

## Cytokine Profile of Bilateral Pseudocyst of the Auricle

Sir,

Pseudocyst of the auricle (PA) occurs on the upper part of the ear with smooth surface as the result of trauma, mechanical stimuli or infection. Although the pathomechanism(s) of the blistering process in PA is still unknown, an overproduction of glycosaminoglycans by repeated minor trauma or mechanical stimuli to the ear cartilage is suggested to be the primary stage of this disorder (1). Recent investigations have revealed that various cytokines are released from cartilages, fibroblasts, activated lymphocytes or monocytes. We examined the cytokine profile of the blister fluids and sera of a patient with bilateral PA and 2 patients with solitary PA.

Sera and blister fluids were collected from a 45-year-old male with bilateral PA, a 64-year-old male with solitary PA, and a 49-year-old female with solitary PA, who were diagnosed by clinical and histopathological examinations, before treatment. Specimens were stored at  $-80^{\circ}\text{C}$  until use. Interleukin-1 $\beta$  (IL-1 $\beta$ ), IL-2, IL-6 and tumor necrosis factor- $\alpha$  (TNF- $\alpha$ ) were measured by ELISA technique. In normal sera, cytokine levels were as follows: IL-1 $\beta$  < 10.0 pg/ml; IL-2 < 0.8 U/ml; IL-6 < 4.0 pg/ml; TNF- $\alpha$  < 5.0 pg/ml.

We demonstrated a marked increase of IL-6 and a slight increase of IL-1 $\beta$  in the blister fluid in the case of bilateral onset, while serum levels were within normal range (Table I). IL-6 was also elevated in the two solitary PA fluids. Cartilage degeneration is thought to be further accelerated by various cytokines released by the resident mesenchymal cells, the infiltrating neutrophils, monocytes or macrophages (2). IL-1 is the most important mediator of inflammation and cartilage destruction, which increases the synthesis of proteases and PGE<sub>2</sub> by chondrocytes, and inhibits the formation of extracellular matrix components (3). IL-6 is induced by IL-1 and is a stimulator of chondrocyte proliferation (4). Furthermore, analysis of the LDH isozyme pattern of the blister fluids revealed an increase in LDH4 (31.6% in lt BF, 23.2% in rt

Table I. Cytokine profiles of sera and blister fluids of patients with pseudocyst of the auricle

Patient	Serum/ blister fluid	IL-1 $\beta$ (pg/ml)	IL-2 (U/ml)	IL-6 (pg/ml)	TNF- $\alpha$ (pg/ml)
45/M	S	<10.0	<0.8	<0.4	<5.0
	BF (lt)	18	<0.8	1740	<5.0
	BF (rt)	15	<0.8	3600	<5.0
64/M	S	ND	ND	ND	ND
	BF	<10.0	ND	1010	ND
49/F	S	ND	ND	ND	ND
	BF	<10.0	ND	2830	ND

BF) and LDH5 (51.7% in lt BF, 42.5% in rt BF) isozymes, while the serum isozyme pattern exhibited a dominance of LDH1 of 25.3% (LDH4; 9.8%, LDH5; 18.8%) in the bilateral case. The LDH isozyme pattern of articular cartilages demonstrates mostly LDH4 and LDH5 isozymes (5). It is suggested that the release of cytokines is derived from infiltrating inflammatory cells or damaged cartilages, which play a role in the formation of PA.

## REFERENCES

1. Choi S, Lam K, Chan K, Ghadially FN, Anthony SMN. Endochondral pseudocyst of the auricle in Chinese. *Arch Otolaryngol* 1984; 110: 792-796.
2. Dinarello CA. Interleukin-1 and other growth factors. In: Kelley WN, Harris ED, Ruddy S, Sledge CB, eds. *Textbook of rheumatology*. 3rd edn. Philadelphia: W. B. Saunders, 1989: 285-299.
3. Mizel SB, Dayer JM, Krane SM, Mergenhagen SE. Stimulation of rheumatoid synovial cell collagenase and prostaglandin production by partially purified lymphocyte activating factor (interleukin-1). *Proc Natl Acad Sci USA* 1981; 78: 2474-2477.

4. Guerne PA, Lotz M. Regulation of Swarm rat chondrosarcoma cell proliferation: synergy between interleukin-6 (IL-6) and transforming growth factor- $\beta$  (TGF $\beta$ ). *J Cell Physiol* 1991; 149: 117-124.
5. Tushan F, Rodnan GP, Altman M, Robin ED. Anaerobic glycolysis and lactate dehydrogenase (LDH) isozymes in articular cartilage. *J Lab Clin Med* 1969; 73: 649-656.

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