Surgical Approaches to Uveitis

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Arachidonic acid cascade and platelet-activating factor in the network of eye inflammatory mediators: therapeutic implications in uveitis

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Abstract

The cellular and biochemical events triggered by uveitis involve a complex array of cells and a heterogeneous network of mediators of intraocular inflammation. Resident cells are activated and inflammatory cells are recruited. Chemical mediators from the arachidonic acid cascade, prostaglandins, hydroxyeicosatetraenoic acids, and leukotrienes, are formed. Several of these metabolites are modulators of cellular functions, but when generated in sustained, excessive amounts, they contribute to enhanced vascular permeability and to the onset of pathophysiological responses. Another very active membrane-derived mediator is platelet-activating factor. This important mediator of immune and inflammatory responses may play a central role in uveitis due to cell priming, since interleukin-1, tumor necrosis factor, and other as yet unidentified mediators are also being generated. The concomitant accumulation of these networks of mediators in various parts of the uveal tract leads to spreading of the intraocular inflammatory response and cellular damage. At both early and late stages of uveitis, the generation of free radicals is also a major contributor to the impairment of function. Free radicals are generated in two distinct sites: in the oxidative burst of recruited white cells and in free radical formation and lipid peroxidation in resident cells. The identification of the cellular events that lead to the accumulation of networks of mediators of inflammation and their effects has important therapeutic implications in uveitis.

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