Expression of Glial Fibrillary Acidic Protein (GFAP) in the Trigeminal Ganglion of Male Wistar Rats

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ABSTRACT
Background Significance: The pseudounipolar neurons in the sensory ganglia are wrapped by small satellite cells which play a similar role like Schwann cells in the peripheral nervous system. The pseudounipolar cells of trigeminal ganglion are surrounded by a capsule formed by satellite glial cells. These satellite glial cells play important role in maintaining normal functions of the neuron.

Aim & Objective: To localise GFAP in satellite glial cells of trigeminal ganglion.

Material & Methods: Six male wistar albino rats trigeminal ganglion were collected and immunohistochemically stained for GFAP in six male wistar albino rats.

Result: GFAP was localised in the cytoplasm of satellite glial cells surrounding the neurons. GFAP was also localised around Schwann cells of an axon.

Discussion: GFAP is an intermediate protein which can get up regulated due to peripheral axonal injury. This GFAP can trigger mediators of inflammation and create a neuralgia or migraine like conditions.

Conclusion: This study concluded that satellite cells are type of glial cells that express GFAP. This GFAP was also expressed by Schwann cells surrounding the axon.

Keywords: GFAP, Trigeminal ganglion, Satellite glial cells, Pseudounipolar neurons

INTRODUCTION
The trigeminal ganglion (also called the semilunar ganglion or Gasserian ganglion) is mainly composed of cell bodies of pseudounipolar neurons and nerve fibres. The cell bodies, predominantly occupy the peripheral part of the ganglion, while the nerve fibres occupy mainly the central regions. The trigeminal ganglion contains the first order neurons of the somatosensory pathways arising from receptors and free nerve endings in the facial and oral tissues, scalp and part of the duramater.¹ There is various neurological disorders like migraine and neuralgia which are associated with the trigeminal ganglion. Migraine is a major disorder resulting due to the consequences of multiple pathophysiological changes in the meningeal tissues, trigeminal ganglion, trigeminal brain stem nuclei based on specific characteristic of trigeminal vascular system.²

Gliarial cells which surround the pseudo unipolar neurons directly modulate neuronal function and activity by changing the ionic concentrations in and around the neurons.³ Interestingly, neuron-glia interactions have been shown to be involved in all stages of inflammation and pain associated with several CNS diseases.⁴ Gliarial cells express characteristic substances in common with immune cells in which they respond to viruses and bacteria, releasing pro-inflammatory cytokines, which create pathological pain.⁴ GFAP is a member of the cytoskeletal protein family, and the principal 8-9 nm intermediate filaments expressed in mature astrocytes of the central nervous system, and satellite glial cells (SGC) of sensory ganglia.⁵

AIM AND OBJECTIVE
To study, the expression of GFAP in the trigeminal ganglion of male wistar albino rats.

MATERIALS AND METHOD
Male albino Wistar rats (n=6) of weight ranging from 200g to 250g was used for Immunohistochemical localization of GFAP in trigeminal ganglia of each side. The rats were obtained from experimental animal facility of All India Institute of Medical Science after prior approval of the experimental procedure by Institutional Animal Ethics Committee (IEAC). The animals were kept in cages with not more than three animals in one cage. They were maintained at 12hr: 12hr light/dark cycles with water and food available ad libitum

TISSUE COLLECTION AND IMMUNOHISTOCHEMICAL LOCALIZATION
Fixation was done by using 500ml of 4% paraformaldehyde in 0.1 M phosphate buffered
saline, through continuous transcardiac perfusion for a period of 1 hour. Then the skull was cut open and trigeminal ganglion was identified and removed. Then the ganglia were placed in chuck embedded with Optimum Cutting Temperature medium and sectioned using cryostat (20Hm). For each tissue the sections were collected separately in the multi vial culture plates and labelled. For free floating immunohistochemical localization the antibodies for GFAP was obtained from Sigma Laboratories (USA). The dilutions ratio for GFAP (1:400) was determined after repeating the histochemical localization at various dilution ratios. The slides stained for GFAP were captured by ProgRes image capture using JENOPTIK ProgRes Capture Pro 2.7 (Germany) in an E-600 Nikon compound light microscope.

RESULT
Immunolocalisation for GFAP:

a) GFAP was localised in the cytoplasm of satellite glial cells (black arrow).

b) GFAP was also localised in schwann cells (blue arrow).

DISCUSSION
Each neuron in the ganglia is completely surrounded by several satellite glial cells which form a sheath or envelope thus forming a distinct morphological and functional unit. The group of neurons are separated from each other by satellite glial cell sheath with minimal connective tissue between them. This complete glial sheath formed by satellite glial cells around the sensory neuron is unique feature and is not commonly observed in CNS. The satellite glial cell envelope usually consists of flat processes that lie close to the neuronal plasma membrane. The distance between glial cell and neuronal surface is about 20nm and therefore extracellular space between the neuron and satellite glial cell is minimal.

GFAP is important in modulating astrocyte motility by providing structural stability to astrocytic processes. Glial fibrillary acidic protein (GFAP) is the archetypal marker for astrocytes is reported to be present at either low level in the normal satellite glial cells or in activated satellite glial cells. Following peripheral nerve injury, satellite glial cells undergo changes similar to those found in CNS and show marked increase in glial fibrillary acidic protein (GFAP). There is increasing evidence that glia within the spinal cordalsa horn contributes to the maintenance of pathological pain. In chronic constriction injury of the sciatic nerve there was up-regulation of TNF-alpha leading to over expression of GFAP in the dorsal root ganglion neurons. Also, it was noted that this TNF-alpha signalling pathway is the major pathological change leading to inflammatory pain.

Increased GFAP immunostaining was observed in the gray matter of the spinal cord ipsilateral to the lesion and specific to spinal segments in which the sciatic nerve is distributed. Elevated GFAP staining density was attributed primarily to hypertrophy of astrocytes rather than their proliferation or migration since counts of astrocyte profiles demonstrated no significant difference when comparing the lesion to the control side. The magnitude of the increase in GFAP staining correlated with the degree of hyperalgesia.

In the present study glial fibrillary acidic protein (GFAP) was localized around the axons i.e. Schwann cells. This finding is supported by a study where they showed that the peripheral nerve regeneration is disturbed in mice that lack glial fibrillary acidic protein (GFAP), a Schwann-cell-specific cytoskeletal constituent which was up regulated after damage.

CONCLUSION
This study concludes that GFAP is expressed by glial cells of trigeminal ganglion namely satellite cells and Schwann cells. In any peripheral injury these cells can over express GFAP triggering inflammatory mediators and gliosis.

REFERENCES
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