

Introduction

Ventral hernia repair (VHR) remains one of the most common general surgery operations. In the face of an ever-changing market of surgical materials, synthetic mesh endures as a mainstay in the armamentarium of a herniologist. Despite mesh robustness, high long-term hernia recurrence rates signify ample opportunity for technical improvement. Platelet-rich plasma (PRP) has been shown to improve surgical soft tissue wound healing and enhance incorporation of acellular dermal matrices. We hypothesized that the addition of PRP would enhance host tissue incorporation after prosthetic VHR in rodents using polyester mesh.

Materials/Methods

Chronic ventral hernias were created in Lewis rats (N = 30) via laparotomy, skin closure and hernia maturation for >28 days. Rats were randomly assigned either to polyester mesh (Parietex™) alone control (A) or PRP-treated Parietex group (B) (N=5/group). Autologous PRP (1x10⁶ platelets/μL) was isolated via double-centrifugation of whole blood, activated with thrombin, and applied to the mesh surface of group B rats implanted in underlay fashion. Animals were sacrificed at 2, 6, or 12 weeks postoperatively, specimens harvested en bloc and stained with H&E for cellular detail. Qualitative and quantitative microscopic analysis was performed on histologic samples for tissue characteristics and neovessel formation. Samples were compared for molecular expression of the inflammatory gene marker iNOS at 2 weeks and regenerative gene markers (Col1a1, Col3a1, and VEGF-A) at 2, 6 and 12 weeks.

Results

Relative expression of the pro-inflammatory gene iNOS was significantly downregulated (147-fold, p<0.01, Fig. 1) in group B rats compared to control. Group B samples showed downregulated expression of Col3a1 at 2 weeks (3.75-fold, p<0.001, Fig. 1), and enhanced expression of all 3 regenerative genes at 6 weeks: VEGF-A (28-fold, p<0.001), Col3a1 (17-fold, p<0.0001), and Col1a1 (39-fold, p<0.001, Fig. 1). This genetic expression translated to significant tissue effects. On qualitative histological analysis, group B specimens had greater tissue deposition overall (Fig. 1). On quantitative analysis, group B rats formed on average 2.7-fold more neovessels than group A rats (179.8 +/- 70.0 vs 66.8 +/- 8.6, p<0.05).

Conclusions

This study demonstrates the ability of PRP to augment the host tissue response to implanted synthetic hernia mesh. By altering local genetic expression to favor regeneration over the innate, foreign body immune response, neovascularization and tissue incorporation were enhanced in Group B rodents. Although correlation with enhanced mechanical strength or decreased hernia recurrence remain to be proven, PRP should be formally investigated as a supplement for synthetic VHR and surgical wound healing.

