Guillain-Barre Syndrome Following Cardiopulmonary Bypass

KARDIYOPULMONER BYPASS SONRASI GUI E LA IN-BA RR E SENDROMU

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SUMMARY

Acute inflammatory demyelinating polyradiculoneuropathy of the Gttillain-Bare syndorome is known to occur after infection (60%), immunization and surgery (10%). We describe one case in which it occurred after cardiopulmonary bypass done during mitral and aortic valves replacement. Tltough it may be no more than a chance association, we should be alert to unexplained weakness occurring after cardiopulmonary byypass.

The Guillain-Barre syndrome (GBS) is an uncommon disorder of obscure etiology: Guillain, Barre and Strohl, in 1916, gave the syndrome its cponym (GBS) (Macleod 1987). Acute inflammatory demyelinating polyradiadoneuroputy is characterized by an evolving symmetrical lower motor neuron paralysis. It occurs worlwide and in all age groups, with a reported incidence of 0.75 to 1.911000 000personslyear (Renlund el al 1987). GBS is known to occur after infection (60%), immunization and surgery (10%) (McDonagh and Dawson 1987, Kaslow et al 1987). It has been estimated that 5% to 10% of cases follow surgery by an interval of 1 to 4 weeks. Tlie occurence has not been dependent on the type of surgery, the presence of infectious complications, or the mode of anesthesia. There is scant literature on the association between

ÖZET

Guillain-Barre Sendromu (GBS) olarak bilinen Akut inflamalıtar demieliıüzan poliradikülonöropati immuniza syondan sonra %60 infeksiyonlardan sonra ve % 10 major cerrahilerden sonra görülebilir. Burada cardiopıdmoner bypass ve iki kapak (aort ve mitral) rcplasmanı sonrası gelişen GBS anlatılacaktır. Bu vak'a münasebeti ile cardiopıdmoner by-pass sonrası hastada gelişebilecek nedensiz bir kas güçsüzlüğünü nörolojik yönden dikkatlice incelenmesi gerektiği vurgulanmıştır.

GBS nadir görülen bir hastalıktır. Ilt olarak Gullain, Barre ve Strohl tarafından I916'da tariflenmiş (Macleod 1987) ve nöropatolojisi açıklanmıştır. Olay, simetrik aşağı motor nöron paralizisine neden olan akut inflamalıtar demieliıüzan, poliradikülonöropatVdir. Her yaş grubunda ve dünyanın her yerinde görülebilir ve %000 0,75-1,9 sıklığındadır. İmmunizasyon sonrası, infeksiyon sonrası %60 ve cerrahi sonrası % 10 sıklığında rastlanır, (McDonagh and Dawson 1987, Kaslow et al (1987). Genellikle operasyondan sonra 1 ile 4 hafta sonra başlamaktadır. Cerrahinin tipiyle, .anestezinin tipiyle ve infeksiyon vb. postoperatuar komplikasyonlarla ilişkili değildir.

Literatürde GBS ile cardiopıdmoner by-pass arası ilişki ile ilgili sınırlı yayın bulunmaktadır.

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REPORT OF A CASE

A 51-year-old woman was operated with cardiopulmonary bypass for mitral and aortic valves re-

Cardiopulmonary bypass and CBS and WC herein report such a case.

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placement. Aortic cross-damp time was 77 minutes. Her postoperative course was complicated in by low caridac output that was treated with intraaortic balloon pumping which was continued for 24 hours. The patient made rapid improvement and was going to be discharged on the eighth postoperative day, taking aspirin, dipyridamol, digoxin and sodium warfarin when she noticed weakness and paresthesia of her legs, which progressed rapidly until he was unable to stand in the ninth postopcraitve day. There was no history of any neurologic disease beforehand. Examination revealed slight hypophonia, hypotonia, areflcxia, only 2-2.5 (+) strenght in all muscle groups and a prominent subjective distal gradient to sensory modalities. Blood chemistry, CBC urinalysis were normal. Lumbar puncture could not be done because of the risk of bleeding secondary to anticoagulant therapy.

Compound muscle action potentials of the right upper extremity revealed dispersion and prolonged duration. No action potentials were obtained with stimulation of the right lower extremity nerves. Needle electromyography of the left abductor pollicis brcvis showed denervation potentials and low amplitude muscle action potentials with reducde recruitment. All of these features support the clinical diagnosis of GBS. Within four days, the patients developed further ascending motor weakness leading to nearly complete paralysis and she required mechanical ventilation in the eleventh postoperative day. She was treated supportively. She did not improve and the mechanical ventilation could not be stopped. Tracheostomy was done and the mechanical ventilation was continued through it. Her course was complicated by pneumonia which was followed by sepsis and she died 95 days after the operation.

COMMENT

The pathogenesis of Guillain-Barre syndrome is poorly understood, but the disease seems to represent the unmasking of a latent neuropathy following-immunologic stress (Renlund 1987). In the majority of patients, this stress is provided by a viral infection, but in approximately 10% of cases, as in th esubject of thes report, the possible precipitant is major surgery. Although the GBS is often thought to have a benign prognosis, 7% of patients die and a further 16% suffer residual diabilily (Winer et al 1985). Recovery may not begin for up to six weeks and full clinical recovery may take up to seven-months after the maximum deficit has occurred (Briscoe etal 1987).

The incidence of GBS after cardiopulmonary bypass is low and it is unlikely that cardiopulmonary bypass represent a special stress or trigger to this disease. Though we might have observed no more than a chance association, wc suggest that patients who complain of weakness or lethargy after cardiopulmonary bypass should undergo careful neurological examination.

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