this study demonstrates a clear preference for the EpiPen auto-injector over the Twinject device. Future studies with larger numbers will be helpful to further substantiate these findings.

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REGULATION OF MONOCYTE CHEMOTACTRANT PROTEIN 1 BY CYSTEINYL LEUKOTRIENE D4 IN HUMAN LUNG EPITHELIAL A549 CELLS

Cysteinyl leukotrienes (CysLTs) are proinflammatory lipid mediators that play an important role in eosinophilic airway inflammation, specifically through bronchoconstriction, mucous secretion, and airway hyperresponsiveness via G-protein–coupled receptors and CysLT receptor 1 (CysLTR1).1 It has been reported that CysLTs may induce monocyte chemoattractant protein 1 (MCP-1) production, and this induction is mediated by mitogen-activated protein kinase and nuclear factor-κB pathways via CysLTR1 in human monocytes and macrophages.2 However, to our knowledge, no published studies have addressed how MCP-1 may be regulated by CysLTs in human epithelial cells considering that airway epithelial cells play a major role in the pathogenesis of asthma.3

The aims of this study were to investigate (1) the effect of CysLT D4 (LTD4) on MCP-1 production in human lung epithelial A549 cells and (2) the inhibitory effect of the CysLTR1 antagonist MK571 on MCP-1 production. Human lung epithelial A549 cells were incubated with interleukin 4 (IL-4) (Sigma-Aldrich Corp, St Louis, Missouri) for 24 hours and then were treated with various concentrations of LTD4 (Sigma-Aldrich Corp) for 6 hours. Expression of CysLTR1 was then investigated using realtime reverse transcriptase–polymerase chain reaction (for messenger RNA) and flow cytometry (for surface expression). Production of MCP-1 was examined using a CC chemokine ligand 2/MCP-1 Quantikine enzyme-linked immunosorbent assay kit (R&D Systems Inc, Minneapolis, Minnesota). The dependency of CysLTR1 on LTD4 for MCP-1 production was examined via pretreatment with the CysLTR1 selective antagonist MK571 (Sigma-Aldrich Corp) or the nonselective antagonist BayU9773 (Sigma-Aldrich Corp) for 30 minutes.

The IL-4–primed (10 ng/mL) A549 cells showed increased CysLTR1 expression in messenger RNA and protein (Fig 1A). Secretion of MCP-1 was also increased by IL-4 priming in A549 cells (Fig 1B). In IL-4–primed A549 cells, LTD4 (100nM) enhanced MCP-1 secretion (Fig 1C). The IL-4–primed A549 cells were treated with the CysLTR1–specific antagonist MK571 at various concentrations to determine whether regulation of MCP-1 by LTD4 is mediated through CysLTR1. Secretion of MCP-1 was specifically inhibited by 10nM MK571 but not by 100nM BayU9773 (Fig 1D).

Epithelial cells are the major target cells and the effector cells that induce inflammation in the asthmatic airway. In this study, a T1/2 environment with IL-4–induced CysLTR1 expression and MCP-1 production in bronchial epithelial cells. Secretion of MCP-1 from IL-4–primed A549 cells was further increased after stimulation with LTD4. The upregulation of CysLTR1 expression by IL-4 can be explained by a signal transducer and activator of transcription 6 element localized in its promoter region1 that may promote the proinflammatory effects of CysLTs. Increased production of MCP-1 in bronchial epithelial cells may promote infiltration of eosinophils,3 which may lead to chronic inflammation in the asthmatic airway. We also noted the inhibitory effect of MK571 on MCP-1 secretion in bronchial epithelial cells. This may highlight the role of a CysLTR1 antagonist capable of suppressing epithelial cell–derived monocyctic and eosinophilic inflammation in the asthmatic airway.

In conclusion, CysLT signaling through CysLTR1 might contribute to the inflammatory reaction of MCP-1 by cooperating with IL-4, which contributes to chronic inflammation in the asthmatic airway. The CysLTR1 antagonists may have a beneficial role in this pathway.

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Anaphylactoid reactions to gadolinium contrast media are rare, with the current literature reporting an incidence of 0.01%. We report a case of a severe anaphylactoid reaction to gadoteridol.

A 58-year-old nonatopic woman with a history of breast cancer and hypertension, treated with a β-blocker drug, underwent magnetic resonance imaging of her abdomen with her first exposure to gadolinium. She received an injection of ProHance gadoteridol and immediately developed nausea and vomiting. Shortly thereafter, she became unresponsive and experienced respiratory and cardiac arrest. Cardiopulmonary resuscitation was initiated, and she was intubated (with noted difficulty exhaling). She received a total of 4 mg of epinephrine and 1 mg of atropine for pulseless electrical activity before her pulse was detected. On physical examination, she had no urticaria, angioedema, wheezing, or laryngeal edema. The patient had negative results on a workup for other causes of pulseless electrical activity, including a cardiology evaluation. Laboratory test results were significant only for transient metabolic acidosis due to lactic acidosis from the prolonged hypoxia. Her total serum IgE level was 98.6 IU/mL. A serum tryptase level was not obtained. The patient recovered quickly and was extubated the same day. She was told to avoid all gadolinium agents in the future and was referred to the Allergy Immunology Clinic at the VA Greater Los Angeles Healthcare System, Los Angeles, California. Confirmatory skin testing to gadolinium was considered and was not performed because of the severity of the patient’s reaction and her β-blocker medication.

The severity of our patient’s reaction is seen in comparison to the adverse reactions to gadolinium in 2 previously reported case series. Most of the reactions in these 2 case series were mild

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