The Egyptian Journal of Radiology and Nuclear Medicine (2016) 47, 297–304

Egyptian Society of Radiology and Nuclear Medicine

The Egyptian Journal of Radiology and Nuclear Medicine

www.elsevier.com/locate/ejrnm
www.sciencedirect.com

ORIGINAL ARTICLE

Magnetic resonance imaging versus transcranial ultrasound in early identification of cerebral injuries in neonatal encephalopathy

Eman A.Sh. Genedi [a], **Noha Mohamed Osman** [a,*], **Marwa Talaat El-deeb** [b]

[a] *Department of Radio-Diagnosis, Faculty of Medicine, Ain Shams University, Cairo, Egypt*
[b] *Department of Pediatrics, Faculty of Medicine, Ain Shams University, Cairo, Egypt*

Received 12 September 2015; accepted 3 January 2016
Available online 14 January 2016

KEYWORDS

Neonatal encephalopathy;
Hypoxic–ischemic encephalopathy;
HIE;
MRI;
Transcranial ultrasound

Abstract *Objective:* Neonatal encephalopathy (NE) is a condition that causes significant morbidity and mortality to the infant. The diagnosis and severity of NE rely heavily on clinical presentation and imaging findings.
The present study was planned to assess the role of MRI and Transcranial ultrasound (TCUS) in the early identification of cerebral injuries in NE.
Patients and methods: Our study enrolled 38 newborns presented with NE. Brain MRI and TCUS were carried out for each case and their results were compared.
Results: MRI was positive in 33 cases. Findings at MRI supported hypoxic-ischemic encephalopathy as an etiology in 25 neonates, and other etiologies included metabolic disorders in 2, congenital neonatal infection in 1, 2 cases of neonatal stroke, congenital brain anomalies in 2 neonates and cerebral venous sinus thrombosis in 1. The overall diagnostic accuracy of TCUS compared to MRI was 78.9%, while the overall sensitivity and specificity were 81.8% and 60% respectively.
Conclusion: TCUS is an effective screening tool in detecting the etiology of NE in suspected cases; it is sometimes crucial in critically sick neonates; however, early MRI is mandatory as it can detect precisely the extent of brain injury compared with TCUS alone.

1. Introduction

Neonatal encephalopathy (NE) is a heterogeneous, clinically defined syndrome characterized by disturbed neurological function in the earliest days of life, manifested by feeding difficulties, irritability, abnormality of tone, seizures, and reduced level of consciousness, and often accompanied by difficulty with initiating and maintaining respiration (1). The terminology NE is preferred to Hypoxic Ischemic Encephalopathy

* Corresponding author at: 81 Mohy Eldeen Abdel Hameed, 8th District, Nasr City, Cairo, Egypt. Mobile: +20 1001798342.
E-mail addresses: emangeneidi@hotmail.com (E.A.Sh. Genedi), drnohaosman@yahoo.com (N.M. Osman), drmarwa_eldeeb@yahoo.com (M.T. El-deeb).
Peer review under responsibility of Egyptian Society of Radiology and Nuclear Medicine.

http://dx.doi.org/10.1016/j.ejrnm.2016.01.001

Table 1 TCUS findings correlated with Brain MRI findings in our 38 patients.

	Positive MRI	Negative MRI	Total
Positive TCUS	3	6	9
Negative TCUS	2	27	29
Total	5	33	38

(HIE) because it does not imply a specific underlying etiology or pathophysiology. Neonatal encephalopathy can result from a wide variety of conditions and often remains unexplained. Perinatal HIE (by far the most common cause) is one subset of neonatal encephalopathy; other subsets include those resulting from metabolic disorder, congenital infection, drug exposure, nervous system malformation, birth trauma and neonatal stroke (2,3).

A clinical history that includes a perinatal insult, low Apgar score, need for resuscitation, decreased cord arterial pH level, other organ failure, respiratory failure, or some combination of these factors increases the level of confidence in a diagnosis of HIE (4).

Neonatal encephalopathy incidence is estimated as 3.0 per 1000 live births (5). Estimates in developing countries range from 2.3 to 26.5 per 1000 live births (6,7). The risk factors for NE vary between developed and developing countries with growth restriction the strongest in the former and twin pregnancy in the latter. It is estimated that 30% of cases of NE in developed populations and 60% in developing populations have some evidence of intrapartum hypoxic-ischemia (5).

Although term infants with mild encephalopathy generally make a full recovery, 20% of affected infants die in the neonatal period and another 25% develop significant neurological sequelae. For preterm infants, compared with term infants, the overall prognosis is worse (8).

Neonatal encephalopathy or HIE has been graded by Sarnat and Sarnat (9) into mild (stage 1), moderate (stage 2) and severe (stage 3). Moderate and severe encephalopathy is attributable to asphyxia in 60% of cases, most of which evolve during labor (10). Early identification of infants at

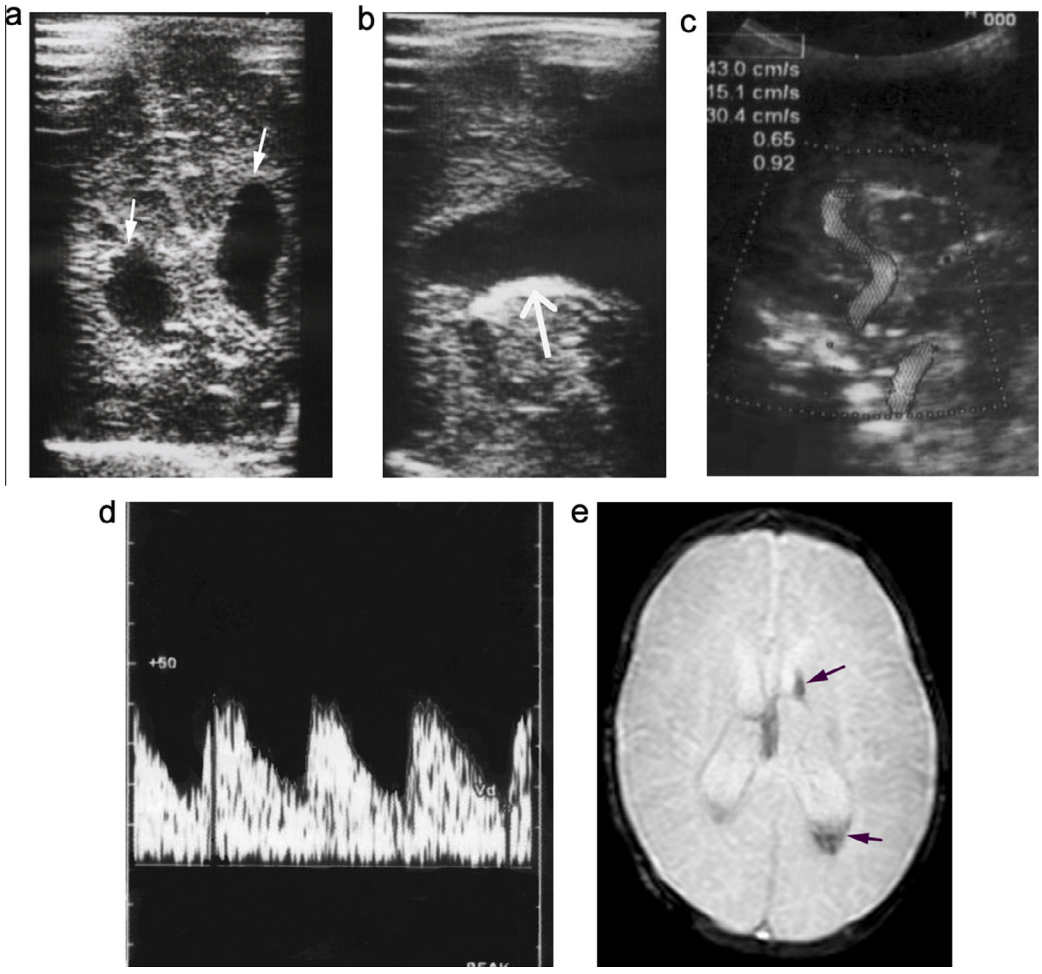

Fig. 1 Preterm neonate delivered at 32 weeks with uncomplicated delivery. Presented by CL, seizures and low Apgar score at day 1 after delivery. TCUS revealed bilateral ventricular dilatation (arrows) (a) and caudothalamic groove echogenicity representing germinal matrix hematoma (arrow). (b) Duplex interrogation revealed reduced RI in the ACA, indicating hyperemia (c & d). A diagnosis of Grade II–III intraventricular hemorrhage was suggested. MRI confirmed the above mentioned findings and hemorrhage was evident in gradient weighted image (arrows) (e).