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Use of Autologous Growth Factors in Lumbar Spinal Fusion

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The results of spinal fusion, especially posteriorly above the lumbosacral junction, have been mixed. Autologous growth factor concentrate (AGF) prepared by ultraconcentration of platelets contains multiple growth factors having a chemotactic and mitogenic effect on mesenchymal stem cells and osteoblasts and may play a role in initiating bone healing. The purpose of this retrospective study is to review our results with AGF in lumbar spinal fusions. To date, AGF has been used in 39 patients having lumbar spinal fusion. The study group consisted of the first 19 consecutive cases to allow at least 6 months follow-up. The average follow-up was 13 months (range 6 to 18 months). Follow-up compliance was 91%. There were 7 men and 12 women. Average age was 52 years (range 30–72 years). Nine patients had prior back surgery. There were 8 smokers. AGF was used in posterior (n = 15) or anterior intradiscal (n = 4) fusions. AGF was used with autograft and coraline hydroxyapatite in all posterior fusions, and autograft, coral, and intradiscal spacer (carbon fiber spinal fusion cages or Synthes femoral ring) in intradiscal fusions. Posterior stabilization was used in all cases. Eight cases were single-level fusions, 6 were two-level, and 1 was a three-level fusion. Autologous iliac crest bone graft was taken in 14 cases and local autograft used in 5 cases. Posteriorly, a total of 23 levels were fused; of these, nine were at L5–S1, eight at L4–L5, five at L3–L4, and one at L2–L3. No impending pseudoarthroses were noted on plain radiographic examination at last follow-up visit. Solid fusion was confirmed in 3 patients having routine hardware removal, and in 2 patients who had surgery at an adjacent level. There was one posterior wound infection, which was managed without sequelae. When used as an adjunct to autograft, AGF offers theoretical advantages that need to be examined in controlled studies. Further study is necessary to determine whether coralline hydroxyapatite used as a bone graft extender in lumbar spinal fusion may help to obviate the need for secondary site graft harvesting. (Bone 25: 47S–50S; 1999) **© 1999 by Elsevier Science Inc. All rights reserved.**

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Introduction

The vital role played by local growth factors in the complex series of events that lead to bone healing and graft incorporation has been known for almost three decades. The discovery of bone morphogenetic protein (BMP) in 1965 by Marshall Urist has led to various studies for identification and isolation of purified forms of a variety of growth factors that play roles in osteogenesis. A large number of polypeptides have been identified since then and have been grouped based upon their structural homologies into at least 20 families and superfamilies.[13] The most widely studied of these are the insulin-like growth factor (IGF), epidermal growth factor (EGF), fibroblast growth factor (FGF), platelet-derived growth factor (PDGF), and the transforming growth factor (TGF) group, of which, the BMPs form a subgroup. The potentially beneficial effects of PDGF on bone formation in vitro has been described in many studies,[2,3,5,6,9,15,17] and in vivo stimulation of bone formation by TGF-β has also been demonstrated in rats.[8,14] Various studies have demonstrated the chemotactic and mitogenic effects of local growth factors on mesenchymal cells as well as osteoblasts and periosteal fibroblasts.[4,5,12,16] Bone formation at the site of a fracture or at the site of grafting is initiated by a process of fibrin clot formation, platelet aggregation, and degranulation. Platelet granules contain a variety of physiologically active substances such as catecholamines, serotonin, calcium ions, ATP, albumin, fibrinogen, osteonectin, osteocalcin, various clotting factors, and the locally active growth factors like PDGF, TGF-β, IGF-I, IGF-II, FGF, and EGF.[17] PDGF has been shown to be chemotactic to fibroblasts as well as to monocytes and primitive mesenchymal cells.[3,16] TGF-β has also been reported to have chemotactic activity towards osteoblast precursors.[10] Both PDGF and TGF-β have mitogenic activity by stimulation of DNA synthesis and cell replication.[6] Caplan[4] has described three sites in bone containing undifferentiated stem cells that are capable of differentiating into osteoblasts, namely, the marrow, periosteum, and perivascular sheath. PDGF and TGF-β have a chemotactic and mitogenic effect on these cells that causes them to multiply and secrete additional growth factors. Then, under the influence of other cytokines and local environmental conditions such as pH, oxygen tension, and micromotion, these cells undergo a differentiation into osteoblasts or chondroblasts. Collagen and protein synthesis by osteoblasts is also stimulated by PDGF but also needs the presence of IGF-I.[7] PDGF probably enhances the secretion of IGF-I by the osteoblasts and mesen-