Medical Journal of the
Islamic Republic of Iran

Volume 3
Number 1,2
Bahar & Tabestan 1368
Spring & Summer 1989

MAST CELLS IN THE TYMPANIC PART OF THE FACIAL NERVE IN CHRONIC OTITIS MEDIA WITHOUT ASSOCIATED FACIAL PARALYSIS

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ABSTRACT

The occurrence of mast cells is studied in the normal facial nerve and in the facial nerve post mortem exam of patients with chronic suppurative otitis media without associated facial paralysis. A small number of mast cells were found in the normal facial nerve. These cells were usually located in close proximity to the endoneural and epineural blood vessels. The number of mast cells was increased in some areas of the facial nerve in the majority of examined cases with chronic otitis media. In these cases, although the facial nerve showed some signs of neuropathy, its function was normal.

MJIRI, Vol.3, No1&2, 21-25, 1989

INTRODUCTION

Mast cells have been demonstrated in peripheral nerves in various pathological conditions including traumatic injuries,[1] metachromatic leukodystrophy, and globoid cell leukodystrophy,[2,3] diabetic neuropathy,[4] von Recklinghausen's disease,[5] and some tumors of the peripheral nerves.[6]

A number of experimental studies have been performed in order to demonstrate whether the mast cells react and participate in different pathological conditions of the peripheral nerves.[7,8,9]

It was found that mast cells increased during the course of peripheral neuropathy. It is now well known that mast cells contain specific cytoplasmic granules which store a variety of biologically active amines (histamine, serotonine, heparin), a battery of enzymes (cytochrome oxidase, alkaline phosphatase, esterase),

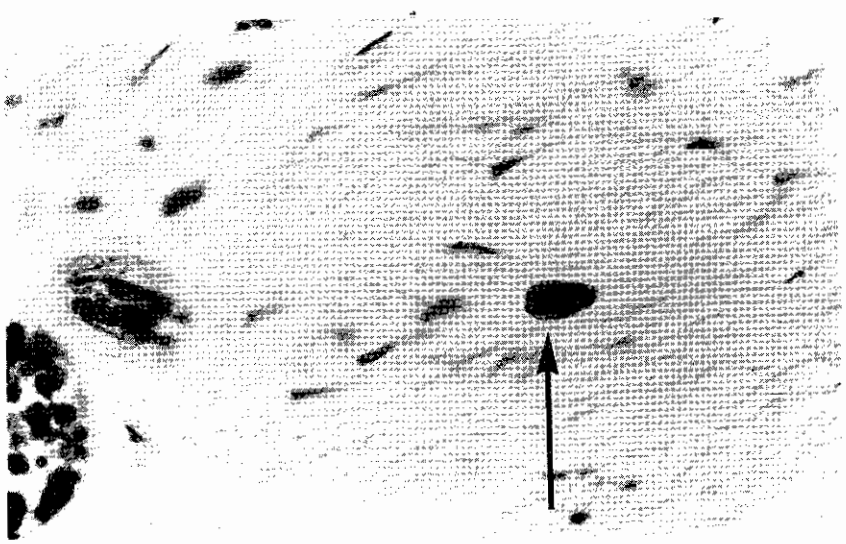

Fig. 1. Epineural mast cell (arrow) in normal facial nerve (Luxol fast blue and cresyl violet, × 200).

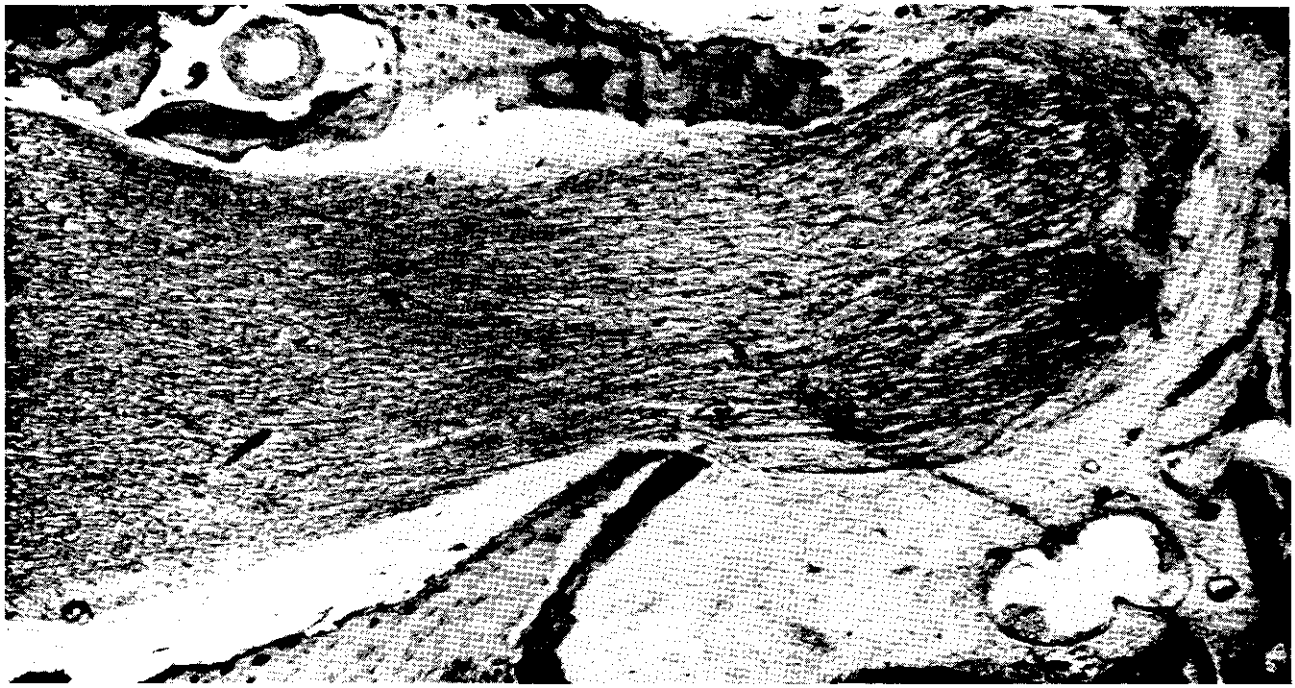

Fig. 2. The exposed facial nerve surrounded by fibrocystic tissue (HE × 45).

mucopolysaccharides, and other compounds.[10] After release from the mast cells to the peripheral nerves, these products may have a different effect on structural elements of the nerve.

The present study was carried out in order to investigate the occurrence of mast cells in the facial nerve in chronic suppurative otitis media without associated facial paralysis. To our knowledge, there have been no descriptions of mast cells in the facial nerve in any pathological conditions and there have been no examinations of mast cells in the facial nerve in chronic inflammation of middle ear in the literature.

MATERIALS AND METHODS

From the collection of temporal bones at the Massachusetts Eye and Ear Infirmary in Boston, two groups of temporal bones were selected. The control group included nine normal temporal bones which had been obtained from subjects with no history of ear or facial nerve diseases. The study group included 20 temporal bones from subjects who had chronic suppurative otitis media without associated facial paralysis. Selection of these temporal bones was based on the following criteria: bones which were obtained from subjects who suffered from facial nerve injury or a systemic disease which could lead to pathologic changes in the facial nerve, were omitted from the study. The ages of persons at death in both groups ranged from 12 to 90 years. The time from death to fixation of the temporal bones ranged from two to 26 hours.

The temporal bones were prepared for light microscopic study by method of decalcification, embedding in celloidan, serial sectioning at 20 micron slices, staining every tenth section with hematoxylin and eosin and mounting on glass slides. These tracer sets of sections were used to estimate bony destruction of the facial canal and to examine the extension of the pathologic process from the middle ear toward the facial nerve. Unstained sections were selected from the tympanic segment of the facial nerve for staining with luxol fast blue and cresyl violet. On these sections the mast cells were identified. The determination of these cells was based on their staining properties as well as their distribution between the nerve fibers. The mast cell population was counted using an ocular grid and a 40 power objective lens and was expressed as the number of mast cells per square millimeter. The population density of mast cells higher than 5 cells/mm^2 was assessed to be increased.

RESULTS

Control group

A small number of mast cells were found in all examined nerves. The mean number of the mast cells was 4 cells/mm^2. Most commonly they were located around the epineural and the endoneural blood vessels

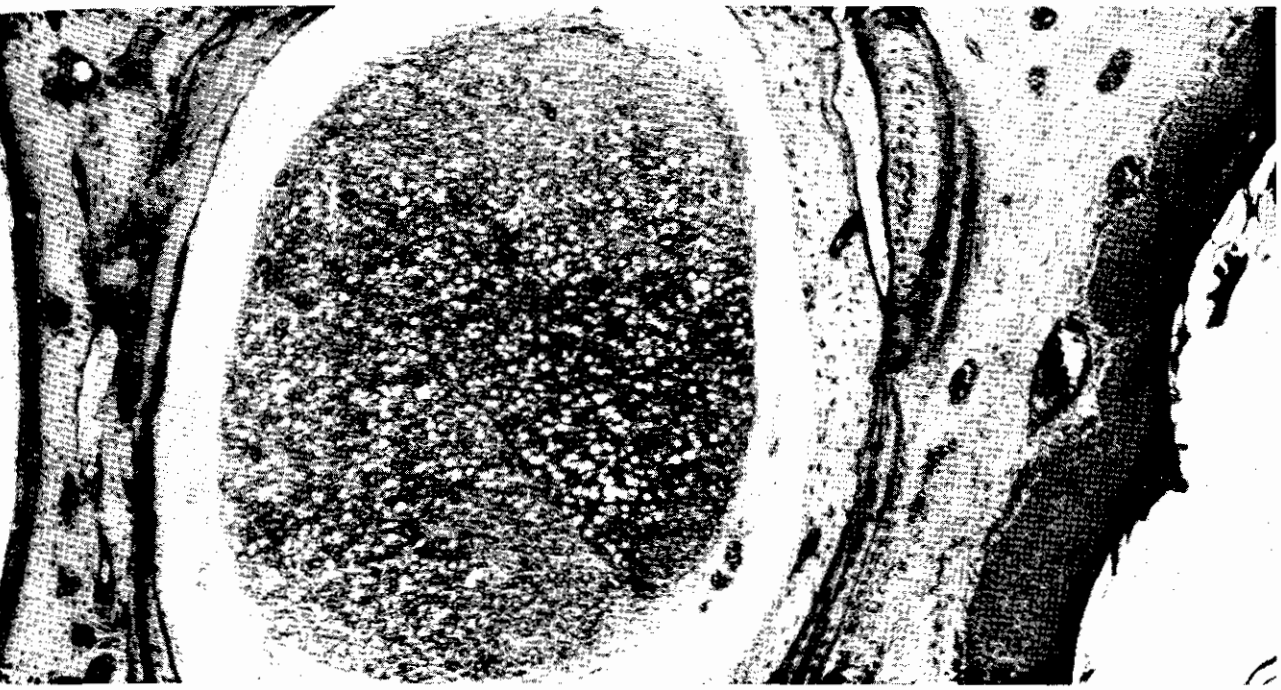

Fig. 3. The exposed facial nerve and destroyed facial canal covered by cholesteatoma (HE × 65).

(Figure 1). There was no obvious difference between the density of mast cells in the endoneurium and the epineurium.

Study group

The tympanic segment of the facial canal showed bony destruction on its lateral wall in all cases. The affected facial canal or the exposed nerve trunk was surrounded by pronounced granulation or fibrocystic tissue (Figure 2), or by cholesteatoma (Figure 3). The epineural tissue of the facial nerve showed a moderate fibrosis in all cases.

The population density of the mast cells was increased in the exposed nerve trunk in 12 of the 20 examined cases. It was found to be in the epineural tissue (Figure 4), or in areas where the nerve showed signs of alterations of the myelin and hypertrophy of the Schwann cells. The number of mast cells ranged from 8 to 16 cells/mm^2. The mean count was 12 cells/mm^2. Most of the mast cells appeared intact. Only in some areas degranulation of mast cells was observed.

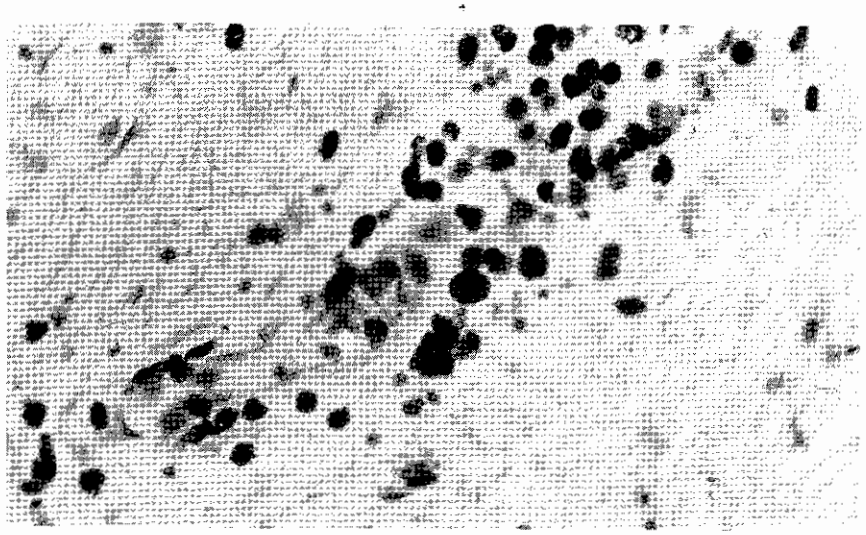

Fig. 4. Numerous rounded dark staining mast cells in the epineural tissue of the facial nerve in chronic otitis (Luxol fast blue and cresyl violet, × 200).

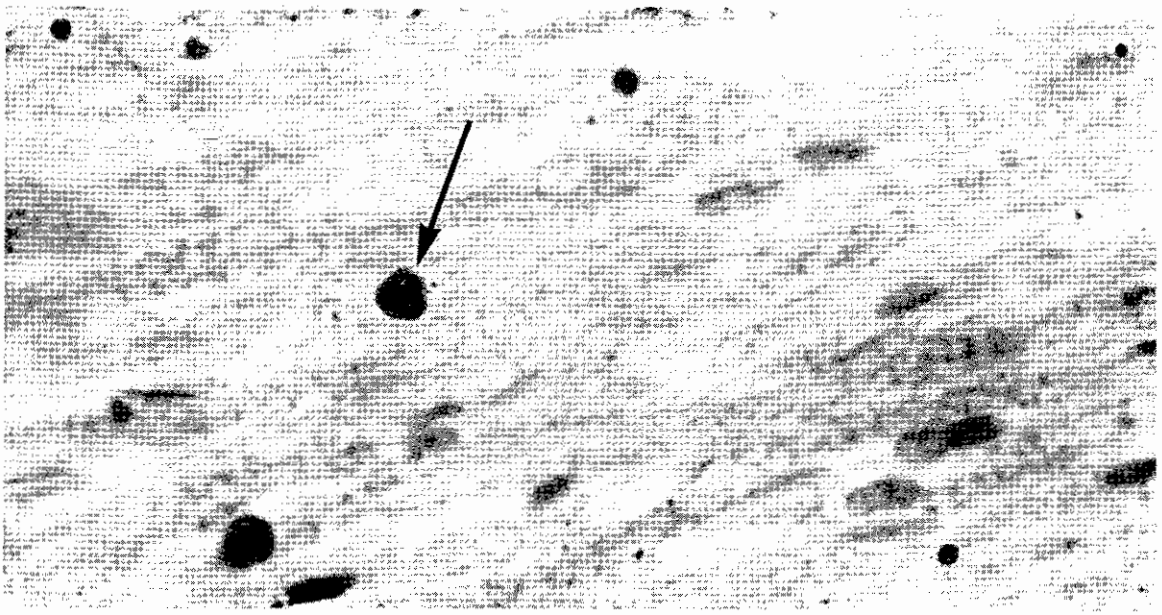

Fig. 5. Numerous rounded dark staining mast cells can be seen in areas of demyelination of the facial nerve from a patient with chronic otitis media. The arrow points out one of them (Luxol fast blue and cresyl violet, × 210).

DISCUSSION

Previous studies have shown that mast cells present as a very reactive and vulnerable structure in the peripheral nerves, which on exposure to various noxious stimuli, such as toxins, trauma or a variety of chemical substances, react with expulsion of their granules and release of their stored products. This reaction of the mast cells occurs within a few minutes after compression of the nerve which does not produce any demonstrable light microscopic changes in the axons or myelin.[1] The effects of most cell products on myelin sheaths and vasa nervorum in lesions of the peripheral nerve trunks may be different.[11,12] A positive correlation between the mast cell degranulation and increased vascular permeability of the vasa nervorum in the peripheral nerves was found. It was also postulated that some products released from mast cells may play a role in the enzymatic breakdown of lipids in the myelin sheaths.[12] In a more recent study, Johnson, et al,[9] observed that the mast cell proteases, released on degranulation, had a high myelinolytic capacity. They pointed out that degranulation of mast calls adjacent to myelin sheaths might serve as focus for inflammatory demyelination.

Our clinical studies have shown that the occurrence

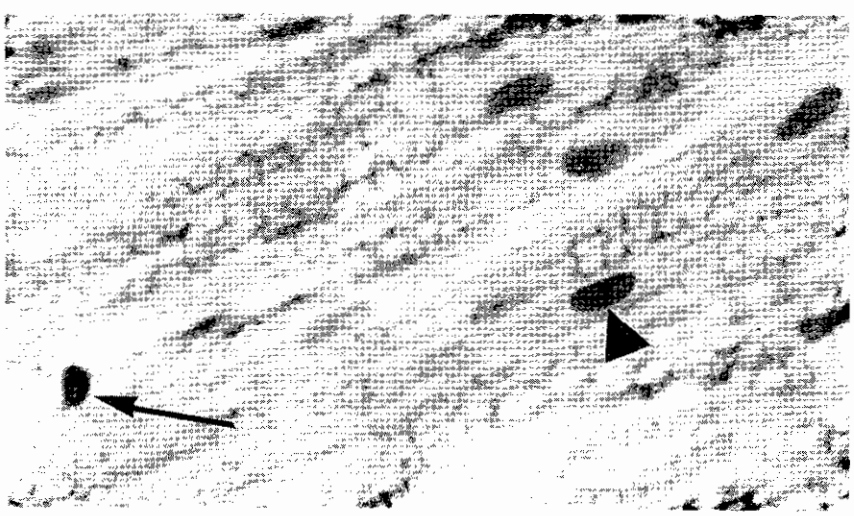

Fig. 6. In this area of the facial nerve from the same patient (Fig.5), only one mast cell can be seen (arrow). Note hypertrophic Schwann cells (▲) (Luxol fast blue and cresyl violet, × 210).

of facial nerve paralysis in chronic otitis media does not depend on the extent of bone destruction of the facial canal.[13] The histopathological studies have demonstrated that the facial paralysis developed when the inflammatory process involved the facial nerve trunk.[14]

The present study demonstrates that mast cells are increased in some areas of the facial nerve trunk in most examined cases of chronic otitis media. In these cases, although there were some evidence of alterations in structural elements of the nerve, its function was clinically normal.

These changes may predispose to the development of facial paralysis during chronic middle ear inflammation.

ACKNOWLEDGEMENT

We are very grateful to Professor Harold F. Schuknecht, M.D., Emeritus Chief of Otolaryngology, Massachusetts Eye and Ear Infirmary, Boston, who provided the samples for this study and for his kind advice in the preparation of this work.

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