Mesotherapy in the Treatment of Localized Fat Deposits

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Abstract

In the past few years the treatment of localized fat deposits with subcutaneous injections of phosphatidylcholine (PC) and sodium deoxycholate (DC) has been popularized. The therapeutic efficacy of these materials is based on discoveries from the intravenous use of these substances in the treatment of hyperlipidemia.

A substantial number of physicians have asserted that this treatment can diminish areas of fat by disrupting fat-storing adipocytes. Until recently, there have been no histological studies to confirm this finding. This is a review of the recent literature and a proposal of a hypothesis for the actions of these materials.

Introduction

Mesotherapy represents a broad area of medical treatment related to treating tissues derived from the mesoderm.[1] The term mesotherapy has also been used to describe the treatment of localized fat deposits, an incorrect definition that nonetheless has become part of the lexicon in North America. While such treatments are a form of mesotherapy, more-specific terminology is needed for proper definition.

The use of mesotherapy techniques to treat localized areas of fat was first demonstrated by Rittes.[2] Based on work by others[3,4] suggesting the ability of PC to lower cholesterol and triglycerides, Dr Rittes utilized PC/DC subcutaneously. Both of these substances act to emulsify fat. PC, derived from soy, is also known as lecithin, and DC is an important emulsifier in bile that is stored within the gallbladder. DC is also utilized as a solubilizing agent with drugs such as amphotericin.

Dr Rittes published her initial work in 2003 and described treating various areas. She used PC and DC in a mixture of 50 mg/cc PC and 42 mg/cc DC. Since her initial work was published, many physicians have learned about her approach and have used the compounds to treat areas of localized fat. Although many physicians have found that the treatments are effective, it has been unclear what the mechanism of action is and what actually occurs to the fat cells. In vitro studies by Rotunda and Ablon have shown that adipocyte metabolism is adversely affected by PC and DC.[5] Their work also suggested that DC may be the compound most responsible for the action on fat when treated with the PC/DC mixture.

In 2005 Rose and Morgan published a study revealing the histological changes associated with the use of PC/DC injected into subcutaneous fat.[6] The study involved obtaining biopsies pre- and post-treatment with PC/DC. The area treated was the flanks of a male patient. A 4-mm punch biopsy was taken from the untreated side, and the other side was treated with PC/DC at 42 mg/cc and 21 mg/cc DC (Kronos Pharmacy, Las Vegas, Nevada). A biopsy was taken from the treated side at 1 and 2 weeks post-treatment.

The tissue was fixed in formalin, and paraffin sections were obtained and stained with hematoxylin and eosin. Microscopic evaluation of the tissue sections demonstrated alteration of

the fat cells at 1 week. The fat cells were disrupted and elongated, forming ovoid shapes. In some instances the fat cells coalesced to form cystic-appearing structures. Concomitantly there was a profound inflammatory component consisting of lymphocytes, neutrophils, plasma cells, and macrophages. The inflammatory reaction surrounded the adipose structures and could be found within the septae as well.

Examination of the 2-week biopsy showed a change in the inflammatory reaction. The population of reactive cells was predominated by histiocytes and lymphocytes. The phages macro contained visible fat inclusions, and a pattern of fat necrosis was evident. This pattern was akin to changes observed with trauma, cold in the newborn, radiation, and lobular panniculitis.

Hasengschwandtner has studied the physiology of this process, documenting an enzyme cascade that is involved in fat degradation.[7] He has noted that the process of degradation persists for approximately 10 weeks. A portion of the fat is apparently metabolized in the liver via high-density cholesterol (HDL).

Discussion

The use of PC/DC to treat localized areas of fat is increasing. Many physicians and healthcare professionals believe that these compounds work to alleviate fat; however, there has been a lack of basic science data to support this conclusion, and the mechanism of the loss of fat is not yet completely understood. Work done by Rotunda provides in vitro evidence that PC and DC can act to impair fat cells.[5]

We have documented the effects of PC/DC in vivo and demonstrated important histological changes. These changes include the actual destruction of fat cells, proving that PC/DC can be used to diminish areas of fat. This fact also counters the theory that the fat cells actually release fat and re-seal after fat has moved out of the cells.

The evidence supports the destruction of fat cells accompanied by an inflammatory process. The inflammatory process may act to further disrupt fat cells or their transport or to engulf the fat. The inflammatory response may occur as a reaction to the release of fat by the detergent effects of PC/DC and the irritant reactions caused by the fat and its by-products. One would expect that the inflammatory response would also be accompanied by the release of various lymphokines and cytokines that could affect the removal and transport of the free lipids. It is thought that some of the fat is picked up by the bloodstream, while part of the lipid debris is taken up by macrophages and further digested. Like other traumatic injuries, the inflammatory response may continue for a prolonged period of time, lasting weeks or perhaps longer.

The authors hypothesize that as the inflammatory reaction diminishes, there may be an influx of fibroblasts or dermal dendrocytes into the area. These cells normally play a role in wound repair and/or scarring, typically producing collagen. These cells and their products may result in localized scarring and remodeling of the subcutaneous fat, producing the salutary effects clinically observed following treatment.

References

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