

olduğu sonucuna ulaşılmıştır.^[1] Bu sonuç, kollateral gelişiminde NO metabolizmasının önemini gösteren çalışmaların sonuçları ile uyumludur.

Geleneksel kardiyovasküler risk faktörleri ile koroner kollateral ilişkisini araştıran çalışmalarda çelişkili sonuçlar elde edilmiştir. Son yıllarda yapılan araştırma sonuçları diyabetin koroner kollateral gelişimini olumsuz yönde etkilediğini destekler yöndedir.^[4] Yazarların da belirttikleri gibi, diyabetik hastalarda çeşitli mekanizmalarla gelişen NO metabolizmasında bozulma sonucunda endotelial disfonksiyon gelişmektedir. Tip 1 ve tip 2 diyabetli hastalarda ADMA konsantrasyonunun artmış olduğu,^[5,6] hipergliseminin ADMA oranını artırdığı^[7] ve tip 2 diyabetiklerde sıkı glisemik kontrolle ADMA seviyesinin azaltılarak anti-aterojenik etki sağlanabileceği gösterilmiştir.^[8] Diyabet ve ADMA ilişkisini gösteren çalışmalar yanı sıra, ADMA'nın nefropati, retinopati gibi diyabetik komplikasyonlarla ilişkisini ortaya koyan klinik çalışmalar da bulunmaktadır.^[6,9,10] Bizim çalışmamızda, hipertansiyon, hiperlipidemi, diyabetes mellitus gibi geleneksel aterosklerotik risk faktörlerinin koroner kollateral gelişimi üzerine etkisi bulunamamıştır. Çalışmamızın kısıtlılıklar bölümünde belirttiğimiz gibi, hasta sayısının görece azlığı bu sonucu etkilemiş olabilir. Çalışmamızda atıfta bulunduğumuz yayınlardan biri olan, Güleç ve ark.nın^[11] endotelial NO sentaz genindeki Glu298Asp polimorfizmi ile kollateral gelişimi arasındaki ilişkiyi araştırdıkları çalışmada, kollateral gelişimi iyi olmayan grupta daha fazla diyabetik hasta olduğu, eNOS Glu298Asp polimorfizminin kötü kollateral gelişimi için tek öngördürücü faktör olduğu sonucuna varılmıştır. Literatürde, diyabetik hastalarda kollateral gelişimi ile ADMA ilişkisini araştıran çalışma bulunmamaktadır. Kardiyovasküler mortalite ve morbiditenin çok yüksek olduğu bir hasta grubu olan diyabetiklerde koroner kollateral arter gelişiminin bu hastaların prognozu üzerine olan etkisi oldukça açıktır. Bu nedenle, diyabetik hastalarda kollateral gelişimini araştıran ileri çalışmalara ihtiyaç vardır.

Yazarlar adına,

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KAYNAKLAR

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Treatment of iatrogenic pneumothorax on pacemaker implantation

2008;36:198

Dear Editor,

I have read the case image titled "A complication of pacemaker implantation: a large pneumothorax compressing the entire left lung" by Çay and colleagues,^[1] and I congratulate the authors. Yet, I want to remark on some aspects of their presentation.

Even though pneumothorax is a rather common complication of pacemaker implantation, surgical intervention is not required in the majority of the patients. Grier and colleagues^[2] reviewed the chest roentgenograms of 600 patients undergoing permanent cardiac pacemaker insertion and identified 15 cases of pneumothorax, one of which required intervention. In a prospective study, Aggarwal and colleagues^[3] reviewed 1,088 consecutive patients who had endocardial permanent pacemaker implantation. The authors reported eight patients (0.8%) who needed active medical treatment for pneumothorax, five of whom had tube thoracostomy and three had aspiration. On the other hand, 11 patients (1.0%) demonstrated insignificant pneumothorax involving less than 10% of the pulmonary field on the chest roentgenogram, which showed no symptoms or progression on subsequent chest roentgenograms. Işıtmangil and colleagues^[4] described their treatment protocol which included tube thoracostomy in iatrogenic pneumothorax cases when the pneumothorax size was more than 25%. If the pneumothorax size was between 15% and 25%, they monitored the patients closely and performed tube thoracostomy if the size of the pneumothorax increased on the chest roentgenogram at 6 hours, or they performed needle aspiration to remove the air if there was no increase in its size. Observation and 100% oxygen inhalation was preferred by the authors if the pneumothorax size was less than 15%, and tube thoracostomy was performed only if the pneumothorax size increased.

Furthermore, the site of tube thoracostomy should vary in iatrogenic pneumothorax due to pacemaker implantation via a subclavian vein puncture. Ventral tube thoracostomy, which is the preferred method in the classical treatment of pneumothorax and performed around the point where the midclavicular line crosses the second intercostal space, can harm the pacemaker or cause pacemaker pocket infection, necessitating generator and electrode removal. Therefore, in such cases, lateral tube thoracostomy should be performed where the midaxillary line intersects the sixth or seventh intercostal space.

Best regards,

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Author's reply

Dear Editor,

I would like to thank the author for his comments on our case report.

Pneumothorax following permanent pacemaker implantation is a rather common, but potentially life-threatening complication, as stated by the author. In addition, surgical intervention with a tube thoracostomy is rarely used for its treatment. However, in cases presenting with a large pneumothorax as in our case, intervention with a tube thoracostomy via the lateral approach has been generally the choice of therapy.

Best regards,

On behalf of the authors,

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