ÖSSZEFOGLALÓ KÖZLEMÉNY
REVIEW ARTICLE

Characteristics of stroke-like lesions on cerebral imaging

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Objective – Stroke-like lesions (SLLs) are pathognomonic for mitochondrial encephalopathy, lactic acidosis, and stroke-like episodes (MELAS) syndrome but occur in other mitochondrial and non-mitochondrial disorders as well. This mini-review aims at summarising and discussing recent findings to open up future perspectives how to manage this fleeting phenomenon.
Results – Typically, SLLs are dynamic lesions, which increase in size and intensity to regress after a nadir. SLLs are incongruent with a vascular territory, originate frequently from the cortex to spread subcortically, can be monofocal or multifocal, run through an acute (attack) and chronic (remission) stage, and may either completely disappear or end up as laminar cortical necrosis, white matter lesion, subcortical atrophy, cyst, or the toenail sign. On cerebral CT, SLLs are hypodense. SLLs can be best visualized on multimodal MRI showing up as hyperintensity on T2, FLAIR, DWI, and PWI, and as hypointensity on OEF-MRI. On MR-spectroscopy, SLLs typically present with a decreased N-acetyl-aspartate peak and an increased lactate peak. DTI in acute SLLs reveals reduced connectivity, increased global efficiency, and reduced focal efficiency. Tc-HMPAO SPECT of SLLs indicates hyperperfusion and L-iomazenil SPECT reduced tracer uptake. FDG-PET typically shows hypometabolism within a SLL.
Conclusion – SLLs present with typical findings on various imaging modalities but the combination of cerebral CT, multimodal MRI, MRS, and PET clearly delineate a SLL from other acute or chronic cerebral lesions.

Stroke-szerű laesiók jellegzetességei agyi képalkotó vizsgálatokkal
Finsterer J, MD, PhD

Célkitűzés – A stroke-szerű laesiók (stroke-like lesion, SLL) a mitochondrialis encephalopathia, a tejsavas acidózis és a stroke-szerű epizód szindróma (stroke-like episodes syndrome, MELAS) jellegzetességei, de egyéb mitochondrialis és nem mitochondrialis betegségekben is előfordulnak. Rövid áttekintésem az SLL-ekkel kapcsolatos legújabb eredmények tárgyalja és foglalja össze abból a célból, hogy bemutassa, a jövőben hogyan lehet majd menedzselni ezt a változatos jelenséget.
Eredmények – Az SLL-ek tipikus formájukban dinamikus laesiók, amiknek először nő a méretük és az intenzitásuk, majd egy maximum elérése után regrediálnak. Kialakulásuk nem mutat szoros összefüggést az erek ellátási területével, gyakran kérgi eredetűek és subcorticalisan terjednek, mono- és multifokálisak egyaránt lehetnek, akut és krónikus fázisuk is van (roham, majd remisszió), lehetséges, hogy teljesen felszívódnak, de olyan is van, ami laminaris corticalis necrosissal, fehérállomány-laesióval, subcorticalis atrófiával, cystakialakulással vagy toenail-jel kialakulásával végződik. Az agyi CT-felvételen az SLL-ek hypodensitasként ábrázolódnak. Legjobban multimodális MR-felvételen vizualizálhatók, T2-, FLAIR-, DWI- és PWI-hiperintenzitásként, valamint OEF-MR-hipointenzitásként. MR-spektoszkópos felvételen az SLL tipikusan csökkent N-acetil-aszpartát-csúccsal és megnövekedett laktátcsúccsal jelentkezik. A DTI akut SLL esetén csökkent konnektivitást, megnövekedett globális effektivitást és csökkent fokális effektivitást mutat. Az SLL Tc-HMPAO SPECT-vizsgálata hiperperfúziót jelez, míg az L-iomazenil SPECT-vizsgálat csökkent tracer-felvételt. Az FDG-PET-vizsgálat általában az SLL-en belüli hipometabolizmust mutatja.
Következtetés – Az SLL a különböző képalkotó modalitásokra tipikus jellegzetességekkel ábrázolódik, de az agyi CT, a multimodális

MR, az MR-spektroszkópia és a PET kombinációja révén pontosan elkülöníthető a többi akut vagy krónikus cerebralis laesiótól.

Keywords: stroke-like episode, stroke-like lesion, MELAS, magnetic resonance imaging, FDG-PET, oxidative stress

Kulcsszavak: stroke-szerű epizód, stroke-szerű laesio, MELAS, mágnesrezonanciás képalkotás, FDG-PET, oxidatív stressz

Stroke-like episodes (SLEs) are a hallmark of mitochondrial encephalopathy, lactic acidosis, and stroke-like episodes (MELAS) syndrome but can occur in other mitochondrial disorders (MIDs) or non-MIDs as well[1, 2]. The morphological equivalent of a SLE on cerebral imaging is the stroke-like lesion (SLL)[1]. SLLs are typically dynamic lesions, which increase in size and intensity during a period of up to one month to regress thereafter within days, weeks, or months. SLLs are not confined to a vascular territory and usually originate from a cortical nucleus to spread subcortically. However, occasionally, SLLs occur exclusively with a subcortical distribution[3]. SLLs can be monofocal or multifocal, and may occur one by one, such that several SLLs of different age may coexist[3].

Despite recent advances in the characterisation of SLLs on imaging[4], there is still no consensus on how to define this fluctuating cerebral abnormality on imaging. Furthermore, the pathogenesis of SLLs remains unclear despite various proposals to explain the enigma. The most commonly discussed pathogenetic hypotheses include the epileptogenic, vascular, and metabolic hypothesis[5]. Recently, it was also proposed that disruption of the blood brain barrier (BBB) may have a causative role[6].

SLLs can be best visualised on multimodal MRI but they can be identified also on cerebral CT (CCT). Additionally, MR-spectroscopy (MRS), diffusion tensor imaging (DTI), single photon emission computed tomography (SPECT), and fluoro-deoxy-glucose-positron emission tomography (FDG-PET) reveal a typical pattern of SLLs. This mini-review aims at summarising and discussing recent findings to open up future perspectives how to manage this fleeting phenomenon.

Method

A PubMed search applying the search terms "stroke-like episode", "stroke-like lesion", "laminar cortical necrosis", "pseudolaminar necrosis", "toenail sign", "mtDNA", "mitochondrial", "mutation", and "MELAS" combined with "imaging", "cerebral computed tomography", "magnetic resonance", "multimodal", "magnetic resonance angiography", "magnetic resonance spectroscopy", "diffusion tensor imaging", "SPECT", and FDG-PET was carried out. After screening the abstracts, articles in English which reported human studies with cerebral imaging techniques of patients with a mitochondrial disorder (MID) that manifested with a SLE were identified. Included were only full-text articles that described in detail morphology and functional findings in SLLs.

Results

CCT

Only few reports about SLLs on CCT in patients with a SLE are available[7]. In all these patients the SLL showed up as homogeneous hypodensity not confined to a vascular territory. An example is a 48 years old Palestine female with MELAS in whom CCT revealed a hypodense lesion in the left parietal lobe[7]. In a study of 7 pediatric patients with MELAS, CCT showed a hypodense lesion in the left parieto-occipital region and at follow-up, 13 days later, a hypodense lesion in the right parieto-occipital region[8]. Hypodensity of the SLL has been also reported in a 38 years old female with MELAS[9]. SLLs on CCT are frequently associated with calcifications of the basal ganglia or the dentate nucleus. Application of contrast medium and CT angiography is crucial to delineate a SLL from various differentials, such as focal edema, tumour, or ischemic stroke. In case of a tiny tumour with big perifocal edema mimicking ischemic stroke, enhancement of the tumour can help identifying the underlying tumour. A small tumour with large bleeding where the large clot compresses and masquerades the tumour can resemble haemorrhagic stroke. Other differentials that need to be ruled out before establishing the diagnosis "SLL" include Wernicke encephalopathy, drug abuse encephalopathy, post ictal phenomena, posterior, reversible encephalopathy syndrome (PRES) and stroke-like migraine attacks after radiation therapy (SMART) syndrome.

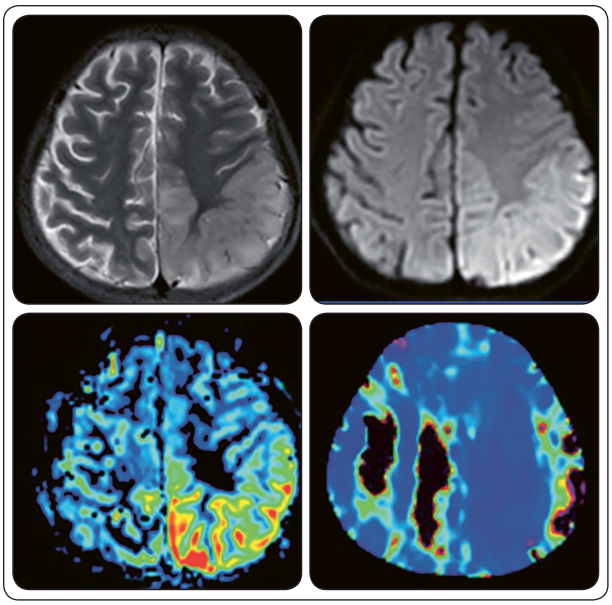

Figure 1. *MRI of a 14-year-old boy with MELAS with an acute SLL 9 days after onset showing high signal and swelling of left parietal lobe on T2WI image (upper left) and DWI image (upper right). Increased CBF is depicted in the lesion on the CBF map (lower left). The OEF is markedly decreased across the brain, especially in the lesion region (lower right). [reproduced from Yu et al, PlosOne 2013;8(11) (with permission)]*

MRI

On MRI SLLs are characterised by hyperintensity on T2, fluid attenuated inversion recovery (FLAIR), diffusion weighted imaging (DWI), and perfusion weighted imaging (PWI) **(Figure 1)**[1]. SLLs are hypointense on oxygen-extraction fraction (OEF)-MRI, indicating that impaired oxidative metabolism within neurons is no longer capable of metabolising oxygen and to meet peak metabolic demands (figure 1). As a compensatory mechanism these areas of hypometabolism are hyperperfused, as indicated on PWI and Tc-HMPAO SPECT **(Figure 1)**[10]. Hypometabolism within an SLL may not only be documented by OEF-MRI but also by FDG-PET showing that the neuronal glucose metabolism is decreased[11]. Apparent diffusion coefficient (ADC) maps are hardly useful to characterise SLLs, as they may be hyperintense, isointense, or hypointense, depending on the stage of the SLL **(Figure 2)**[12]. In areas not affected by the SLL, ADC may be higher than in healthy controls[13]. In a recent study it has been found that the cortical portions of a SLL represent a cytotoxic edema (DWI hyperintense, ADC hypointense), whereas the subcortical portions represent a vasogenic edema (DWI hyperintense, ADC hyperintense)[14]. These may give rise to mixing up SLL with ischemic stroke but other imaging techniques may help to correctly identify the SLL.

SLL run through various stages. The acute stage of a SLL (attack stage) is characterised by hyperintensity on T2, FLAIR, DWI, and PWI (hyperperfusion)[12], hypointensity on OEF-MRI, hyperperfusion on Tc-HMPAO SPECT, and hypometabolism on FDG-PET. The SLL is hypointense on T1 and does not enhance upon gadolinium[9]. The chronic stage of a SLL (remission stage) is characterised by regression of the hyperintensities and hypoperfusion, and successive normalisation of FDG-PET. SLLs may occur in a monofocal or multifocal distribution. SLLs may not only occur supratentorially but also infratentorially, including the cerebellum. Multifocal SLLs can be of different stages as they may develop one by one. SLLs may resolve spontaneously without a residual lesion but can also end up as white matter lesion, laminar cortical necrosis, focal subcortical atrophy, cyst, or as toenail sign[1]. Differentials that need to be ruled out on MRI include Wernicke encephalopathy, drug abuse encephalopathy, post ictal phenomena, PRES, and SMART syndrome.

MRS

With regard to 1H-MRS, SLLs present with a decrease in N-acetyl-aspartate (NAA) and an increase in lactate[10].

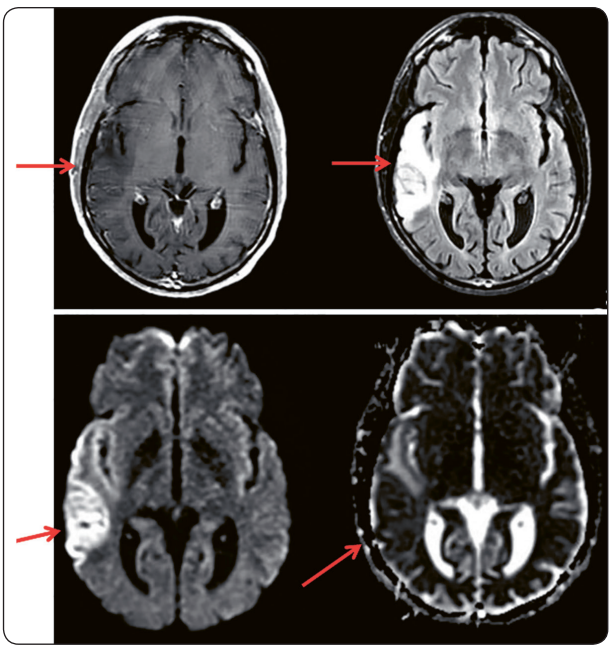

Figure 2. *Multimodal, cerebral MRI of a patient with MELAS manifesting with hearing loss and acute onset seizures showing a typical SLL in the right posterior temporal lobe with hypointensity on T1 (upper left), hyperintensity on T2 (upper right), hyperintensity on DWI (lower left), and isointensity on ADC (lower right) [reproduced from Liu et al, J Magn Reson Imaging 2013;37 (with permission)]*

MR spectroscopy of SLLs may not only show a lactate peak within the SLL but also in the surrounding area[15].

SPECT

With regard to Tc-HMPAO single photon emission computed tomography (SPECT) findings for SLLs in MELAS, an overall trend of hyperperfusion in the acute stage, which is within 1 month of onset of the SLE, and hypoperfusion in the chronic stage, which starts one month after onset of the SLE, has been reported[10]. Remarkable hyperperfusion may be particularly found if SLLs are associated with seizures[16]. In the hyperacute stage of a SLL (2nd day after onset) Tc-HMPAO SPECT may show hypoperfusion followed by hyperperfusion on day 3[17]. Hyperperfusion during an acute SLL has been also reported in a single patient undergoing Xenon-CT[18]. Hyperperfusion in acute SLLs is in accordance with temporary vasodilation of the left posterior cerebral artery (PCAS) and the left middle cerebral artery (MCAS) in a MELAS patient with a left occipital SLL[19]. Vasodilation in this patient was documented by MR angiography (MRA) and computed tomography angiography (CTA)[19]. In a SPECT study of a 31 years old female with MELAS, manifesting as SLE with focal seizures of the right hand, 123I-iomazenil SPECT showed reduced tracer uptake in the corresponding left post-central gyrus[20]. The lesion was hyperintense on FLAIR. Hyperintensity completely disappeared one month after onset of the SLE. In contrast, 123I-iomazenil SPECT still showed the reduced tracer uptake in this area[20]. It was concluded that reduced cortical benzodiazepine receptor binding potential on late images of 123I-iomazenil SPECT indicates neuronal damage in the cortex and that this damage may persist despite normalisation on MRI[20].

FDG-PET

Only few FDG-PET studies of SLLs have been carried out so far. Usually FDG-PET shows hypometabolism within SLLs[11]. In a study of 4 patients with MELAS, 6 patients with external ophthalmoplegia, and 5 patients with mitochondrial myopathy, cerebral glucose uptake was impaired particularly in the occipital and temporal regions in all patients disregarding the presence or absence of clinical cerebral manifestations[21]. Unfortunately, it was not indicated how many of these patients presented with an acute SLE. In a study of a MELAS patient by means of a combination of a double PET method with the tracers [(62)Cu]-diacetyl-bis(N4-methylthiosemicarbazone) ((62)Cu-ATSM) and [(18)F]-fluorodeoxyglucose ((18) FDG) to visualise regional oxidative stress and glucose metabolism and blood flow in SLLs non-invasively and rapidly, it was demonstrated that oxidative stress and hyperemia along with increased glucose metabolism play a crucial role in the pathogenesis of SLLs[22].

DTI

In a recent DTI study of MELAS patients with SLEs Wang et al. identified four dynamic functional connectivity (dFC) states[4]. Particularly in the acute stage of a SLL MELAS patients spent more time in a state with weaker

Table 1. *Imaging identifiers of SLLs*

Imaging modality	Acute (attack) stage	Chronic (remission) stage
CCT		
Native images	hypodense	hypodense
CTA	vasodilation	nr
MRI		
T1	hypointense	nr
T2	hyperintense	hyperintense
FLAIR	hyperintense	hyperintense
DWI	hyperintense	hyperintense
PWI	hyperintense	hypointense
OEF-MRI	hypointense	nr
MRA	vasodilation	nr
Enhancement	none	nr
MRS	NAA ↓, lactate↑	nr
SPECT		
Tc-HMPAO	hyperperfusion*	hypoperfusion
Iomazenil	tracer uptake ↓	tracer uptake ↓
PET		
FDG	hypometabolism	nr
DTI		
Connectivity	↓	nr
Global efficiency	↑	↓
Local efficiency	↓	↑
fMRI		
Spontaneous activity	↓	recovering

NAA: N-acetyl-aspartate, nr: not reported, *: in the hyperacute stage (day 1-2) hypoperfusion has been reported

connectivity and less time in states with stronger connectivity. In addition, volumes of acute SLLs were positively correlated with mean dwell time and negatively correlated with the number of transitions. Furthermore, acute SLLs exhibited significantly increased global efficiency and decreased local efficiency compared to controls and chronic SLLs[4]. Chronic SLLs only showed significantly decreased local efficiency compared to controls. It was concluded that there are similar and distinct dFC alterations within SLLs during the acute and chronic stages, providing novel insights for understanding the neuropathological mechanisms of SLLs[4]. In a single MELAS patient presenting with a SLL, DTI revealed volume loss in the chronic stage of the SLL[23].

fMRI

In one of the few fMRI studies, in 20 patients experiencing an acute SLL (attack stage) and 20 patients in the remission stage, Wang et al. found that spontaneous cerebral activity was decreased within the area of the SLL and that spontaneous activity was less decreased beyond the SLL area[24]. It was concluded that the fractional amplitude of low frequency fluctuations in the left precuneus may be a useful biomarker for monitoring the disease status in MELAS[24].

Conclusion

SLLs are, according to imaging findings **(Table 1)**, the morphological equivalent of a focal or regional metabolic disruption in neuronal, glial, endothelial, or vascular smooth muscle cells of the brain. Metabolic disruption most likely is triggered by stressors which demand excess metabolic output that cannot be provided by the genetically defective oxidative metabolism. The increased demand results in a breakdown of cell functions, which may either result in cell necrosis or temporary failure to meet peak metabolic needs. SLLs not only occur in adults[25] but also in pediatric patients[26].

DECLARATIONS – The authors declare no conflicts of interest. No funding was received. Informed consent was obtained from all individual participants included in the study.

ETHICS APPROVAL – The study was approved by the institutional review board. All procedures performed in the studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

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