUFAs) have demonstrated protective effects in various neurological disorders, including uremic brain injury. This study aimed to investigate whether ω-3 PUFAs could mitigate BBB damage caused by uremia and contrast agents in a uremic mouse model, and to assess the underlying mechanisms.
Methods : C57BL/6 mice (eight weeks old, male) and fat-1 mice (b6 background/eight weeks old, male) were categorized based on uremic induction, CM exposure, and ω-3 PUFA administration. Uremia was induced through 24-hour ischemia-reperfusion (IR) renal injury. Brain tissue, kidney tissue, and blood were collected one day after CM treatment.
Results : The study revealed increased expression levels of glial fibrillary acidic protein (GFAP), claudin 5, CD31, laminin α4, and laminin α5 in ω-3 PUFA + CM-treated uremic mice and the brains of fat-1 + CM-treated uremic mice compared to CM-treated uremic mice alone. Pro-apoptotic protein expression decreased, while anti-apoptotic proteins increased in ω-3 PUFA + CM-treated uremic mice and fat-1 + CM-treated uremic mice compared to CM-treated uremic mice. Furthermore, the brain-expression levels of p-JNK, p-P53, and p-P38 decreased in the ω-3 PUFA + CM-treated uremic mice and fat-1 + CM-treated uremic mice compared to wild-type uremic mice.
Conclusions : In conclusion, both uremic toxin and CM were found to damage the BBB, leading to brain-cell death. The study suggests that ω-3 PUFAs play a crucial role in protecting the BBB from CM-induced damage in uremic mice.