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A Review Progress in Nanoplatforms for the Treatment of Neuropathic Pain

Dhage Shivraj Sopan[1] and Dr. Pramod V Burakle[2]
[1]Research Scholar, Department of Pharmaceutics
[2]Research Guide, Department of Pharmaceutics
Sunrise University Alwar (Raj.) India

Abstract: *The goal was to provide an overview of the research on nanoplatforms and spinal cord injury (SCI) and explain how they may help with SCI studies. Drug therapy is now the main therapeutic treatment for neuropathic pain (NP), however these conventional medications have a number of drawbacks, including high dosage, quick clearance from the circulatory system, cytotoxicity, and off-target adverse effects. Furthermore, the blood-brain barrier makes treating NP more difficult. Nanomedicine has drawn more attention in recent years; this innovative approach may aid in the delivery of medications to treat NP using nanoplatforms, making it a viable substitute treatment.*
By preventing quick blood clearance or guaranteeing sufficient concentration in the lesion, the use of nanoplatforms may improve pharmaceutic efficacy. Nanoplatforms that have been reported in experimental investigations of neuropathic pain were the main focus of a review of the literature. RESULTS: We go over the potential development of the nanoplatform system for NP and provide a short overview of the functions of liposomes, polymeric nanoparticles, metal nanoparticles, micelles, and dendrimers in the treatment of NP. The development of several nanoplatform drug delivery methods may be a useful resource for the prompt identification and efficient management of NP associated with SCI.

Keywords: Nanoplatforms, Neuropathy, Analgesic, Nanocarriers, Liposome, Nanoparticles, Biocompatibility.

I. INTRODUCTION

With a prevalence rate of approximately 7–8% in the general population, neuropathic pain is caused by a lesion or disease that affects the somatosensory component of the nervous system. It is characterized by either positive or negative sensory signs and symptoms, such as burning sensation, induced pain, and abnormal temporal summation. Furthermore, NP, a prevalent chronic pain syndrome that significantly lowers overall quality of life, affects about 80% of individuals with spinal cord injury (SCI), with 40% of those cases being classified as severe NP. SCI often results from localized distortion of the spine brought on by mechanical harm, direct compression, and injury to blood vessels and nerve components from fractures, displaced bone fragments, or disc debris. While at-level pain might comprise both peripheral and central NP, below-level pain is a central NP condition. SCI encompasses both at- and below-level SCI-based NP.

Reactive oxygen species (ROS) concentrations, intracellular and extracellular glutamic acid neuropeptide adenosine triphosphate proinflammatory cytokines, and hyperactivity of neurons and glial cell activation may all result from SCI. These elements cause synaptic circuit disruption and spinal cord maladjustment, which intensifies spinal dorsal horn pain and results in NP that is permanent. Patients with SCI-based NP continue to have poor treatment outcomes despite the development of several therapy regimens for NP. These patients also continue to experience pain-related anxiety, depression, and sleep difficulties. Anticonvulsants like lamotrigine, pregabalin, gabapentin, tricyclic antidepressants, selective serotonin–norepinephrine reuptake inhibitors, lidocaine, capsaicin, tramadol, potent opioids (morphine and oxycodone), and botulinum toxin A are currently the standard pharmacological treatments for NP.

The therapeutic effectiveness of these traditional analgesics is limited, and some of them such as tricyclic antidepressants, anticonvulsants, and stronger opioids may cause common central adverse effects including fatigue and

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lightheadedness, which might increase the risk of falls. Patients with peripheral local NP respond only temporarily to lidocaine and capsaicin 8% patches, and most non-invasive medication delivery methods are unable to reach the central nervous system (CNS) because to the unique characteristics of the neurovascular system. Additionally, the majority of these medications have low water solubility. Therefore, in order to overcome these issues and create anti-NP targeted therapeutics, it is imperative to investigate new and effective alternative therapeutic strategies.

A nanoplatform drug delivery system for the treatment of NP may be developed thanks to developments in biochemistry and nanotechnology. Drugs may be delivered via nanoplatforms by lysing, implanting, encapsulating, or attaching to nanoparticles, according to research. Central inflammatory areas may be targeted by nanoparticles, which are made of natural or synthetic polymers and range in size from 1 to 1000 nm. Additionally, the addition of active targeted molecules to nanoparticles enhances therapeutic effectiveness, facilitates drug distribution, facilitates transport across the blood-brain barrier, and eliminates undesirable offtarget effects. Table 1 introduces and describes the latest developments and advancements in drug delivery nanoplatforms for NP therapy. For the treatment of NP, a variety of nanocarrier types are being researched and produced, including as dendrimers, metal nanoparticles, liposomes, polylactic-coglycolic acid (PLGA) nanoparticles, nanomicelles, and nanocapsules. The functions of liposomes, polymeric nanoparticles, metal nanoparticles, micelles, and dendrimers in the treatment of NP are therefore briefly described in this study. Additionally, the future development of the nanoplatform system for NP, particularly SCI-based NP, is discussed.

II. RESEARCH ADVANCES IN NANOPLATFORMS FOR THE TREATMENT OF NP POLYMER NANOPARTICLES POLY (LACTIC-CO-GLYCOLIC ACID) (PLGA)

PLGA has garnered significant interest because to its biodegradability and biocompatibility, controlled drug release, ability to change the surface for disease diagnostics and therapy, and protection of the drug or gene from decomposition. Because of its nanosize, PLGA is the most popular drug delivery method and can effectively penetrate tissues.

Because of its many benefits, including continuous intracellular drug release, cheap cost, simplicity of manufacture, safety, and a controlled rate of degradation, PLGA has emerged as a viable nanoplatform for the treatment of NP. Long-term oxaliplatin usage may cause NP by suppressing the expression of Thioredoxin Interacting Protein (TXNIP) in sciatic nerve cells, which control autophagy. By triggering an inflammatory response via the inflammatory body axis, TXNIP causes NP in microglia. Liu et al. employed the double emulsion solvent diffusion approach to create a PLGA-metformin nanoemulsion, which targets TXNIP by employing PLGA as a carrier and metformin as a cargo.

The nanoparticle may also raise the threshold of mechanical prickling and heat sensitivity, reduce NP, block neuropathic sensitivity, and cause a notable elevation of TXNIP and autophagy-related markers. Because it can break down mRNA molecules by creating RNA-induced silencing complexes to disrupt the expression of certain protein-coding genes, gene therapy based on siRNA silencing has gained interest recently due to its therapeutic potential in a variety of disorders. These molecules may be totally eradicated since they are unable to cross the blood–brain barrier and reach their CNS destinations. In order to successfully embed hydrophilic P38 siRNA in PLGA nanoparticles, Shin et al. created P38 siRNA-loaded PLGA nanoparticles using the conventional double emulsion approach (W/O/W).

Following p38 siRNA-PLGA nanoplatform application in a solid lipid nanoparticles (SLN) mouse model, p38-related inflammatory gene expression, including TNF-a, was altered. This led to the loss of p38 phosphorylation in spinal dorsal horn microglia, which inhibited microglia activation and reduced NP. The siRNA-PLGA nanoplatform has the potential to penetrate the central nervous system (CNS), concentrate p38 siRNA in spinal cord microglia, and target genes unique to microglia that are important in NP. According to different research, CX3CL1 and its receptor CX3CR1 may also be significant and potential new targets for the therapy of NP. Similarly, spinal microglia cells need a drug delivery method that can efficiently distribute CX3CR1 siRNA. Using ultrasound and the traditional double emulsion process (W/O/W), Noh et al. created PLGA-coated CX3CR1 siRNA nanoparticles, which they then administered intrathecally to mice (Fig. 1).

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Mice treated with CX3CR1 siRNA-PLGA nanoplatform showed a considerable decrease in microglial activation in the spinal dorsal horn, a significant downregulation of proinflammatory mediators, and a significant alleviation of mechanical pain. According to these investigations, by lowering microglial activity and proinflammatory mediator expression, PLGA-encapsulated CX3CR1 siRNA nanoparticles may successfully mitigate SNL-induced NP in mice models. PLGA nanoparticles have shown exceptional benefits in improving the targeting of several medications to the central nervous system, particularly microglia cells.

A range of therapeutic drugs and very precise microglial markers may embed it, enabling completely focused treatment without affecting other vital functions. The encapsulation efficiency and siRNA delivery capacity of PLGA nanoparticles are inferior to those of recombinant viruses, which is one of their drawbacks. Hering et al. tested the toxicity of polymer nanoparticles using the fish embryo toxicity test and found that PLGA still exhibited some harm, although very little. The PLGA terminal cap had a substantial impact on the drug loading rate, and the ester-capped group had a greater encapsulation while the acid end group had a lower drug packing efficiency. Lagreca et al. also discovered that a number of other factors influence the drug loading of PLGA. As a result, there are stringent manufacturing criteria. However, in the precise treatment of NP, PLGA may be a viable and efficient drug delivery nanoplatform.

III. CONCLUSION

We compiled a list of many nanoplatforms for NP therapy that are essential in resolving issues with the present single-drug approach to NP, particularly NP associated with SCI. These may lessen the buildup of medications in non-target nerve tissues and more effectively transfer medications to the CNS target, hence lowering a number of harmful side effects. To better use nanoplatforms for NP, various issues still need to be resolved, including neurotoxicity, decreased blood–brain barrier crossing rate, inadequate drug circulation time in the body, and instability of nanoparticles. Moreover, polymeric PLGA has disadvantages including inadequate drug loading and possible chronic-systemic toxicities, whereas silver nanoparticles may lower transendothelial resistance and destroy the blood–brain barrier.

Like PLGA, lipid-based nanoparticles have drawbacks such as inadequate biodistribution, the potential for toxicity, and the possibility of inciting an immunological reaction. Surface-modified metal nanoparticles readily penetrate the blood–brain barrier. As a consequence, they have a tendency to build up in the brain, particularly in the cortex and hippocampus, which lowers glutamate absorption by astrocytes. This may have a negative impact on brain function and raise the risk of neurodegenerative processes. Micelles' short shelf life, poor stability, and interbranch repeatability restrict their usage. The poor loading capacity of lipophilic medicines and their unstable properties are among the disadvantages of lipid-based nanoparticles that are addressed by nanocapsules.

Nevertheless, nanocapsules have a poor mechanical system stability and a complicated production procedure. Dendrimers' therapeutic efficacy is diminished by a number of drawbacks, including their quick clearance by the reticuloendothelial system, toxicity from the amine-terminated group's contact with the cell membrane, poor transfection effectiveness, and lack of controlled-release behavior.

To guarantee full conversion of nanomaterials into clinical products for the treatment of NP, careful consideration of the design of the nanoparticles, mass production capacity, and standardized characterization are required. This is because variations in the size of nanoplatforms may impact their physiological stability.

Additionally, additional research is required on the toxicity, breakdown products, metabolic pathways, and system performance assessment of nano-drug delivery systems in vivo. To guarantee the use of nanoplatforms in clinical settings, it is essential to design appropriate clinical trials. To show the impact of nanoparticles alone, the foundation of nanoplatforms, clinical trials should establish them as a control group. Individual, personalized, and patient-centered drug delivery system selection should be adhered to. In conclusion, patients with NP, particularly those with SCI-based NP, may find the nanoplatform drug delivery system to be a potential future therapeutic use. In addition to improving NP diagnosis and treatment outcomes for patients with SCI, we anticipate that therapeutic approaches and nanoplatforms will contribute to a new area of pain research.

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