Commentary

How would extracorporeal shockwave therapy possibly promote wound healing in colon anastomosis?

Extracorporeal shockwaves therapy (ESWT) has been in use by urologists for lithotripsy for over three decades. Orthopedic surgeons became their first proselytes with its application in the management of delayed or nonunion of fractures where it is claimed to enhance wound healing.[1,2] It has also been used in soft tissue disorders like tendinitis and plantar fasciitis with claims of resolution of symptoms.[3,4]

How does ESWT promote wound healing as a premise for these observations? One can only surmise for now in the absence of hard-core evidence from elaborate studies to answer these questions. Angiogenesis and fibroplasia may offer answers.

Shock waves in ESWT (a form of energy) are acoustic waves which when travelling through any media are either reflected or absorbed, in parts or in whole, (except when traversing vacuum). Since energy cannot be destroyed (the law of conservation of energy), the absorbed waves have to be transformed to other forms of energy in the body. The advancing compressive positive pressure followed by distracting negative pressures produce oscillations in intervening structures resulting in microscopic structural strain. Unlike the case with the powerful energy from ionizing radiations like x-rays and ultraviolet rays, it is reasonable to assume that the energy absorbed from ESWT is mainly transformed to mechanical and thermal energy just like the way the microwave oven works. The amount of energy released depends on the frequency of the wave, the duration, and the physical characteristics of the medium. It is tantalizing to hypothesize that the resulting energy released could cause sublethal injury to cells. The elasticity of soft tissues might make them resilient to mechanical strains (recall the safety profile of lithotripsy on surrounding soft tissues).

What if neural sensors in the tissues exposed to ESWT are tricked into initiating an inflammatory response despite the absence of lethal cell injury?

The axon reflex initiating the inflammatory response stimulates monocytes/macrophages to release cascades of biochemical signals to elaborate chemokines and growth factors locally to enhance angiogenesis and fibroblast activity.

Of the several known neuropeptides linked to inflammatory angiogenesis, two deserve mention. Substance-P, elaborated in unmyelinated nerves in response to the axon reflex, has been identified as a key chemical inducing angiogenesis by acting on the Neurokinin-1receptor. It also upregulates the production of the angiogenic growth factor fibroblast growth factor-2 in coronary venular endothelial cells, an effect that is thought to be mediated by nitric oxide.[5] Substance-P has also been implicated in the induction of monocytes/macrophages to elaborate the angiogenic factors, tumor necrosis factor-alpha, IL-1, IL-6, IL-8, IL-10, and histamine.[6,7]

The second neuropeptide is calcitonin gene-related peptide. Apart from its ability to stimulate monocytes/macrophages, it is pro-angiogenic in human placenta and has been demonstrated to enhance skin graft survival both by its powerful vasodilatory effect and by an independent angiogenic effect.[8,9]

Just imagine the advantages that will accrue from harnessing this response in injured tissues with deficient healing capacity. Colorectal surgery could use this boost to the advantage of the patient.

Leakage of anastomosis on the colon (an ever-present risk!) can be the product of poor operative techniques, but even the seasoned operator may rarely perform a faultless anastomosis and still end up with leakages. Some patients may have to be operated emergently with coexistent risk factors. Debility from unrelieved obstruction, septicemia, diabetes, uremia, morbid obesity, malnutrition, hypoalbuminemia, hypoxemia, and steroid therapy may militate against the healing process. Sustained intraoperative hypovolemia, intra-abdominal hypertension, and postoperative chemotherapy are added dimensions that will tip the scale in favor of sepsis and tissue hypoxia, leading to multisystem organ failure and death. If ESWT promotes healing, then research in this area should be much more than a clinical curiosity and should be rigorously pursued. The study in this issue of the journal to which this article is an opening act is an interesting attempt to push therapeutic frontiers and open the doors of clinical inquiry. Reproducing this result by independent teams will doubtlessly be profitable.

What is the optimal dose of ESWT that would provide optimal results? Would a single large dose administered intraoperatively have comparable results as fractionated postoperative doses administered transabdominally as used in this study?

In medicine, as in business, nothing ventured, nothing is gained. We need fresh ideas and people with explorative minds to think outside the box to steer us to limitless possibilities and potentials as is often the case in the profession. If the mistakes of the past like the scandalous delay that trailed the introduction of hysteroscopy by gynecologists by over two decades until the revolutionary advent of laparoscopic surgery in the 1980s are to be avoided, then the potentials for ESWT in promoting wound healing should be explored further without delay.

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