Destructive Effect of Monopolar Electrocautery Knife Usage on Femoral Artery Endothelium During Spine Surgery: Experimental Study

Spinal Cerrahide Monopolar Elektrokoter Kullanımının Femoral Arter Endoteline Etkisi: Eksperimental Çalışma

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Abstract

Objective. Circulatory disorders are observed in the lower extremities of some patients after spinal surgery when a monopolar electrocautery knife (MEK) is used. Despite known the knowledge that electric currents can be hazardous to living tissues, MEK has been widely used in spine surgery. In this study, we investigated if MEK can cause endothelial injury in femoral arteries (FA).

Materials and Methods. Eighteen rabbits were included in this study, two of which were used as the reference group. The remaining animals were subjected to paravertebral soft tissue dissection along levels L1-L4. Half of the animals were dissected with MEK, and the other half were dissected with scissors and bipolar cautery. One month after the surgery, endothelial changes of the FAs were examined stereologically.

Results. Endothelial desquamation, cytoplasmic and nuclear condensations, cellular shrinkage and cellular loss were developed in the FAs of the MEK group. The endothelial cell density was 270 cells/mm2 in normal animals, 240 cells/mm2 in the non- MEK group and 190 cells/mm2 in the MEK group. The difference between the non-MEK and normal groups was not meaningful (P>0.05). The difference between the MEK and non-MEK groups was statistically significant (P<0.05); differences between the MEK and normal animals were even more significant (P<0.001).

Conclusion. Sharp dissection with MEK can have a detrimental effect on the endothelial cells of the FAs and cause postoperative circulatory disorders in the lower extremities. Therefore, long-term high-voltage electrocauterization should be avoided during spinal surgery unless absolutely necessary.

Keywords: Electrocauterization, Sharp dissection, Femoral artery, Endothelial cells

Özet

Amaç. *Bazı hastalarda spinal cerrahide monopolar elektrokoter (MEK) bıçak kullanımı sonrası alt ekstremitede sürkülasyon bozuklukları gözlenmektedir. Elektirik akımın yaşayan dokular için zararlı olduğu bilinmesine rağmen MEK spinal cerrahide kullanılır. Bu çalışmada MEK in femoral arter endotel hasarı yapıp yapmayacağını araştırmayı amaçladık.*

Gereç ve Yöntem. *Bu çalışmaya 18 tavşan alındı ve 2 si kontrol grup olarak kullanıldı. Geri kalan deneklerin paravertebral yumuşak dokuları L1-L4 mesafesi arasından diseke edildi. Deneklerin yarısın da MEK diğer yarısında bipolar koter kullanıldı. Cerrahiden 1 ay sonar femoral arter endoteli değişiklikleri sterolojikal olarak incelendi.*

Bulgular. *MEK grubunun femoral arter endotelinde endotelyal deskuamasyon, sitoplazmik ve nükleer kondensasyon, hücresel çekilme ve hücre kaybı gelişti. Endotel hücre dansitesi normal hayvanlarda 270 cells/mm2, non- MEK grubunda 240 cells/mm2, MEK grubunda 190 cells/mm2 idi. Non- MEK ve normal grup arasında anlamlı fark yoktu (P>0.05). Non- MEK ve MEK arasında önemli fark vardı (P<0.05). MEK ve normal grup arasında fark daha önemliydi (P<0.001).*

Sonuç. *MEK le yapılan keskin diseksiyon femoral arter endotelini etkilemektedir ve postoperative alt ekstremite sirkülasyon bozukluklarına neden olabilir. Bu nedenle uzun sureli ve yüksek voltajlı elektrokoterizasyon spinal cerrehide eğer kesinlikle gerekli değilse kullanılmasını önermiyoruz.*

Anahtar Kelimeler: *Elektrokoterizasyon, Keskin diseksiyon, Femoral arter, Endotel hücre.*

Introduction

Monopolar electrocautery knives (MEK) have largely been used for soft tissue dissections during spinal surgery, despite harmful effects. Living bones can transmit electric currents to neural and vascular structures, damaging them when the electric energy is converted to thermal energy [1]. The degree of electrical damage depends on the duration of the electric current; orientation and location of the cells in the current path; density, type and size of the contacted tissue; tissue susceptibility and metabolic rate [2-4]. MEK application can be hazardous to nerve roots, dorsal root ganglion (DRG) and radicular arteries [1,5,6]. Femoral arteries (FA) are innervated by the L1-5 segments of the spinal nerves [7]. Thus, application of MEK on these regions may result in FA injury via destruction of its neural control circuits, resulting in devastating effects. It is, however, also possible that postoperative circulatory disorders in the lower extremities may develop secondary to the application of MEK. In this study, we aimed to investigate the possible detrimental effects of MEK on the FAs. Stereologic and basic geometrical methods were used to estimate histopathological changes [8]. Our data suggest that there is a linear relationship between MEK use and FA endothelial injury.

Materials and Methods

Eighteen male hybrid rabbits were used in this study. The animals were approximately 1 year old and weighed 3.5±0.5 kg. Animal experimentation was carried out in an ethically proper way according to the guidelines set by the ethical committee of our university. Two of the rabbits (n=2) were used as a reference group for assessing femoral arteries in normal rabbits. The remainder were anaesthetized by subcutaneous injection of a mixture of ketamine hydrochloride (25 mg/kg), lidocaine hydrochloride (15 mg/kg) and acepromazine (1 mg/kg). After the surgical field was prepared, the skin between the L1-L4 levels was incised longitudinally. The paravertebral soft tissues were then dissected with MEK in half of the animals, and the other half was dissected with scissors and bipolar cautery. The MEK was created using an electrocoagulator set at 220 V/50 Hz, mimicking those used in human spinal surgery. After the operation, the rabbits were housed in individual cages and were given antibiotic-analgesic therapy for six days. One month after surgery, all animals were sacrificed. The femoral arteries, nerve roots and L1-5 dorsal root ganglions were removed bilaterally. These specimens were preserved in a 10% formalin solution embedded in paraffin blocks. After the sections were stained with hematoxylin and eosin, all specimens were observed histopathologically. Endothelial cell counting was made by three-dimensional constructions of four parallel sections in each FA. Statistical analysis was made using t-test.

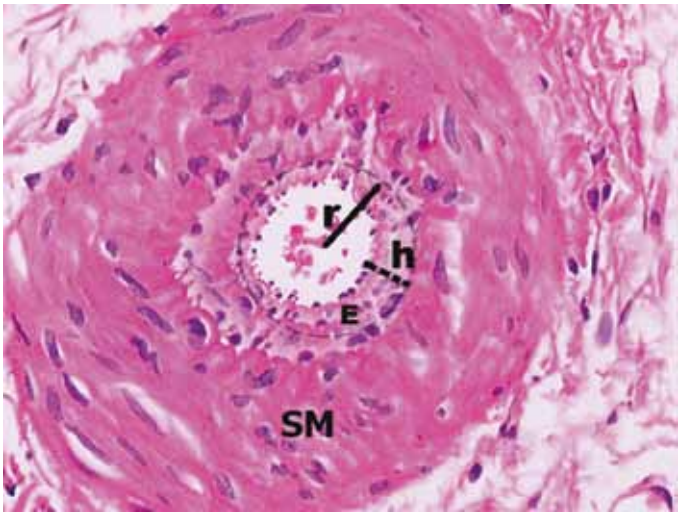

Fig. 1 — Volume estimation and cell counting methods are illustrated. Three-dimensional reconstructions were formed using four consecutive sections of femoral artery, vertically arranged. Four consecutive sections of the femoral artery are constructed three-dimensionally, and an imaginary cylinder was created. The height of the cylinder was represented as h, and the radius of cylinder was represented as r. h represents 5 µm sections of the femoral artery and is calculated as h=5x4=20 µm. The inner diameter of the femoral artery was 0.9 mm, r=0.45 mm=45 µm.

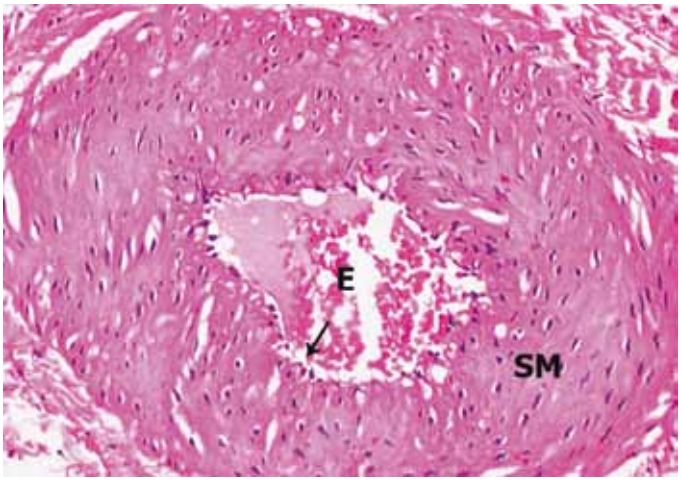

Fig. 2 — Histopathological appearance of the femoral artery of a healthy rabbit (SM, Smooth muscle; E, Endothelium) (LM, H&E, x40).

Luminal volumes of FAs and endothelial cell counts were done by using stereologic and basic cylindrical geometry methods. Four consecutive sections of femoral artery are constructed three-dimensionally, and an imaginary cylinder was created. These calculation methods are illustrated in Figure 1. The height of the cylinder is represented as h, and the radius is represented as r. h represents all 5 µm sections of the femoral artery, and is calculated as h=5x4=20 µm; the inner diameter of the FA was 0.9 mm, r=0.45 mm=45 µm. The volume of our imaginary vascular reference cylinder was calculated as follows: $V = \pi r^2 h$

Reference volumes were calculated for each FA and statistical analyses were calculated for these results.

Endothelial cell density was found by using cylindrical equations. Endothelial cells are originally arranged in a rectangular plane, formed by the inner surface of the cylindrical cavity of the FA. The borders of the reference cylinder is represented as $2\pi r$ and h. Thus, the surface of the reference counting plane is calculated by the following equation [9]: $S = 2\pi r \times h$

In the same way, the endothelial cell number was deter-

mined in each reference plane and used as the endothelial cell density. Statistical analyses were calculated for these results.

Results

No animals died during the procedures or postoperative period. However, paraparesis, hypoalgesia, hypotonia and pulse deficits of the femoral arteries were observed at the lower extremities of animals in the MEK group. Neuronal degeneration was more prominent in the MEK group compared to the non-MEK group.

The normal histological appearance of a coronary artery is shown in Figure 2, and a magnified histological appearance of a coronary artery in a normal rabbit is shown in Figure 3. Minimal endothelial degenerative changes were found in the coronary arteries of the non-MEK group (Figure 4). However, endothelial cell flattening, cellular shrinkage, condensations of the cytoplasm in the nucleus, endothelial cell necrosis and cellular loss were observed in the MEK group. Also, smooth muscle cell injury and atrophy, intimal edema and myocyte derangements were observed in the FAs (Figure 5).

Endothelial cell density was 270 cells/mm2 in normal animals. The FA cell density was lower in the MEK group (180 cells/mm2) than in the non-MEK group (240 cells/mm2) ($P<0.05$). The difference between the non-MEK and normal groups was not meaningful ($P>0.05$). The difference between the MEK and non-MEK groups was, however, statistically significant ($P<0.05$); and the differences between the MEK and normal groups were more significant ($P<0.001$).

Discussion

Vascular damage can be a dangerous complication of high voltage electric injuries. Electrical injuries can lead to vasospasm, thrombus development, tissue ischemia and necrosis [10,11]. Neurodegeneration of the nervi vasorum, thermal coagulation and spasm of the vasa nervorum were detected after electrical injury [12]. Also, a high-voltage alternating current (20.000 V) caused delayed motor neuron disease due to demyelination of the vasa nervorum [13]. Electric injury destroyed all endothelial and medial smooth muscle cells, and induced platelet-rich mural thrombosis in the arteries [14-16]. Kornowsky et al. [14] observed that electrical stimulation to the femoral artery of rabbits resulted in endothelial injury and arterial thrombosis. Electrical current injury can result in thrombus development via endothelial cell lesions and platelet aggregation [6].

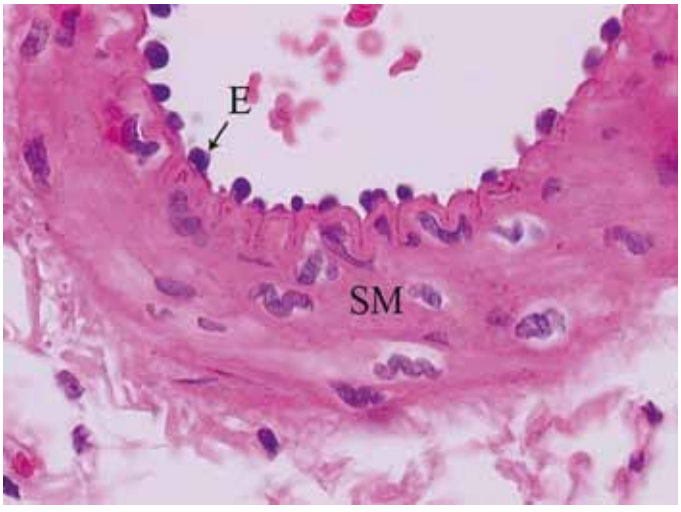

Fig. 3 — Magnified histopathological appearance of the femoral artery of a healthy rabbit. Internal elastic membrane, smooth muscles and endothelial cells are present with normal histological structures (SM, Smooth muscle; E, Endothelium) (LM, H&E, x100).

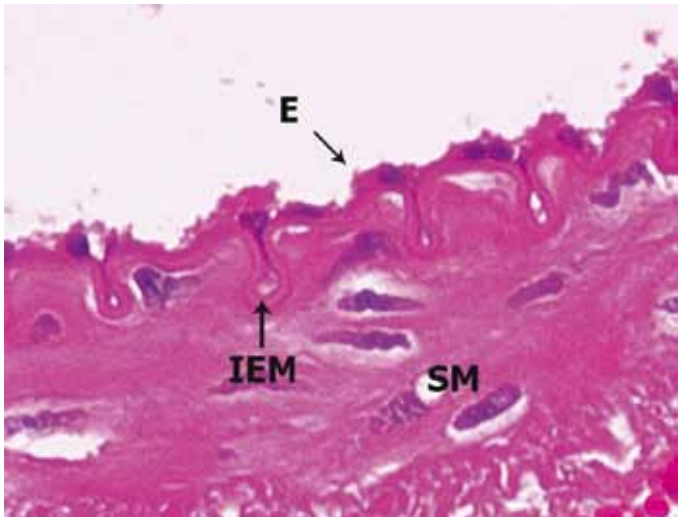

Fig. 4 — Magnified histopathological appearance of the femoral artery of a rabbit in the non-MEK group. Internal elastic membrane and smooth muscles are normal. Minimal endothelial cell shrinkage is present (SM, Smooth muscle; E, Endothelium, IEM, internal elastic membrane) (LM, H&E, x100).

Electrical shock may cause demyelination due to its detrimental vascular effects [17]. If the contact time is brief, non-thermal mechanisms of cell damage will dominate, and the damage is relatively restricted to the cell membrane. When contact time is much longer, the entire cell is directly affected, and the larger cells are more vulnerable to damage. In addition, vascular complications of lightning injury may result in acute renal failure, rhabdomyolysis, respiratory distress syndrome, autonomic dysfunction, paresis, plegia and even quadriplegia, muscle atrophy, sensory deficit, causalgia and reflex sympathetic dystrophy, which could result in a recovery time of several months [4,18]. Extracellular hypoxia is unlikely to be a significant factor in the neural injuries of peripheral nerves [14,19]. Spinal cord injury following an electrical accident can lead to motor and sensory dysfunctions. It was shown that facet denervation with MEK can lead to thrombus development in radicular arteries [20]. Aydin et al. [5] also showed that MEK usage in spinal surgery can result in nerve root and DRG degeneration. Motor and sensory innervations of the FAs are supplied by the L1-4 nerve roots [10].

In conclusion, direct effects of electric current, deteriorated neural control due to DRG degeneration may lead to functional, vasculo-mechanical and endothelial derangements in FAs. Endothelial injury was the most prominent finding in our study. Therefore, injured FAs may be responsible for circulation disor-

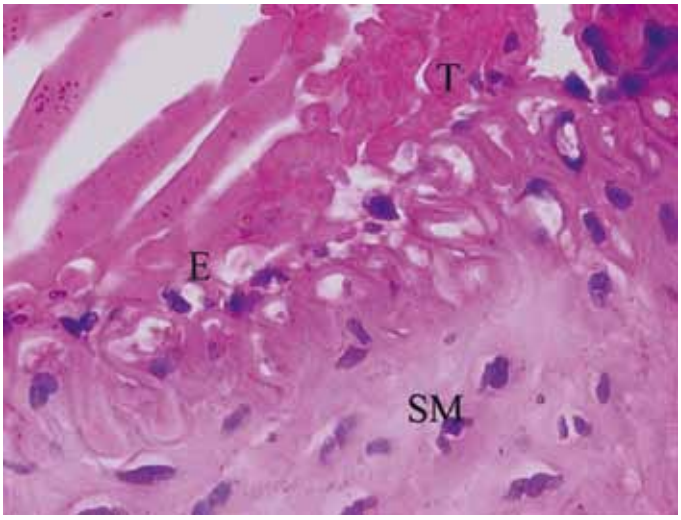

Fig. 5 — Magnified histopathological appearance of the femoral artery of a rabbit in the MEK group. Internal elastic membrane is more curled and locally ruptured. Smooth muscles are contracted and intimal edema is present. Endothelial cell shrinkage, nuclear and cytoplasmic condensations and cellular atrophy and loss are present. In the meantime, thrombus development is seen in the artery lumen (SM, Smooth muscle; E, Endothelium, T, Thrombus) (LM, H&E, x100).

ders in the lower extremities. We recommend that long-term high-voltage MEK should only be used in spinal surgery if absolutely necessary.

Conflict interest statement The authors declare that they have no conflict of interest to the publication of this article.

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