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Review

Intracranial evaluation and laser ablation for epilepsy with periventricular nodular heterotopia

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ABSTRACT

Surgical treatment of focal epilepsy in the presence of periventricular nodular heterotopia (PVNH) poses a challenge, as the relative roles of the nodular tissue and the overlying cortex in the generation of seizures can be complex and variable. Here, we review the literature on chronic invasive EEG recordings in humans with this substrate and present two illustrative cases from our practice. We found that while inter-ictal spiking from nodules is common, clinical seizures rarely arise solely from nodular tissue. More typically, ictal onset is simultaneous with overlying neocortex or mesial temporal structures. Surgical outcome is more favorable in cases with unilateral (as opposed to bilateral) PVNH, and when a substantial or complete ablation of PVNH is performed. In rare cases, nodular ablation alone may be sufficient, as may be completed by MRI-guided laser interstitial thermal therapy. The mechanism(s) by which PNVH interacts with overlying cortex are not fully understood, but we suggest that PVNH either orchestrates or amplifies local network epileptogenicity. At present, invasive recordings with penetrating depth electrodes are required prior to surgical therapy, as illustrated in our cases.

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1. Introduction

The disruption of radial neuronal migration during brain development results in malformations composed of heterotopic gray matter [1]. These may form continuous bands (laminar heterotopia) or discrete nodules, either in subcortical or periventricular locations (periventricular nodular heterotopia, PVNH). PVNH typically resides along the walls of the ventricular trigone, either unilaterally or bilaterally [2]. Nodular heterotopia is often associated with other types of cortical malformation, including polymicrogyria (PMG) [3] and focal cortical dysplasia [4], and is a common cause of refractory focal epilepsy [5,6].

The presence of epileptiform activity in heterotopic gray matter was first demonstrated by Morrell et al. [7] over two decades ago, yet the role of heterotopia in the pathophysiology of epilepsy remains controversial. Specifically, the question of how the PVNH should factor into the surgical treatment of the associated focal epilepsy is not well answered [8]. PVNH may also be associated with hippocampal sclerosis [9], raising the question of whether or not PVNH is independently epileptogenic, as a 'dual pathology' [10]. Recent studies have investigated the functional and structural connectivity between PVNH and overlying cortex noninvasively. EEG–fMRI [11,12], and diffusion tractography [13] remain promising tools in the pre-surgical work up; nevertheless, invasive EEG recordings are typically indicated to more fully delineate whether or not PVNH is within the putative epileptogenic zone. There are only a few case series and case reports describing chronic invasive EEG recordings in PVNH, and most of these utilize an SEEG approach (Table 1). From these we hope to understand the complex inter-ictal and ictal neurophysiology of PVNH.

2. Inter-ictal neurophysiology

The two largest case series on PVNH differ somewhat in their inter-ictal findings. Tassi et al. [2] found inter-ictal spiking in the PVNH of all patients, though not in all sampled nodules. Spikes were typically asynchronous between nodules, synchronous with neocortex, but seldom synchronous with mesial temporal structures. In contrast, Aghakhani et al. [14] demonstrated independent nodular spiking in only 3 out of 10 cases; however, these same

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Table 1
Summary of reported cases of heterotopia with chronic intracranial recordings.

References	Case	Gender	Age	B/L	U/L	Invasive	Note	Interictal	Nodule only	Nodule + neocortex	Neocortex only	Nodule + mesial	Mesial only	Resection	Ablation	Note	Engel score
Tassi 2005 [2]	1	M	39		–			NA	NA	NA	NA	NA	NA	T	total	CD	Ia
	2	F	38		–	–		–		5 cases (of 8)	3 cases (of 8)	2 cases (of 6)		T	partial	CD	Ia
	3	F	33		–	–		–						T/O	total		Ia
	4	F	24		–	–		–						T	partial	CD	Ia
	5	F	20		–	–		–						T/O	partial	CD	Ia
	6	M	27		–	–		–						T	partial	CD	Ia
	7	M	24		–	–		–						T/O/P	partial	CD	Ia
	8	F	40	–		–	no nodular sampling	NA	NA					F	none	CD	IIIa
	9	M	39	–		–		–						T/O/P	partial	CD	IIIa
	10	M	21	–		–		–						T/O/P	partial	CD	IIa
Aghakhani 2005 [14]	1	F	37	–		–							–	SAH	none		III
	2	M	43		–	–							–	SAH	none	contralateral to nodule	Id
	3	M	39		–	–						–		SAH	partial		Ia
	4	M	31		–	–		–				–		SAH	total		Ia
	5	M	43		–	–		–		–				O	total		Id
	6	F	16	–		–		–	subclinical	–				SAH	partial		IV
	7	F	26		–	–				–				none			NA
	8	F	38	–		–			subclinical	–				none			NA
Li 1997 [10]	1	M	23		–	–	no nodular sampling	NA						ATL	none	HS	IIIa
	2	F	28	–		–	no nodular sampling	NA						ATL	none	HS	IIIa
	3	M	27	–		–	no nodular sampling	NA						ATL	none	HS	IIIa
	4	F	33	–			no nodular sampling	NA						ATL	none	HS	Iva
	5	F	37	–		–	no nodular sampling	NA						SAH	none		IIIa
	6	M	28	–		–	no nodular sampling	NA						ATL	none	HS	IIIa
	7	F	25		–		intraop sampling							ATL	partial		IIb
	8	F	19	–			no nodular sampling	NA						ATL	partial		IVb
	9	M	25	–			no nodular sampling	NA						ATL	partial		Ia
	10	M	27		–	–	no nodular sampling	NA						ATL	none	HS	IVb
Dubeau 1995 [5]	4	M	27	–		–		NR		–				ATL	partial	HS	III
	7	F	34	–		–		NR					–	none			
	8	M	19		–	–		NR				–	–	none			
	16	M	38	–		–		NR			–			ATL	none		IV
Kothare 1998 [19]	1	M	19		–	–	no cortical sampling	NR	–					none			
	2	M	32	–		–	no cortical sampling	NR	–					none			
	3	F	21		–	–	no cortical sampling	NR	–			–		none			
Scherer 2005 [20]	1	M	23		–	–		–	–					PVNH only	total		seizure free
Francione 1994 [28]	1	F	29		–	–	subcortical nodule	–				–		T	total	HS	seizure free
Mai 2003 [29]	1	F	19	–		–	laminar heterotopia	–		–			–	T	partial		90% reduced
Agari 2012 [22]	1	M	35		–	–	SDG + depth			–				PVNH only	total		seizure free
Kitaura 2012 [24]	1	M	22	–		–	SDG + depth	NR				–		ATL	partial		seizure free
Valton 2008 [23]	1	M	41	–		–		–	–			–		NR			
Schmitt 2011 [30]	1	M	56		–	–	no cortical sampling	NR	–					PVNH only	total	RF	Ib
Esquenazi 2014 [26]	1	F	48		–	–	SDG + depth			–				PVNH only	complete	LITT	Ivb
	1	M	25	–		–	SDG + depth	–		–			–	ATL	partial	LITT	IIIa
Wu 2012 [21]	1	F	25		–	–	SDG + depth	–	–					PVNH only	total	SRS	I
	2	F	26		–	–	SDG + depth	–		–				PVNH only	total	SRS	I
	3	M	27		–	–	SDG + depth	–	–	–				PVNH only	total	SRS	I

B/L: bilateral; U/L: unilateral; NA: not applicable; NR: not reported; SDG: subdural grid; SAH: selective amygdalohippocampectomy; ATL: anterior temporal lobectomy; HS: hippocampal sclerosis; CD: cortical dysplasia; RF: radiofrequency; LITT: laser interstitial thermal therapy; SRS: stereotactic radiosurgery