ABSTRACT

Intraneural Ganglion Cysts [IGC] are a set of medical conditions that result in denervation of the muscles innervated by the cystic nerve leading to pain and loss of function. Current treatment approaches only temporarily alleviate pain and denervation which, however, does not prevent cyst recurrence. Hence, a mechanistic understanding of the pathogenesis of IGC can help clinicians understand them better and therefore device more effective treatment options. In this study, a preliminary analysis methodology is established to investigate the pathogenesis of IGC.

INTRODUCTION

The cross section of a nerve, as shown in Fig. 1 (Spinner et al., 2007), reveals tubes called fascicles which are bundles of nerve cells. The fascicles are embedded in a mass of connective tissue and fat called the inner epineurium which serves to cushion and insulate individual fascicles. These nerve contents are surrounded by a connective tissue sheath called the outer epineurium. Intraneural ganglion cyst (IGC) is a condition of fluid accumulation within the outer epineurium which causes the nerve to expand radially and nerve contents to be compressed.

One of the primary nerves innervating the lower leg is the common peroneal nerve (CPN) (Fig. 2) and it is frequently a site for Intraneural Ganglia. The CPN consists of three branches: the articular (AB), the deep (DPN) and the superficial (S). The superior tibio-fibular joint, which is a synovial joint, exists at the meeting point between the tibia and the fibula and is innervated by the articular branch. In a synovial joint the meeting point between two bones is encapsulated by a joint capsule containing fluid. Previous clinical research (Spinner et al., 2003) supports the theory that synovial fluid from the superior tibio-fibular joint enters the articular branch subsequent to joint capsule disruption through injury or degeneration. The increased pressure caused by continuous influx of fluid compresses the nerve contents, expands the nerve radially (Fig. 2 - stage 1) and causes further propagation proximally into the CPN (Fig. 2 - stages 2, 3 and 4). To effectively treat IGC and eliminate the common situation of postoperative recurrence, surgeons would benefit from an understanding of the underlying mechanics that influences cyst growth. It has been recently suggested (Elangovan et al., 2009) that Finite Element Analysis (FEA) be used to predict the growth behavior of cysts. The objective of this study is to use FEA to study proximal cyst propagation at the AB/DPN junction just before it reaches the CPN.

FINITE ELEMENT MODEL

A finite element model of the junction between the articular and the deep branches was created as shown in Fig. 3. The superficial branch was omitted since intra-operative images indicate a considerable distance between the location at which the articular branch meets the deep branch and the point where the superficial branch meets the deep branch. Dimensions for the model were averages of the dimensions measured from the left and right limb of a cadaveric specimen.

The current literature on nerves is lacking in the area of hyperelastic material properties. Hence appropriate assumptions were made judiciously by analyzing the load bearing components of a nerve (Sunderland, 1948; Topp and Boyd, 2006). It is known that the outer epineurium consists of elastin fibrils and collagen fibrils (type 1 & 2) aligned along the length of the nerve. Comparatively, the inner epineurium is similar but contains a significant amount of adipose tissue (fat) to cushion and insulate the fascicles. The main load-bearing portion of the nerve is the perineurium (Fig. 1), the outer sheath of the fascicles, which can be composed of up to fifteen layers of collagen fibrils (type 1 & 2) which are alternately aligned in the longitudinal, transverse, and oblique directions. The inner matrix of the fascicle, called the endoneurium (Fig. 1), consists of collagen fibrils (type 1 & 2) aligned longitudinally and endoneurial fluid.

The material discretization shown in Fig. 3 was performed for two reasons: 1) IGC usually occur in the outer epineurium and hence a separate material is necessary to specify the cyst in the model and 2)
the response of the contents inside the outer epineurium is dominated by fascicle response and hence they can be homogenized as one material for simplicity.

While the load-bearing components of a nerve are known, the relative proportion of each is not. However, one aspect is clear: the fascicles are much more collagenous than the outer epineurium. Also, since nerves undergo large deformations, only a hyperelastic material model will describe its behavior accurately. A simple and effective strain energy function for hyperelastic incompressible materials is the Mooney-Rivlin model given by

\[ \Psi = c_1^m (I_1 - 3) + c_2^m (I_2 - 3) \]  

where \( \Psi \) is the strain energy density, \( c_1^m \) and \( c_2^m \) are material property constants for a material ‘m’, and \( I_1 \) and \( I_2 \) are the first and second invariants of the right Cauchy-Green deformation tensor. Due to the collagenous nature of the fascicular region, it is considered as a ligament material with hyperelastic Mooney-Rivlin properties (\( c_1^E = 0.1653 \text{ N/mm}^2, c_2^E = 0.00588 \text{ N/mm}^2 \)) (Hirokawa and Tsuruno, 1997) and the outer epineurium is assigned properties that are an order of magnitude lesser i.e. \( c_1^E = c_1^m / 10 \) and \( c_2^E = c_2^m / 10 \).

The cyst is represented by the hollow crescent-like region shown in the outer epineurium in Fig. 4. The length of the cyst \( l \) is the length measured along the articular branch and the cyst arc angle \( \theta \) is the angle subtended by the arc of the cyst. The cyst thickness \( t \) represents the distance between the top and bottom surfaces of the cyst and decreases to 50% of its value at the rear end of the cyst to represent a crack-like scenario. The rear face of the cyst is called as the cyst face.

The cyst is pressurized by the continuous production of cyst fluid from the superior-tibio-fibular joint, which accumulates in the outer epineurium. In the finite element model, this cyst fluid pressure is represented by a pressure load applied on the inner surfaces of the cyst except at the cyst face. Since it is very difficult to measure the pressure of the cyst fluid, an arbitrary pressure of 0.0035 N/mm\(^2\) is chosen. To prevent rigid body motion errors, the ends A and B noted in Fig. 4 are fixed with respect to all displacements.

RESULTS

The failure of the cyst material is attributed to the principal stress 1 at the point which is the norm of the maximum normal stress theory. Fig. 5 shows that the maximum normal stress on the side face varies from 0.018 to 0.1 N/mm\(^2\) (eliminating stress concentrations). Also the cyst blow-out is consistent with intra-operative observations. The maximum stretch is given by 1.147, which occurs on the roof of the cyst. The principal stress 1 value on the left side face is the same as the right side face, which is expected since the structure is symmetric about the x-y plane. The stresses on the cyst face vary between -0.009 to 0.063 N/mm\(^2\). Comparing the stress values on the side against that on the cyst face, it is found that the magnitude at the sides is greater. This indicates that the cyst in this stage tends to engulf the sides before propagating down the branch. Apart from the principal stress magnitude, the direction in which the vectors are oriented is also examined to see in which direction these stresses will cause propagation.

CYST PROPAGATION

The next goal is to simulate the propagation of the cyst from one point to another. This is done by selecting a threshold value of principal stress 1 (0.02 N/mm\(^2\) in this case) above which failure occurs (maximum normal stress theory). Next, the portion of the cyst geometry corresponding to all elements that exceed the threshold stress value is removed and the cyst is remodeled as shown in Fig. 6. Hence the cyst dimensions increase by a \( d \) and \( \delta d \) and using many such small steps the cyst can be made to propagate from one point to another. After remodeling, a relaxed value of pressure (0.0025 N/mm\(^2\)) is reapplied and the cycle is repeated to simulate the continuous supply of cyst fluid pressure as the cyst grows.

SUMMARY

Intraneural ganglion cysts are introduced and the necessity for better understanding of their pathogenesis is noted. A preliminary finite element study is undertaken where different components of the nerve and the cyst are represented in the finite element model and simulated using pressure and displacement boundary conditions. Further, a method to simulate cyst propagation by remodeling geometry is devised. The results support clinical observation, assuring the reliability of the method.

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REFERENCES


FIGURE 1 – CROSS SECTION OF A NERVE (USED WITH PERMISSION OF MAYO FOUNDATION FOR MEDICAL EDUCATION AND RESEARCH)

FIGURE 2 – STAGES OF INTRANEURAL GANGLION CYST PROPAGATION (USED WITH PERMISSION OF MAYO FOUNDATION FOR MEDICAL EDUCATION AND RESEARCH)

FIGURE 3 – FINITE ELEMENT MODEL

FIGURE 4 – CYST GEOMETRY

FIGURE 5 – PRINCIPAL STRESS 1 ON THE CYST LEFT FACE

FIGURE 6 – INITIAL AND REMODELED CYST GEOMETRIES