

BÖLÜM 13

DİYABETİK SANTRAL SINİR SİSTEMİ KOMPLİKASYONLARI



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GİRİŞ

Diyabetes Mellitus (DM), epidemî haline gelmiş ve çeşitli komplikasyonları nedeniyle yükü hızla artan bir hastalıktır (1). Otoimmünite, çevresel faktörler ve pankreas β hücrelerinin işlevsel yetersizliğine bağlı olarak insülin sekresyonunda azalmaya yol açan, insülin direnci ile ilişkili olan, hiperglisemi durumu ve hipoglisemi atakları ile karakterize bir metabolik hastalıktır (2, 3). Beyin, fonksiyonları için gerekli enerji üretimi ve nörotransmitter sentezi için glikoza bağımlı olduğundan glikoz regülasyonu kritik öneme sahiptir. Glikoz nörolojik hastalıkların patofizyolojisinde etkili olan oksidatif stres, programlanmış hücre ölümü, plastisite ve hipotalamik devrelerin düzenlenmesinde rol oynamaktadır (4, 5).

DM hem merkezi hem de periferik sinir sistemi üzerinde etkilere neden olur ve başlıca komplikasyonları aksonopatiler, nörodegeneratif hastalıklar, nörovasküler hastalıklar ve bilişsel bozulmadır (6). DM'nin inme, encefalopati ve demans gibi kronik dönem komplikasyonları olduğu gibi koma, fokal nörolojik kayıp ve nöbet gibi akut dönem komplikasyonları da bulunmaktadır.

İNME

DM'nin mikrovasküler ve makrovasküler etkileri sonucu gelişen en önemli santral sinir sistemi komplikasyonlarından biri inmedir. Yapılan çok merkezli

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nundan 20-120 dakika sonra ve sıkılıkla plazma glikoz konsantrasyonu normale dönmeden önce EEG'nin normale döndüğü bildirilmiştir (93).

Hipoglisemide DWI ağırlıklı kranial MRI'da sitotoksik ödeme bağlı gelişen geri dönüşü olan lezyonlar ile karşılaşılabilir (89, 94). Bu lezyonlarının gelişmesi üzerinde hipogliseminin süresi şiddetinden daha önemli bir faktördür (95). Bu nedenle hipogliseminin hızla tanınması ve tedavi edilmesi beyin hasarını önleyebilir. Tutulum olan bölgeler korpus kallozum arka bacağı, korona radyata, sentrum semiovale, korteks, bazal gangliya ve hipokampus gibi oldukça değişkenlik gösterebilir ve lezyon yaygınlığı arttıkça прогноз kötü seyretmektedir (94).

Hipoglisemi oral yada intravenöz tedavi gerektirir, iyileşme sıkılıkla hızlıdır ancak hipoglisemi süresine bağlı olarak daha uzun sürebilir. Komada olan hastalarda Wernicke-Korsakoff sendromu için önce empirik intravenöz tiamin ardından rebound hiperglisemiyi önlemek için yakın takip ile intravenöz deks-troz içeren solusyonlar verilmelidir (86). Hipoglisemik semptomların tanınması, egzersiz periyotlarının uygun şekilde düzenlenmesi, antidiyabetik tedavi ve kan şekeri takipleri konusunda hasta eğitimi verilmesi önerilir.

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