Serum-mediated osteogenic effect in traumatic brain-injured patients

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BACKGROUND: Patients with a traumatic brain injury (TBI) and bone fractures often show an enhanced fracture healing, as well as an increased incidence of heterotopic ossifications (HO). It has been suggested that unknown osteoinductive factors may be released by the injured brain into the systemic blood circulation and act peripherally on the affected tissues. The aim of this study was to investigate whether serum from TBI patients is osteoinductive.

METHODS: Sixty-one consecutive patients were classified into four groups: TBI and long-bone fracture (group I, n = 12), isolated severe TBI (group II, n = 21), isolated long-bone fracture (group III, n = 19) and controls (group IV, n = 9). Blood samples were collected at 6, 24, 72 and 168 h post-injury. The osteogenic potential was determined by measuring the in vitro proliferation rate of the human fetal osteoblastic cell line hFOB1.19, and primary human osteoblasts. Additionally, serum induced osteoblastic differentiation was assessed by measuring the mRNA expression of specific osteoblastic markers, including alkaline phosphatase, runt-related transcription factor 2, cathepsin K and serine protease 7.

RESULTS: The sera of group I induced a higher mean proliferation rate of primary human osteoblasts at all time points of sampling than group III (P < 0.05). Group I had a higher mean proliferation rate of hFOB1.19 cells than all other groups at 6, 24 and 72 h post-injury (P < 0.05). The expression of alkaline phosphatase, cathepsin K and runt-related transcription factor 2 mRNA was increased in group I compared with group III and serine protease 7 was exclusively expressed in group I.

CONCLUSION: The study results strongly support a humoral mechanism in enhanced fracture healing and the induction of HO after TBI. Increased proliferation of osteoblastic cells and an accelerated differentiation of osteoprogenitor cells may be responsible for increased osteogenesis in TBI.

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