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REVIEW ARTICLE

Update on biomaterials for prevention of epidural adhesion after lumbar laminectomy

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Summary Lumbar laminectomy often results in failed back surgery syndrome. Most scholars support the three-dimensional theory of adhesion: Fibrosis surrounding the epidural tissues is based on the injured sacrospinalis behind, fibrous rings and posterior longitudinal ligaments. Approaches including using the minimally invasive technique, drugs, biomaterial and nonbiomaterial barriers to prevent the postoperative epidural adhesion were intensively investigated. Nevertheless, the results are far from satisfactory. Our review is based on various implant biomaterials that are used in clinical applications or are under study. We show the advantages and disadvantages of each method. The summary will help us to figure out ideas towards new techniques.

The translational potential of this article: This review summarises recent biomaterials-related clinical and basic research that focuses on prevention of epidural adhesion after lumbar laminectomy. We also propose a novel possible translational method where a soft scaffold acts as a physical barrier in the early stage, engineered adipose tissue acts as a biobarrier in the later stage in the application of biomaterials and adipose-derived mesenchymal stem cells are used for prevention of epidural adhesion.
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Introduction

About 8—40% patients after lumbar laminectomy suffered from failed back surgery syndrome (FBSS), and 4—9% patients suffered from the second surgery [1]. Extension of scar tissue into the neural canal and adhesion to the dura mater are considered to be the major reason for leg and back pain. The formation and repair process of scar tissue can be classified into three phases (See Figure 1). The first phase is the local inflammatory reaction in the first 3—5

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days after surgery, mainly including haemostasis and coagulation process and chemokine release such as phospholipase A2, which causes the aggregation of macrophagocytes, fibroblasts, mastocytes and endotheliocytes [2]. The second phase lasts 2—3 weeks. Fibroblasts proliferate and differentiate into fibrocytes, secrete collagenous fibres in the defect lesion and form granulation tissues gradually. Fibroblast proliferation, immigration and extracellular matrix synthesis are regulated by various cytokines, such as transforming growth factor (TGF)-β1, interleukin-6 (IL-6) and fibroblast growth factor (FGF). Fibroblasts could also secret TGF-β1, IL-6 and FGF-2 to improve fibroblast proliferation and extracellular matrix synthesis [3]. The third phase is tissue reconstruction which lasts months to years; fibrillar connective tissues deposit around the defect lesion and transform into scar tissues [4].

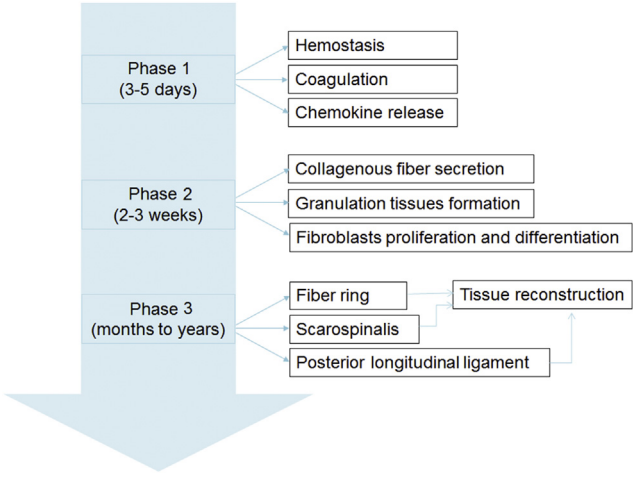

Figure 1 Process of adhesion after lumbar laminectomy.

Origination of the epidural scar

People tried for a long period to find the origination of the epidural scar. Is the epidural scar originated from injured tissue due to surgical approach, including sacral spine muscle, lamina, ligamentum flavum, posterior longitudinal ligament or fibre ring? Key and Ford [5] considered injured intervertebral disc fibre is the major source of epidural adhesion. LaRocca and Macnab [6] performed the surgery in dogs and considered the rough surface of sacrospinalis in the surgical lesion behind the spinal canal to be the major source. Fibroblasts in the deep layer of sacrospinalis proliferate and form the laminectomy membrane from sacrospinalis to the side of the dura mater. However, so far, the most approved mechanism of adhesion is raised by Songer and Ghosh Spencer [7]. They proposed a three-dimensional theory. It is said that the scar tissue around the dura mater originates not only from sacrospinalis behind, but also from the fibre ring and posterior longitudinal ligament ahead. The hyperplasia of fibrous tissue around the ventrolateral nerve root caused epidural adhesion.

Evaluation of epidural adhesion

How to evaluate the degree of epidural adhesion? Generally, there are three main aspects to evaluate the adhesion: macroscopic analysis, histological analysis and magnetic resonance imaging (MRI) analysis. Macroscopic analysis is carried out in a space between the dura mater and surrounding soft tissues. It is based on the quality of wound healing, possible adverse effects and epidural adhesion. Adhesion tenacity is also evaluated using Rydell and Balazs's standard score. Grade 0 shows no obvious adhesion between the dura mater and the scar; Grade 1 shows scattered and slight adhesion between the dura mater and the scar which is easily separable; Grade 2 shows extensive and compact adhesion between the dura mater and the scar, where it is difficult to separate adhesion surrounding the dura mater while keeping the dura mater complete; Grade 3 shows severe adhesion between the dura mater and the scar, and separation means destroying the dura mater [8].

Histological analysis is based on haematoxylin and eosin (H&E) staining and Masson's trichrome staining. H&E staining focuses on cell activity. Masson's trichrome staining shows inflammatory factor and fibrin. Modified Henderson's grading system is based on epidural fibrosis, abscess, acute inflammation and necrosis, dividing into grade 0 (no fibrosis, no inflammation, no abscess and no necrosis), grade 1 (mild interstitial fibrosis, mixed inflammation (25%), abscess area <2 and necrosis area <2), grade 2 (mild interstitial fibrosis, mixed inflammation (50%), abscess area >2 and necrosis area >2), grade 3 (marked fibrosis collagen formation, mixed inflammation (75%), marked abscess and marked necrosis) and grade 4 (massive fibrosis, massive inflammation, massive abscess and massive necrosis) [9,10].

MRI observations of implant materials after lumbar laminectomy show the size of the material and that the shape changed along with the shape of the dura mater. The remodelling of the material occurs in relation to the postoperative transient shrinkage and expansion of the dura mater. MRI can monitor the state of implant to evaluate the function of implant based on the signal and diameter [11,12].

Current strategies for prevention of epidural adhesion

Various methods have been studied to prevent epidural fibrosis and to reduce the pain, such as developing drugs to reduce the inflammation, modifying surgical techniques, using roentgenotherapy and implanting a barrier between epidural space and its overlying muscles. The current methods used clinically include the minimally invasive technique, the usage of drugs such as mitomycin C [13], dexamethasone [14], hydroxycamptothecine [4], rosuvastatin [15] and non-steroidal anti-inflammatory drugs [16,17], low dose radiation, traditional Chinese drugs and biomaterials such as autologous tissue [12,18] and biodegradable polymeric materials(See Figure 2). Biomaterials have some important characteristics such as large molecular weight, complex structure, wide varieties and extensive biological function [19]. As for implants used as physical barriers, optimal biomaterials have progressively become a primary strategy to prevent epidural adhesion

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