DIVERTICULAR DISEASE AND OXIDATIVE STRESS: THE LINK WITH MITOCHONDRIA ALTERATIONS IN THE DIFFERENT CLINICAL PHENOTYPES

Lucia Pallotta

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A laboratory study of surgical specimens from diverticular disease patients demonstrates mitochondrial dysfunction and oxidative stress in longitudinal smooth muscle, with severity correlating to disease stage and partial reversal by polyphenol treatment in vitro.

KEY POINTS

- Oxidized protein content was elevated in both uncomplicated diverticula (5.0-fold) and complicated stenotic diverticular disease (2.6-fold) compared to controls, with progressive loss of antioxidant thioredoxin (70.9% and 83.3% decrease respectively).
- Mitochondrial complex II succinate dehydrogenase decreased by 43.4% in uncomplicated and 67.9% in complicated disease, while compensatory increases in complex IV cytochrome c oxidase and mitochondrial mass occurred, most prominently in complicated disease.
- Cellular glutathione content progressively decreased (51.9% in uncomplicated, 72.2% in complicated disease), associated with impaired smooth muscle contraction (approximately 51-52% reduction) and relaxation (approximately 35-38% reduction) in both phenotypes.
- Twenty-four hour incubation with 14mM polyphenols restored glutathione content and relaxation function in cells from both disease phenotypes.
- This material would interest researchers investigating diverticular disease pathophysiology and potential antioxidant therapeutic strategies.

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