Pictorial essay

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Hypoxic ischemic cerebral lesions of the newborn – ultrasound diagnosis. Pictorial essay.

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Abstract

Transcranial ultrasonography is the most widely used neuroimaging technique in both premature and full term infants. The high susceptibility to hypoxia of the preterm brain explains the raised prevalence of intracranial haemorrhages at this group of patients. Ultrasound examination contributes to assessment of the neurologic status in children by diagnosing and staging of the intracranial bleeding, and brings informations about immediate and long term prognosis. The two major pictures of cerebral damage secondary to perinatal hypoxia are: peri and intraventricular haemorrhages and periventricular leucomalacia respectively. This paper present the major features for ultrasound diagnosis in both pathological situations.

Keywords: intracranial hemorrhage, preterm, full term, ultrasound

Introduction

The newborn brain, especially in preterm, is highly susceptible to hypoxia. Two major pictures of cerebral damage secondary to perinatal hypoxia are described: peri and intraventricular haemorrhages (PVH-IVH) and periventricular leucomalacia respectively (PVL). It is demonstrated that transfontanelar ultrasound sensibility and accuracy in PVH-IVH diagnosis is similar to CT [1-3].

I. Peri and intraventricular hemorrhage

PVH-IVH classification is detaliated in table I.

Table I. Peri and intraventricular hemorrhage classification (adapted from Papile, et al) [4]

Grade I	Subependimal (germinal matrix) hemorrhage
Grade II	Hemorrhage extension to the ventricular system, occupying less than 50 % of one of the lateral ventricles; no acute ventriculomegaly
Grade III	Hemorrhage extension to the ventricular system occupying more than 50 % of one or both lateral ventricles; acute uni or bilateral ventriculomegaly
Grade IV	Hemorrhage grade I, II or III with extension to the cerebral tissue

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Grade I PVH-IVH (fig 1-3) is produced at the germinal matrix level and is specific to preterm infants. The germinal matrix is a stress sensitive structure, composed by thin-walled vessels, migrating neuronal components, and vessel precursors. It is located in the thalamic - caudate groove on the subependymal region, between the caudate nucleus and thalamus (fig 1). This transient, highly vascular structure maturates by the 34th gestational week, explaining why haemorrhages at this level become very unlikely after this age. Around 30-55 % germinal matrix haemorrhages occur in preterm less than 32 weeks gestational age and less than 1500 grams [5]. In most cases, the ultrasound abnormalities are observed during the first week of life (days 4-7). This haemorrhagic process may remain isolated or, less common, may extend from the thalamic - caudate groove into the lateral ventricle and/or to the periventricular cerebral parenchyma. Newborns with isolated grade I haemorrhages are generally asymptomatic or manifest discreet clinical signs; their long time follow-up demonstrate no major neurological sequels.

Grade II intracranial haemorrhage (fig 4) might be isolated or secondary to a grade I hemorrhage extending to the ventricular system. The haemorrhagic process occupying less than 50 % of one lateral ventricle is not correlated with acute ventriculomegaly. The outcome is generally favourable with complete resolution in about 3 - 5 weeks. In few cases a slight unilateral, nonevolutive ventriculomegaly is noticed. Neurological outcome is generally good [6,7].

Grade III intracranial haemorrhage. The abundant bleeding inside the ventricular system leads to ventricular distension, commonly asymmetrical, affecting the body, the posterior horns, or the whole lateral ventricle, sometimes associating IIIrd and IVth ventricle dilation (fig 5, fig 6). Resolution of the hemorrhagic process usually needs 5 to 6 weeks, w various degrees of ventricular distension. Grade III haemorrhages outcome is generally severe, with immediate complications, along with an

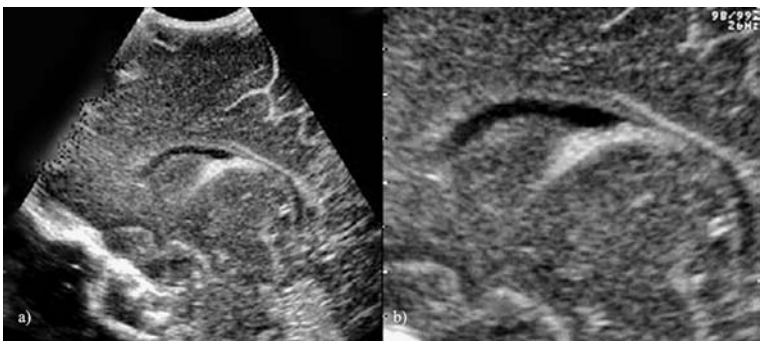

Fig 1. a) Sagital view at lateral ventricle level: echogenic material in the thalamo-caudate groove; b) Detail: the irregular echogenic deposit is located between the caudate nucleus (anterior) and thalamus (posterior). ***Diagnostic: small subependimal grade I haemorrhage.***

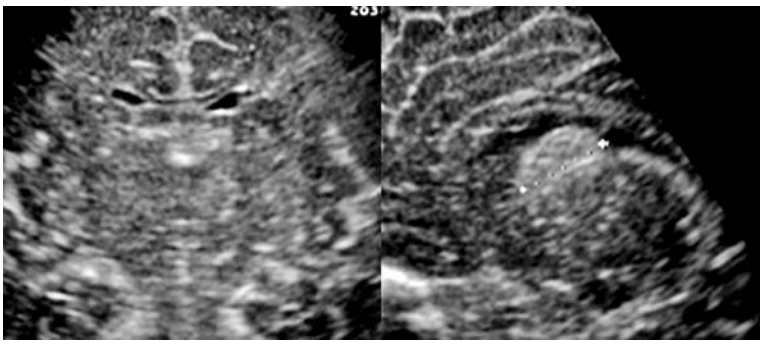

Fig 2. a) Coronal view at the frontal horns level: echogenic material on both supependimal regions, more abundant on the left; b) Sagital view: oval shaped echogenic deposit in the left thalamo-caudate groove, apparently protruding into the lateral ventricle. ***Diagnostic: subependimal grade I haemorrhage.***

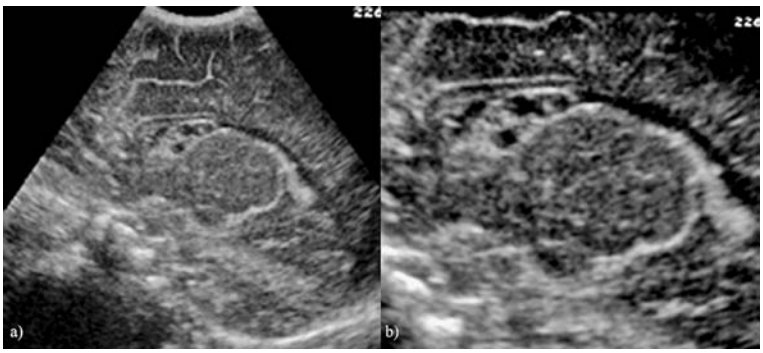

Fig 3. a) and b) Hemorrhagic process outcome: sagital section in a 8 weeks of age infant- pseudocyst protruding to the left ventricle. ***Diagnostic: subependimal haemorrhage with left residual subependimal cyst.***

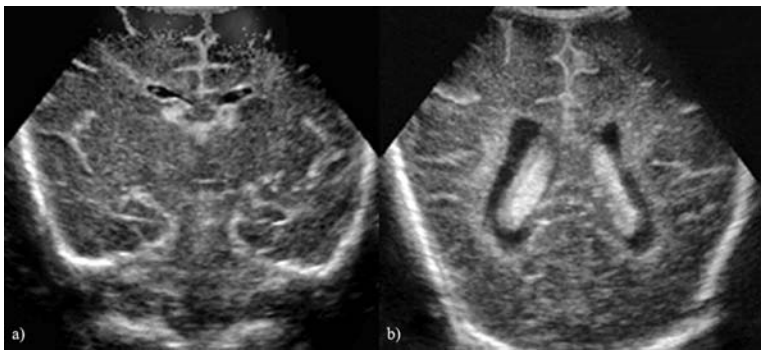

Fig 4. a) Coronal view: the thalamic and caudate grooves are filled with hyperechoic material, more abundant on the right side; b) Coronal view at the lateral ventricles body: asymmetric left ventricles: minimal right ventricle dilation. ***Diagnostic: bilateral subependimal haemorrhage with right intraventricular haemorrhage and minimal ventricular distension – grade II haemorrhage.***

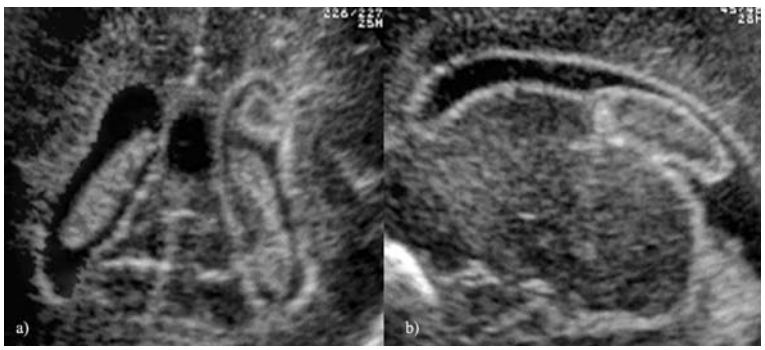

Fig 5. a) Coronal view at ventricular body level: both ventricles are distended; on the left side an echogenic clot extends to the frontal horn; b) Sagital view demonstrate oval shaped irregular clot on the left ventricle's body, distinct by echogenicity and location from the choroid plexus which can be seen posterior. ***Diagnostic: grade III intraventricular haemorrhage.***

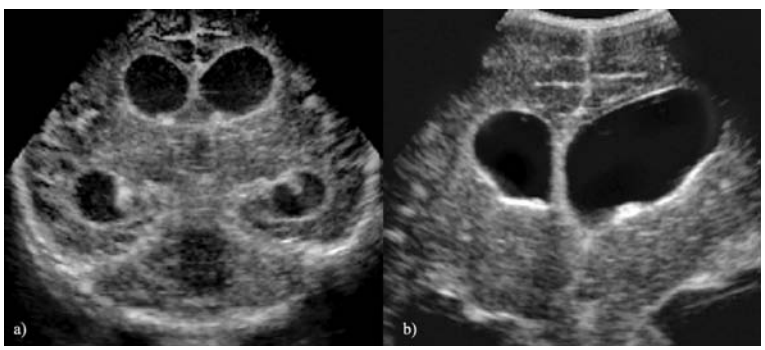

Fig 6. Grade III intraventricular haemorrhage outcome: ultrasound examination at 5 and 8 weeks postnatal. a) Coronal view: dilated frontal and temporal horns, mild IIIrd ventricle distension; b) Asymmetrical frontal and temporal horns distension. ***Diagnostic: intraventricular grade III haemorrhage with secondary hydrocephalus.***

increased risk of neurological sequels [7]. Post hemorrhagic hydrocephalus is noticed in 55-60% of cases.

Grade IV intracranial haemorrhage (fig 7-9)**.** Originally this haemorrhage was thought to represent the extinction of a subependymal bleeding into the adjacent brain parenchyma. It is now recognised that a different pathophysiologic mechanism is involved. Venous hemorrhagic infarction, resulting from outflow compression of the veins by the subependymal hemorrhage is nowadays the major incriminated mechanism [8,9]. The venous infarctions sometimes resolve with porencephalic cyst formation (fig 10). About 75-80 % of grade IV haemorrhages associate intraventricular haemorrhages, leading to uni- or bilateral ventricular dilations. Grade IV bleeds have severe long-term deficits, but the outcome is usually worse when residual marked hydrocephalus and extended parenchyma injury has occurred.

II. Hypoxic ischemic encephalopathy – periventricular leucomalacia

Based on diferences in cerebral maturation, consequences of hypoxic trauma at birth differ in preterm infants compared to full term children [8]. In preterms, the most vulnerable area to hypoxia is the periventricular white matter; on the contrary, in full term infants the profound white matter located near the cerebral cortex and the adjacent subcortical areas are the most affected. Focal or diffuse periventricular leucomalacia represent the most common white matter lesion in preterms with birth asphixia. In full term, the hypoxic ischemic encephalopathy usually manifests by cortical and subcortical white matter lesions.

The typical microcystic lesions in PVL are usually noticed in the first 10-14 days of life, explainig why the ultrasound examination performed immediately after birth usually show only periventricular hyperecogenicity with macrogranular echodensities [11,12]. Differentiation between this aspect and the normal periventricular hyperecoge-

nity of the newborn is sometimes difficult. An echogenicity equal or superior to the chorid plexuses is sugestive, but not pathognomonic for PVL. In preterms with gestational ages less than 32 weeks, cysts may develop lately, till the 4th week after birth and that's why weekly ultrasound scans are mandatory till 40 weeks of age [13].

PVL classification according to lesions extesion and severity [13]

Grade I prolonged periventricular densities, persisting more than 7 days

Grade II periventricular densities, evolving to small periventricular cysts (fig 11)

Grade III periventricular densities, evolving into extensive periventricular fronto-parieto-occipital cystic lesions (fig 12)

Grade IV densities extending into the subcortical white matter, evolving into extensive periventricular and subcortical cystic lesions. This type of lesion is more common in full term children with severe perinatal hypoxia (fig 13).

Hipoxic ischemic encephalopathy severity and neurological sequels are mainly related to location and extension degree of the lesions. At least 86% from survivors with PVL in the perinatal period may develop cerebral palsy and 64% of them will have major intellectual disabilities. Mortality rate of these cases is around 59% [14].

Ultrasonography is the method of choice for screening and follow-up of patients with PVH-IVH. Because most lesions appear in the first week of life, the initial examination is recommended on days 4-7, followed by a second one between days 14-21 and the third, at 3 month of age [7]. It is also the technique of choice for post hemorrhagic hydrocephalus follow-up; weekly ultrasonography examinations are recommended for the progression of hemorrhage follow-up and for post hemorrhagic hydrocephalus development [15].

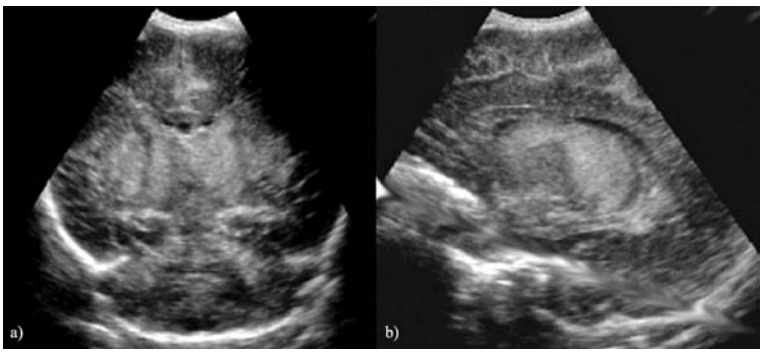

Fig 7. Full term new born with severe birth hypoxia- examination on day 5. a) Coronal view: both lateral ventricles fulfilled by an echogenic structure, consistent with bilateral intraventricular clots. Hyperechoic periventricular parenchyma, suggestive for venous infarctions; b) Sagital view at lateral ventricle level: the hyperechoic clod extends to almost entire ventricle. ***Diagnostic: grade IV intraventricular haemorrhage.***

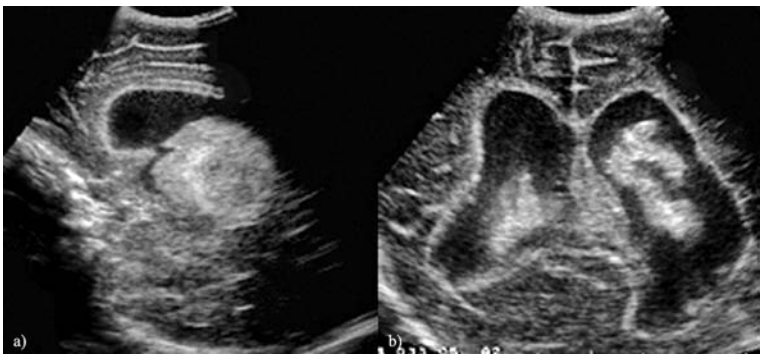

Fig 8. The same case as in fig 7 reexamined at 18 days of age. a) Sagital view: pronounced distension of the left lateral ventricle with persistent hyperechoic imagines inside. B. Coronal view: bilateral ventriculomegaly, intraventricular clot on a slow resolution process. ***Diagnostic: grade IV intraventricular haemorrhage. Post hemorrhagic hydrocephalus.***

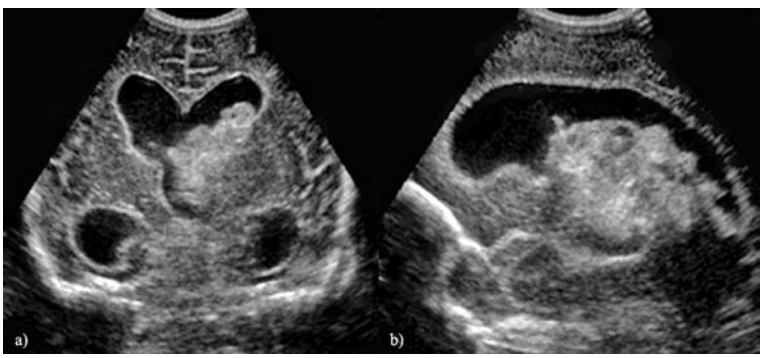

Fig 9. A 3 days old newborn, 28 weeks gestational age, birth weight 1900 gr, with severe perinatal hypoxia, Apgar score 3/1 min, 5/5 min. Head ultrasound on day 7. a) Coronal view: both frontal and temporal horns are distended by an echogenic, irregular, inhomogeneous structure inside the ventricles. Dilated third ventricle; b) Sagital view at the left lateral ventricle level: voluminous clot with irregular borders leading to secondary ventricular distension. ***Diagnostic: grade IV intraventricular haemorrhage. Secondary hydrocephalus.***

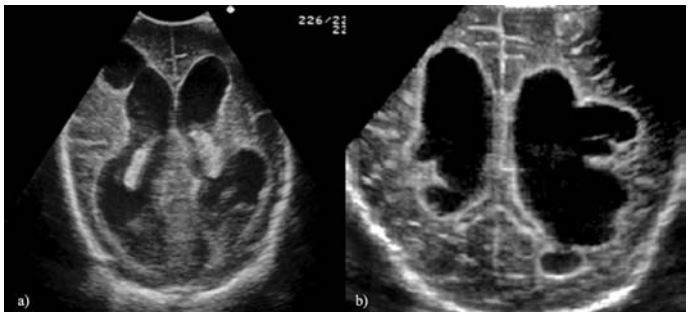

Fig 10. a) and b): Coronal views: distended lateral ventricle with porencephalic cyst formation. ***Diagnostic: grade IV intraventricular haemorrhage. Post hemorrhagic hydrocephalus, porencephalic cysts***

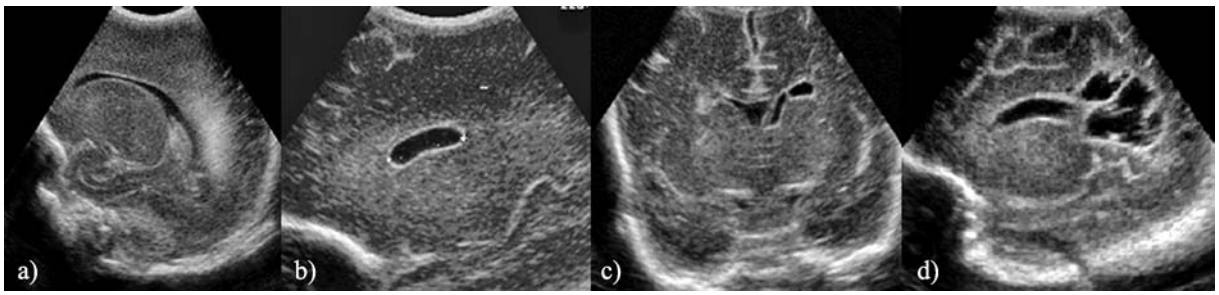

Fig 11. Preterm newborn examined for periventricular hyperechogenicities observed at 48 h of age. a) Sagital view day 3 periventricular hyperechogenicities superior to the echogenicity of choroid plexus; b) Examination on day 10: small cyst in the periventricular area; c) Coronal view cyst on the external angle of the left frontal horn; d) Sagital view: multiple periventricular cystic cavities. ***Diagnostic: grade II PVL.***

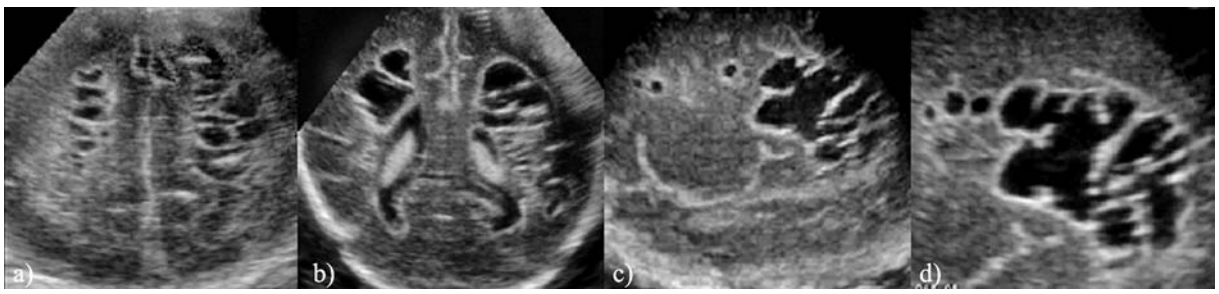

Fig 12. a) and b) Coronal views: in various cerebral regions- multiple, extensive cystic cavities on the parieto-fronto-occipital regions; c) and d) Sagital views: multiple cysts with various shapes and dimensions located on both periventricular areas. ***Diagnostic: hypoxic ischemic encephalopathy, grade III PVL.***

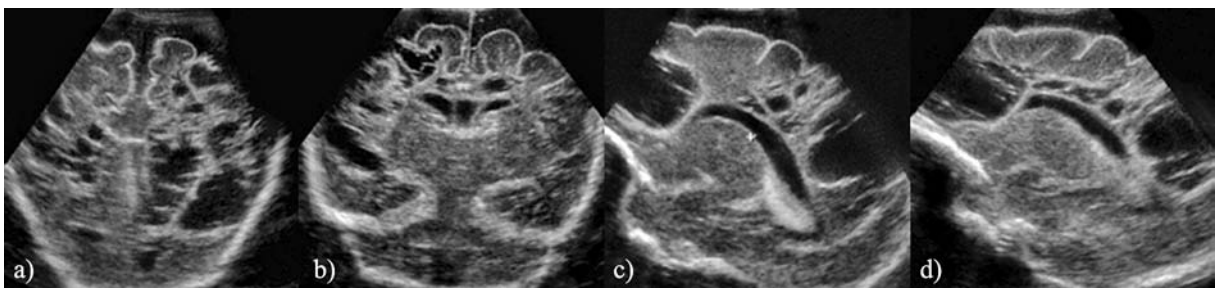

Fig 13. Preterm newborn - 36 weeks gestational age, Apgar score 2/1min, 4/5 min, examined in the 14th days of life. a) and b) Coronal views: numerous cysts of different shapes and dimensions extending periventricular bilaterally. Marked extraaxial spaces distension with anechoic content; c) and d) Sagital views: periventricular cysts with fronto-parieto-occipital extension. Enlarged subarachnoid space. Note the normal dimensions of the lateral ventricles. ***Diagnostic: hipoxic ischemic encephalopathy, grade IV PVL.***

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