

LUARAN HIPOKSIA ISKEMIK ENSEFALOPATI DERAJAT BERAT PASCA TERAPI HIPOTERMIA

INTISARI

Latar Belakang: Hipoksia iskemik ensefalopati (HIE) merupakan salah satu penyebab kejang neonatal. Meskipun terapi hipotermia terbukti menurunkan mortalitas, sebagian pasien berisiko mengalami sekuela neurologis berupa epilepsi, gangguan perkembangan, *cerebral palsy*, serta gangguan kognitif. Pemantauan perkembangan dan tata laksana multidisiplin diperlukan untuk mengoptimalkan luaran pasien.

Kasus: Bayi laki-laki lahir dari ibu 32 tahun, G5P3A1, usia kehamilan 38 minggu dengan bantuan induksi. Bayi lahir tidak menangis dengan tonus otot yang lemah. Resusitasi tahap awal dilanjutkan sampai ventilasi tekanan positif. Pasien mengalami kejang pada usia satu jam lalu dirujuk ke RSUP Dr. Sardjito untuk menjalani terapi hipotermia selama 72 jam. Selama perawatan, pasien mendapatkan obat anti-epilepsi serta pemantauan menggunakan *amplitude-integrated electroencephalography* (aEEG). Skor Thompson membaik dari 16 menjadi 6 setelah terapi hipotermia. Kejang klinis menghilang pada hari ketiga, sedangkan aktivitas kejang subklinis masih terdeteksi hingga hari kesepuluh. Pasien diizinkan untuk rawat jalan pada usia 14 hari. Pada evaluasi lanjutan, pertumbuhan pasien ditemukan baik, namun ditemukan peningkatan tonus pada keempat ekstremitas. Pemeriksaan EEG menunjukkan perlambatan aktivitas dasar difus yang mendukung diagnosis HIE, sedangkan MRI otak memperlihatkan lesi hiperintens pada talamus, mesensefalon, dan serebelum yang mengindikasikan cedera otak akibat hipoksia-iskemia berat. Selama enam bulan masa tindak lanjut, pasien tidak mengalami kekambuhan kejang dan terapi antiepilepsi berhasil dihentikan. Pada usia empat bulan, anak mengalami keterlambatan perkembangan motorik kasar yang menunjukkan perbaikan setelah menjalani fisioterapi dan stimulasi perkembangan.

Kesimpulan: Kasus ini menggambarkan perjalanan klinis seorang bayi dengan HIE akibat asfiksia perinatal yang mendapatkan terapi hipotermia sesuai indikasi. Walaupun kontrol kejang baik, pasien tetap mengalami keterlambatan perkembangan motorik kasar yang memerlukan rehabilitasi dini. Tata laksana komprehensif yang meliputi pengendalian kejang, rehabilitasi, serta evaluasi komplikasi HIE merupakan kunci untuk mengoptimalkan luaran jangka panjang.

Kata kunci: hipoksia iskemik ensefalopati; hipotermia; neonatal; kejang; prognosis

OUTCOME OF SEVERE HYPOXIC-ISCHEMIC ENCEPHALOPATHY FOLLOWING THERAPEUTIC HYPOTHERMIA

ABSTRACT

Background: Hypoxic-ischemic encephalopathy (HIE) is one of the leading causes of neonatal seizures. Although therapeutic hypothermia has been shown to reduce mortality, a considerable proportion of affected infants remain at risk of long-term neurological sequelae, including epilepsy, developmental delay, cerebral palsy, and cognitive impairment. Therefore, longitudinal developmental surveillance and multidisciplinary management are essential to optimize patient outcomes.

Case Presentation: A male neonate was born at 38 weeks of gestation to a 32-year-old gravida 5 para 3 abortus 1 mother following labor induction. At birth, the infant did not cry and exhibited poor muscle tone. Initial resuscitation was performed and escalated to positive pressure ventilation. A seizure occurred at one hour of age, prompting referral to RSUP Dr. Sardjito for therapeutic hypothermia, which was administered for 72 hours. During hospitalization, the patient received antiseizure medications and continuous monitoring with amplitude-integrated electroencephalography (aEEG). The Thompson score improved from 16 to 6 following hypothermia therapy. Clinical seizures resolved by the third day, whereas subclinical seizure activity persisted until the tenth day. The patient was discharged at 14 days of age. During follow-up, growth was appropriate; however, increased muscle tone was noted in all four extremities. Electroencephalography demonstrated diffuse background slowing consistent with HIE, while brain magnetic resonance imaging revealed hyperintense lesions involving the thalamus, mesencephalon, and cerebellum, indicative of severe hypoxic-ischemic brain injury. Throughout six months of follow-up, no seizure recurrence was observed, and antiseizure medications were successfully discontinued. At four months of age, the patient developed gross motor delay, which showed improvement following physiotherapy and developmental stimulation.

Conclusion: This case illustrates the clinical course of an infant with HIE secondary to perinatal asphyxia who received timely therapeutic hypothermia according to current recommendations. Despite successful seizure control, the patient developed gross motor delay requiring early rehabilitation. Comprehensive management, including seizure control, rehabilitation, and surveillance for HIE-related complications, is crucial for optimizing long-term outcomes.

Keywords: hypoxic-ischemic encephalopathy; therapeutic hypothermia; neonate; seizures; prognosis