Anesthetic Management of a Hypothyroid Patient Undergone Deep Hypothermic Circulatory Arrest

DERİN HİPOTERMİK SİRKULATUVAR ARREST UYGULANAN HİPOTİROİDİ OLGUSUNDA ANESTEZİ YÖNETİMİ

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Abstract

It has been shown that deep hypothermic circulatory arrest during cardiac surgery influences the responses of the thyroid axis in euthyroid children. In contrast, the effect of deep hypothermic circulatory arrest (DHCA) during cardiopulmonary bypass (CPB) on thyroid hormone metabolism in an adult hypothyroid patient has not been reported. The anesthetic approach in a hypothyroid patient who underwent DHCA during urgent ascending aortic aneurysm repair presented here might suggest that the titration of anesthetic agents, directed by hemodynamic and EEG values and replacement of thyroid hormones immediately after surgery may contribute to the protective effects of DHCA on the neurohormonal mechanism.

Key Words: Hypothyroidism; circulatory arrest, deep hypothermia induced; cardiopulmonary bypass

Özet

Derin hipotermik sirkülatuvar arrestin (DHCA), kalp cerrahisi uygulanan ötiroid çocuklarda tiroid aksisini etkilediği bilinmektedir. Buna karşılık kalp cerrahisi süresince uygulanan DHCA'nın hipotiroid yetişkin bir hastada tiroid hormon metabolizması üzerine etkisi bildirilmemiştir. Çıkan aorta anevrizması nedeniyle acil olarak kardiyopulmoner bypass ile DHCA uygulanan hipotiroidi olgusunda anestetik ajanların hemodinamik ve EEG monitorizasyonu ile titre edilmesi ve cerrahiden hemen sonra tiroid hormonlarının verilmesi, DHCA'nın nörohomoral mekanizmalar üzerindeki koruyucu etkisine katkıda bulunmuş olabilir.

Anahtar Kelimeler: Hipotiroidi; derin hipotermik sirkulatuvar arrest; kardiyopulmoner bypass

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Thyroid hormones directly affect the heart and peripheral vascular system. These hormones can increase cardiac output by increasing myocardial inotropy, heart rate, and dilate peripheral arteries. A deficiency of thyroid hormones can cause cardiovascular disease and aggravate many preexisting conditions.[1]

Cardiopulmonary bypass (CPB) produces various alterations in endocrine homoeostasis which may exert important haemodynamic effects postoperatively. It is increasingly evident that cardiopulmonary bypass affects thyroid hormone metabolism, leading to a transient depression characterized by low levels of circulating TT_4 (total thyroxin), TT_3 (total triiodothyronine) and FT_3 (free triiodothyronine).[1-3]

Although limited data suggest a more frequent incidence of heart failure in hypothyroid patients undergoing cardiac surgery,[4] several reports suggest that patients with mild-to-severe hypothyroidism can undergo cardiac surgery without significant morbidity or mortality compared with euthyroid patients.[5,6]

Use of various methods of deep hypothermic circulatory arrest (DHCA) has long been the standard practice in treating pediatric patients with complex congenital heart disease and life threatening aneurismal lesions in adults.[2] DHCA during cardiac surgery also has an influence on the responses of the thyroid axis.[7] However, we have not found any reports about the effects of DHCA during CPB on thyroid hormone metabolism in a hy-

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pothyroid patient. The authors report the anesthetic approach and endocrine status in a hypothyroid patient who underwent DHCA for repairing the ascending and arch aorta aneurysm repair.

Case Report

The patient was a 74 years old woman (weight: 70 kg, height: 162 cm) with a two year history of the hypothyroidism, one year history of Dressler syndrome (acute pericarditis and pericardiac tamponate) which occurred one month after an inferior myocardial infarction, hyperlipidemia, and an aneurismal mass arising from the ascending aorta was scheduled for an urgent repair with cardiopulmonary bypass. Her preoperative medications included levatyroxine 0.05 mg/day, fluvastatin 40 mg/ day. Laboratory values were normal except for a thyroid function tests: thyroxine (T_4): 3.63 µg/dl (normal, 5.1-14.1), free thyroxine index (FT_4): 0.3 ng/dl (normal, 0.93-1.7), triiodothyronine (T_3): 0.3 µg/dl (normal, 0.3-2), free triiodothyronine index (FT_3): 0.42 ng/dl (normal, 1.45-3.48), thyroid stimulating hormone (TSH): 26 µIU/ml (normal, 0.27-4.2). Physical examination outside the operating room revealed a heart rate of 60 beats/minute and arterial blood pressure of 100/60 mmHg. Computerized tomography showed an aneurismal mass arising from the ascending aorta (6 cm) and extending to the beginning of the aortic arch.

Monitoring included invasive arterial pressure (by an arterial cannula in the radial artery), central venous pressure (by a central venous catheter in the internal jugular vein), end-tidal carbon dioxide pressure, oxygen saturation (SpO_2) and nasopharyngeal temperature. Brain function was monitored with the bispectral index (BIS) (Aspect A 2000 EEG monitor, Aspect Medical System Inc., Natrick, MA, USA).

General anesthesia was induced with midazolam 3mg, fentanyl 250 µg, and pancuronium 7 mg. The administration of anesthetic drugs (100 µ/ hour fentanyl infusion, midazolam 2 mg/ hour and, 0.5 MAC of isoflurane) was titrated to keep BIS 40-50 throughout the procedure except during DHCA.

The aneurysm was approached through median sternotomy. CPB was instituted via femoral arteriel and right atrial venous cannula. The patient was cooled down to 18°C. Replacement of ascending and hemiarcus aorta was then performed by using woven Dacron tubuler graft (Vascutek 734030) under DHCA. Although the arterial pressure and heart rate were slightly increased at the time of institution and termination of CPB (Figure 1), the opera-

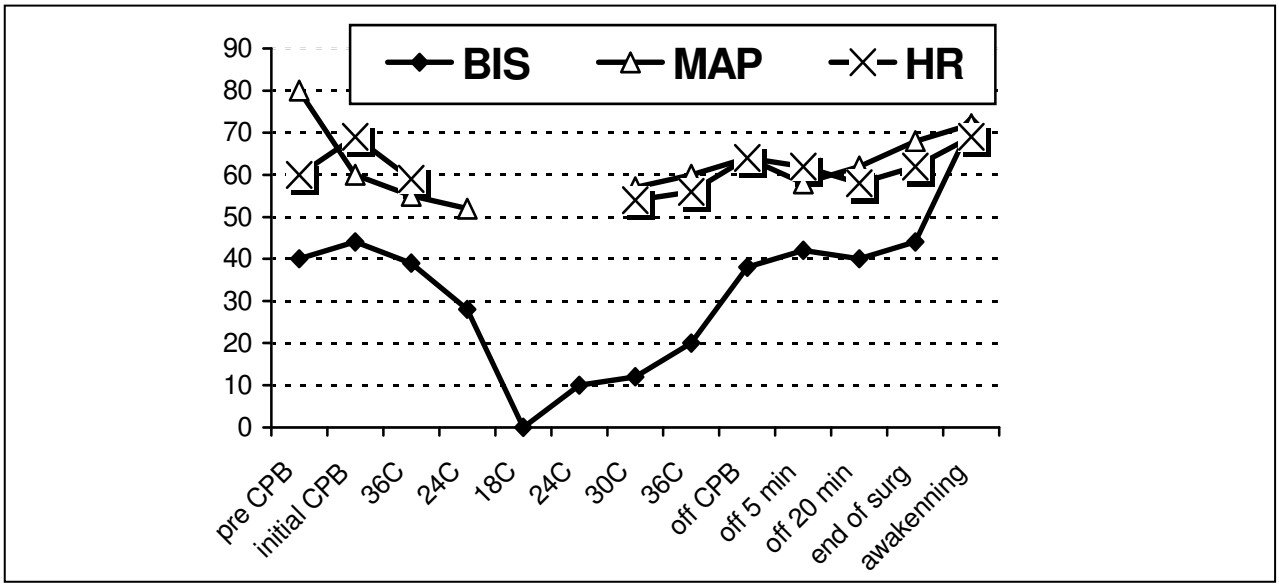

Figure 1. The changes in heart rate (HR, beat/min), mean arteriel pressure (MAP, mmHg), and bispectral index (BIS) before, during and following cardiopulmonary bypass (CPB).

tive hemodynamic course was uneventful before, during and post cardiopulmonary bypass period BIS values were recorded throughout the operative period (Figure 1). The BIS was 44 at the initiation of CPB. As cooling progressed, the BIS dropped preciously. The BIS reached 0 at 18 ^{o}C and remained at 0 throughout the DHCA period, which lasted 30 minutes. Additional drugs given on bypass period were thiopental 250 mg, methyl prednisolene 250 mg, pancuronium 10mg and 100 ml of 20% mannitol for cerebral protection. CPB and aortic cross clamp times were 155 and 47 minutes, respectively. The patient was rewarmed over 60 minutes to 36^{o}C. Bypass was successfully terminated with moderate doses of inotropic support (dopamine 5 µg/kg/min, dobutamine 5 µg/kg/min) and the BIS value was 38. Thereafter until the end of surgery, the BIS ranged from 38-44. BIS monitoring was continued for postoperative first day and BIS gradually rose to 72, at which time the patient began to awaken. The patient recovered full consciousness over the next eight hours and was extubated 18 hours postoperatively without any hemodynamic event.

When the patient was admitted to the ICU, levathroxine 0.05 mg was immediately given by nasogastric route and 0.1 mg was followed every day thereafter. Postoperative first day, her thyroid function tests were T_4: 3.93 µg/dl, FT_4: 0.4 ng/dl, T_3: 0.32 µg/dl, FT_3: 0.45 ng/dl, TSH: 12.2 µIU/ml. Postoperative third day, her thyroid function tests were T_4: 4.2 µg/dl, FT_4: 0.43 ng/dl, T_3: 0.34 µg/dl, FT_3: 0.6 ng/dl, TSH: 9.4 µIU/ml. She was also eventually discharged from the ICU without any complication on postoperative day three. After a year, her thyroid function tests were close to normal values (T_4: 4.93 µg/dl, FT_4: 0.8 ng/dl, T_3: 0.38 µg/dl, FT_3: 1.2ng/dl, TSH: 4.2 µIU/ml).

Discussion

We presented the anesthetic approach and endocrine status in a hypothyroid patient who underwent DHCA was uneventful discharged from hospital.

The management of hypothyroid patients before coronary artery bypass graft (CABG) surgery is unclear.[2] Hypothyroid patients tend to have a decreased cardiac output, resting heart rate, and serious pleural effusions, any of which may require preoperative therapy.[2,8] Patients with mild to moderate hypothyroidism tolerate cardiac surgery well without preoperative thyroid hormone replacement. If these individuals develop perioperative complication, (e.g., atelectasis, pneumonia, and prolonged ventilator dependency), a more in depth analysis of their thyroid condition and associated therapy is warranted.[2]

Our patient had moderate hypothyroidism, determined by her clinical condition and laboratory values. She has taken thyroxine preparations preoperatively (by oral route) and postoperatively (by nasogastric and oral route). Several studies have advocated thyroid hormone replacement during the early postoperative period in hypothyroid patients.[1,9-12] Thyroxine (T_4) is generally preferred as initial therapy over T_3 because of its long history of clinical efficacy, ability to prevent relapse, longer-half life, and the avoidance of supranormal levels of T_3 that have been associated with malignant arrhythmias and early mortality after treatment with intravenous T_3.[12]

There are no studies, which have analyzed the anesthetic requirements of hypothyroid patients although by clinical observation they appear to have increased sensitivity to anesthetic drugs[1]. Increased drug sensivity is probably secondary to reduced cardiac output, decreased blood volume, abnormal baroreceptor function, decreased hepatic metabolism and decreased renal excretion.[1,9] Hypothermia occurs quickly in these patients and is difficult to prevent and treat. Hypothermia also produces a linear decrease in anesthetic requirements.[1] Anesthetic agents enhance neuromuscular dysfunction particularly with the respiratory muscle. Pancuronium is a preferred muscle relaxant because of its vagolytic properties; however, reduced skeletal muscle activity in these patients coupled with a reduction in hepatic metabolism necessitates cautious dosing and careful monitoring of neuromuscular function. Hypothyroid patients can be extremely sensitive to narcotics and sedatives.[1] Unresponsive hypotension may require

supplemental steroid administration. During this procedure, anesthesia induction was accomplished with low doses of the induction agents and titration of the fentanyl and midazolam infusions and isoflurane concentrations were kept low because of our patient's hypothyroidism.

Cardiopulmonary bypass has been associated with alterations in thyroid hormone levels that resemble the 'euthyroid sick syndrome'. This syndrome is characterized by depression of TT3 and FT3 concentrations, a concomitant increase in reverse T3 (rT3) levels, and normal concentrations of TSH, TT4, and FT4.[1] The mechanism by which CPB affects thyroid hormone level remains unclear. Hemodilution, altered concentration of thyroid binding globuline, displacement of the hormones by drugs, hypotalomo-pituitary-thyroid axis dysfunction, changes in metabolism, and hypothermia have been speculated as the cause of post-CPB thyroid dysfunction.[1,7]

Ririe et al.[8] examined thyroid hormone levels in children who underwent DHCA or CPB alone and suggested that DHCA could promote release of thyroid-releasing hormone (TRH) from the hypothalamus, directly stimulating TSH release from the pituitary gland or maintaining adenohypophyseal responsiveness to TRH. They also determined that CPB alone decreases TSH levels and to blunt the response of TSH to TRH. These authors suggested that CPB with deep hypothermia might also inhibit the release of a unknown mediator that suppresses the hypothalamic-pituitary axis.[8]

Depth of anesthesia was controlled with monitoring of hemodynamic and neurological functions during our patient's operative procedure. The initiation and termination periods of CPB are critical times in regards to the patient's hemodynamic and neurologic status. Such periods of gross hemodynamic change are associated with alterations in drug concentration, a change in body temperature, vasoactive drug administration and direct cardiac manipulation, which may be primarily responsible and likely unrelated to anesthetic depth.[2,13] The arterial pressure and heart rate at the time of institution and termination of CPB was slightly increased in our patient.

Electroencephalography (EEG) is used to monitor the function of the central nervous system in a variety of surgical procedures.[14] The bispectral index (BIS) is a new technique of computer-processed EEG that enables the assessment of anesthesia as well as the detection of awareness. The scale used to record the BIS is set arbitrarily from 0 (complete electrical silence) to 100 (fully awake). Reported benefits of BIS monitoring during anesthesia include decreased drug use, faster wake-up and extubation, and better operating room and postanesthesia care unit utilization.[13,14] Since, there is a greater potential for awareness during cardiac anesthesia, BIS monitoring may be of greater value[15]. The administration of anesthetic drugs was titrated to keep BIS 40-50 through out the procedure except for DHCA in our patient, since the BIS was zero secondary to the lack of cerebral perfusion.

The most important cerebral protective measure used during procedures requiring circulatory arrest is deep hypothermia [13]. In circulatory arrest, BIS monitoring offers several potential advantages over the use of a conventional EEG monitor (i.e. BIS of 0 and suppression ratio of 100%).[15] Burst suppression also has been used as an indicator of adequate brain protection during DHCA.[16] The BIS reached 0 (burst suppression ratio 100%) at 18°C in our patient.

In conclusion, the anesthetic approach in a hypothyroid patient who underwent DHCA during aortic aneurysm repair presented here might suggest that the titration of anesthetic agent with monitoring of hemodynamic and EEG values as well as replacement of thyroid hormones immediately after surgery might improve the preventive effects of DHCA on the neurohormonal mechanism.

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