PAPER

Localising and lateralising value of ictal piloerection

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Background: Piloerection is a rare clinical symptom described during seizures. Previous reports suggested that the temporal lobe is the ictal onset zone in many of these cases. One case series concluded that there is a predominant left hemispheric representation of ictal cold. The aim of this study is to evaluate the localising and lateralising value of pilomotor seizures.

Methods: Medical records of patients who underwent video electroencephalogram (EEG) monitoring at the Cleveland Clinic between 1994 and 2001 were reviewed for the presence of ictal piloerection. The clinical history, physical and neurological examination, video EEG data, neuroimaging data, cortical stimulation results, and postoperative follow ups were reviewed and used to define the epileptogenic zone. Additionally, all previously reported cases of ictal piloerection were reviewed.

Results: Fourteen patients with ictal piloerection were identified (0.4%). Twelve out of 14 patients had temporal lobe epilepsy. In seven patients (50%), the ictal onset was located in the left hemisphere. Four out of five patients with unilateral ictal piloerection had ipsilateral temporal lobe epilepsy as compared with the ipsilateral side of pilomotor response. Three patients became seizure free after left temporal lobectomy for at least 12 months of follow up. An ipsilateral left leg pilomotor response with simultaneously recorded after-discharges was elicited in one patient during direct cortical stimulation of the left parahippocampal gyrus.

Conclusions: Ictal piloerection is a rare ictal manifestation that occurs predominantly in patients with temporal lobe epilepsy. Unilateral piloerection is most frequently associated with ipsilateral focal epilepsy. No hemispheric predominance was found in patients with bilateral ictal piloerection.

Ictal piloerection (goose bumps) is a rare, frequently overlooked, clinical symptom during seizures. It was first described in 1896[1] and its prevalence was estimated at 1.2% in a group of 420 patients with pharmaco-resistant temporal lobe epilepsy.[2] Pilomotor seizures are classified as a subtype of autonomic seizures[3] and are rarely the principal ictal manifestation.[4 5] Ictal piloerection may be distributed unilaterally or bilaterally, occasionally with a somatotopical spread pattern.[6] To date, 54 cases of ictal piloerection have been reported in the literature.[1 2 4–27]

The localising and lateralising value of ictal piloerection is unclear. We studied patients with ictal piloerection to evaluate its localising and lateralising value, and compared our findings to previously reported patients.

METHODS

Medical records of 3500 consecutive patients who underwent video electroencephalogram (EEG) monitoring at the Cleveland Clinic between 1994 and 2001 were reviewed for the presence of ictal piloerection. The patient population consisted of 75% with focal epilepsy (temporal: 48%; extratemporal: 19%; non-classifiable focal: 8%), 5% with generalised epilepsy, 1% with multifocal epilepsy, and 19% with non-epileptic seizures.

Patients were monitored with scalp electrodes according to the 10–20 international system.[28] In selected patients, sphenoidal electrodes and additional electrodes were placed over the temporal regions according to the 10–20 system. Patients were included if ictal piloerection was either recorded on video, observed by neurological staff, or described by the patient. Clinical history, physical and neurological examination, video EEG data, neuroimaging data, cortical stimulation results, and postoperative follow ups were reviewed. Semiological characteristics of the seizures, video EEG data, and neuroimaging data were used to define the epileptogenic zone. In this study, the seizure classification system currently applied at the Cleveland Clinic Foundation was used.[3 29–31]

RESULTS

Fourteen right-handed patients (ten males, four females) with ictal piloerection were found (table 1). None of these patients had ictal piloerection as the sole seizure manifestation. Ictal piloerection was documented in nine cases by observation and video recordings and in five cases by the patients' history. In 12 cases (85%), the suspected epileptogenic zone was located in the temporal lobe as demonstrated either by EEG (three patients), neuroimaging findings (six patients), or by seizure freedom after temporal lobectomy (three patients). In the other two cases the location of the epileptogenic zone could not be defined with certainty because of seizure recurrence in spite of temporal lobectomy. In seven patients (50%), the ictal EEG onset was located in the right hemisphere and one patient had pilomotor seizures with ictal EEG seizure patterns arising independently from the left and right hemisphere. Nine patients (64%) experienced bilateral ictal piloerection and five patients had unilateral (or initially unilateral) ictal piloerection. Five out of the nine patients (56%) with bilateral piloerection had right temporal lobe epilepsy. Four out of the five patients (80%) with unilateral (or initially unilateral) ictal piloerection had the ictal onset in the hemisphere ipsilateral to the side of piloerection. Five patients underwent left and two patients right temporal lobectomy. Three patients became seizure free for at least 12 months of follow up, in two patients follow up is pending, and two patients continue to have seizures 2 and 6 months after surgery but only one of

Abbreviations: CNS, central nervous system; EEG, electroencephalogram; MRI, magnetic resonance imaging; PET, positron emission tomography

Table 1 Fourteen patients with ictal piloerection

Age, sex, handedness	Related condition	Seizure semiology (frequency)	Awareness of pilo	Distribution of pilo	EEG	Imaging findings	Surgery, outcome
36, M, R	None	Olfact/gust aura →automotor (LOC) →pallor/diaphoresis, L pilo (1/d)	No	L body, ipsilateral	Interictal: LT 90%, RT 10% Ictal: LT	MRI: normal	–
41, M, R	Head trauma	HV/pilo/diaphoresis (→arm and leg tingling) (→GTC) (3/d)	Yes	Bilateral	Interictal: IRS LT Ictal: LT	MRI: normal	–
51, M, R	None	Cold, pilo →nausea, SOB, loneliness, distress, tachycardia (1–2/d)	Yes	Bilateral	Interictal: RT Ictal: RT	MRI: R HA	–
15, F, R	TS, mild DD	Cold, pilo, nausea →LOC, automatisms (→GTC) (1–2/d)	Yes	Bilateral	Interictal: RT Ictal: RT	MRI: R HA and multiple tubers PET: RT hypometabolism Ictal SPECT: R anterior T hyperperfusion	RT, seizures returned 2 mo after surgery (1/mo)
	S/p RT lobectomy	After surgery: pilo/cold (1/mo)	Yes	Bilateral	Ictal: R FP Interictal: LT 75%, RTP 25%	MRI: S/p RT lobectomy, multiple bil hamartomas	
27, M, R	None	L arm pilo, cold, diaphoresis, SOB→automatisms (LOC) →GTC (2/week)	Yes	Initial L body, ipsilateral	Interictal: LT 65%, RT 35% Ictal: LT	MRI: cavernous angioma L superior T gyrus PET: LT hypometabolism	L superior T, with intraoperative language mapping, seizure free
21, M, R	None	Hyperventilation, gust aura, hypersalivation, pilo→LOC, automatisms, hyperlacrimation (→GTC) (3/week)	No	Bilateral	Interictal: LT Ictal: L T	MRI: L T MCD PET: L>RT hypometabolism	LT, rare auras returned after 6 mo (hand tingling and gust, 4/y)
57, M, R	None	Paresthesias in the nose, light-headed, bilateral arm and leg pilo (2/week)	Yes	Bilateral	Interictal: none Ictal: R T	MRI: R HA	–
24, F, R	Head trauma age 8 mo	Two seizure types were recorded: 1. warning (strange feeling), perioral paresthesias, staring, LOC, automatisms (3/week) 2. staring, LOC, pilo R postictally (1/week)	 No	 R body, ipsilateral	Interictal: RT 95%, LT 5% 1. Ictal: LT 2. Ictal: RT	MRI: L HA	–
53, M, R	Head trauma age 36	Abd aura, pilo both arms (GTC) (5–6/d)	Yes	Bilateral	Interictal: RT Ictal: RT	MRI: R HA	–
21, M, R	Meningitis age 1 month, febrile convulsions age 1 mo	Pilo L leg, then bil, cephalic sensation, staring, LOC, hand automatisms, R arm stiffening, occasional postictal aphasia (1/d)	Yes	Initial L body, ipsilateral	Interictal: LT Ictal: LT, confirmed by invasive recordings	MRI: L HA and L FP encephalomalacia PET: LT hypometabolism	LT, seizure free
45, M, R	Head trauma in childhood	Abd and olf aura, light-headed, difficulties understanding, bil pilo (15/d)	No	Bilateral	Interictal: LT Ictal: LT	MRI: LH enlargement, enhancement of LH head after gad Path: CNS-vasculitis	LT, seizure-free
49, M, R	None	Cephalic sensation, anxiety, tachycardia, pilo (1–2/d)	No	Bilateral	Interictal: RT Ictal: RT	MRI: normal	–
53, F, R	Low grade glioma	Urinary urge, fear, tachycardia, HV, abd aura, bil pilo, chills, L face tonic, GTC (1/mo)	Yes	Bilateral	R TP	MRI: R FT low-grade glioma PET: R hemispheric hypometabolism	LT, lost to follow up
54, F, R	AVM rupture	HV, chills, fear, tachycardia, shaking, L arm pilo, then bil, urinary urge, automatisms, no LOC (3–4/mo)	Yes	Initial L body, contralateral	Interictal: RT 90%, RTP 10% Ictal: RT, confirmed by invasive recordings	MRI: R superior T encephalomalacia after AVM rupture	RT, follow up pending

→, evolves into; A, atrophy; abd, abdominal; AVM, arteriovenous malformation; bil, bilateral; CNS, central nervouse system; d, day; DD, developmental delay; EEG, electroencephalogram; F, female; GAD, gadolineum; GTC, generalised tonic-clonic seizure; gust, gustatory; H, hippocampal; HV, hyperventilation; IRS, intermittent rhythmic slow; L, left; LOC, loss of consciousness; M, male; MCD, malformation of cortical development; mo, months; MRI, magnetic resonance imaging; olfact, olfactory; R, right; SOB, shortness of breath; T, temporal; O, occipital; P, parietal; PET, positron emission tomography; pilo, piloerection; S/p, status post; SPECT, single photon emission computed tomography; TS, tuberous sclerosis; yr, year.

them still has seizures with piloerection. Repeated monitoring in this case demonstrated seizures arising from the right fronto-parietal region.

Piloerection in our 14 patients was preceded by fear (three patients), nausea (three patients), loss of consciousness (three patients), olfactory auras (two patients), gustatory auras (two patients), cephalic auras (two patients), loneliness (one patient), and automatisms (one patient). Piloerection was followed by automatisms (five patients), loss of consciousness (three patients), nausea (two patients), unilateral tonic seizures (two patients), cephalic auras (two patients), bilateral somatosensory aura (one patient), and perioral or nasal paresthesias (two patients) (table 1). Associated autonomic features included shortness of breath and hyperventilation (six patients), tachycardia (four patients), ictal cold (three patients), diaphoresis (three patients), urinary urge (three patients), pallor (one patient), salivation (one patient), and hyperlacrimation (one patient). Nine patients were aware of the piloerection during and after the seizure, whereas five patients did not notice the ''goosebumps''.

Magnetic resonance imaging (MRI) showed a lesion in 11/14 cases: four patients had hippocampal atrophy; two had hippocampal atrophy associated with fronto-parietal encephalomalcia in one and multiple bilateral tubers due to tuberous sclerosis in another; one patient had a low-grade glioma; one patient had a cavernous angioma; and another patient had encephalomalacia because of a ruptured arteriovenous malformation. Furthermore, temporal malformation of cortical development with bilateral frontal and occipital encephalomalacia was found (one patient), and an enlarged hippocampal formation because of suspected central nervous systems (CNS) vasculitis (evidence by pathology; one patient) was seen.

Case report (case 6)

A 21 year old right-handed man with intractable left mesial temporal lobe epilepsy underwent video EEG monitoring in November 1997 and in January 1998. His seizures were characterised by oral automatisms and an altered state of consciousness. Seizures were usually preceded by unilateral ictal piloerection on his left arm. Postictal aphasia occurred occasionally. Epilepsy risk factors included a suspected neonatal bacterial meningitis and a single febrile seizure at the age of 1 year. Neurological examination and neuropsychological testing were normal except for decreased verbal memory testing scores. Video EEG monitoring with scalp and sphenoidal electrodes showed interictal intermittent left temporal slowing. Ictal EEG demonstrated left temporal seizure onset with the maximum amplitude at the left sphenoidal electrode. MRI showed left temporal malformation of cortical development, but also bilateral frontal and occipital encephalomalacia. Positron emission tomography (PET) revealed left temporal hypometabolism. Intracarotid amobarbital test showed left hemispheric speech dominance and predominantly right hemispheric memory representation.

The patient underwent invasive EEG recording with subdural grid electrodes and cortical stimulation because of suspected dual pathology. Eight subdural grids, including a total of 128 electrodes, were placed over the left hemisphere covering the frontal convexity, the orbital frontal area, the anterior lateral basal temporal area, the lateral temporal convexity, the anterior mesial frontal area, the posterior frontal mesial area, the anterior basal temporal area, and the posterior basal temporal area. The most mesial electrodes of the plate over the anterior temporal area covered the left parahippocampal gyrus. The most frequent interictal spikes were recorded from the basal temporal plates with highest amplitude at the most mesial electrodes. No spikes were seen in the mesial frontal or orbital frontal plates. The EEG seizure onset started from the two most mesial electrodes of the basal temporal plates.

During direct cortical stimulation of the left parahippocampal gyrus, a pilomotor response in the ipsilateral left leg was observed simultaneously with an after-discharge confined to the stimulated electrode. A left anterior temporal lobectomy was performed and histology revealed malformation of cortical development. Rare seizures returned 6 months after surgery, but seizure semiology changed and did not include piloerection postoperatively (table 1).

DISCUSSION

Ictal piloerection occurs predominantly in patients with temporal lobe epilepsy. This is consistent with previously reported cases as evidenced by EEG[2 4 6 9 11 12 14 17 19 22–24 26] and imaging.[2 4 6 11 14 23–26] Only a few exceptions have been described with seizures arising from the frontal,[7 19] parieto-occipital,[5] fronto-parietal,[13] and fronto-temporal[8] brain regions.

Although a previous case series found left hemispheric epilepsy in 18/26 patients (69%) with ictal cold shiver (five patients), ictal piloerection (nine patients), or both (12 patients),[9] the lateralising value of ictal piloerection seems to be less clear. In our series, six out of 14 (43%) patients had left hemispheric epilepsy. In 23/38 (61%) of reported patients with ictal piloerection in whom the lateralising information was provided, the epileptogenic zone was most likely in the left hemisphere.[2 6 9 17 18 21–27] Therefore, ictal piloerection in general seems to have no lateralising value. The left hemispheric predominance in the series of Stefan *et al*[2 9] may be related to the inclusion of patients with the feeling of ictal cold.

In contrast, unilateral (or initially unilateral) ictal piloerection is usually associated with ipsilateral seizure onset. Four of our five patients with unilateral onset of ictal piloerection had ipsilateral focal epilepsy. Review of the literature revealed 18 cases with clearly unilateral (or initially unilateral) ictal piloerection (table 2).[7 9–11 13 14 16 22–27] In 14 of these patients, the epileptogenic zone could be lateralised. Twelve of these 14 patients (86%) had an epileptogenic zone that was in the hemisphere ipsilateral to the side of ictal piloerection as documented by successful epilepsy surgery[7 9 26] and by imaging and EEG findings.[9–11 14 16 23–27] In four patients, the epileptogenic zone could not be lateralised.[9 13 27] Only two previously reported cases and one of our cases showed ictal piloerection contralateral to the (probable) hemisphere of seizure onset.[9 22] In summary, we found 19 patients with unilateral ictal piloerection in whom the epileptogenic zone could be lateralised. In 16 of these patients (84%), ictal piloerection was ipsilateral to the epileptogenic zone.

In our patients, ictal piloerection was never the only seizure manifestation. This corresponds with descriptions of ictal piloerection in conjunction with an epigastric sensation,[4–6 8 9 12 14 22 24] feelings of fear or other experiential sensations,[4 8 9] olfactory hallucinations,[9 10 12 25] unspecified warning sensations,[4] or localised tingling.[1 13 18 27] Additional autonomic features in the literature include descriptions of shivering or vasomotor symptoms in the literature.[1 2 4 9 10 14–17 20–27] We cannot exclude the possibility that piloerection could be secondary to psychic auras with anxiety or fear, or secondary to fear of an impending seizure. However, ictal piloerection was the first seizure symptom in five out of 25 patients with detailed seizure descriptions.[7 15 23 24 26 27]

In some of our patients there were other clinical symptoms regularly following the piloerection—for example,

Table 2 Literature review of 18 patients with documented unilateral (or unilateral onset of) ictal piloerection

Author, year	Age, sex	Symptomatology	Side of anatomical lesion	Side of EEG abnormality		Distribution, lateralisation of the epileptogenic zone
				Interictal	Ictal	
Féré, 1904[13]	53, M	Visual aura/somsens aura right body/pilo	–	–	–	R body, epileptogenic zone not defined
Landau, 1953[16]	29, M	Psychic aura→cold/pilo	–	R temporal	–	R body, ipsilateral
Green, 1984[14]	44, M	abd rising→pilo/cold→(olfact)	R temporal	R fronto-temporal	R fronto-temporal	Initial R body, ipsilateral
Andermann, 1984[10]	61, M	Olfact→pilo/cold	R fronto-temporal	–	–	R body, ipsilateral
Tyndel, 1986[25]	56, M	Olfact→pilo/fear/cold	L temporo-parietal	–	–	Initial L body, ipsilateral
Scoppetta, 1989[23]	58, M	Cold/pilo→LOC	L temporal	–	L temporal	Initial L body, ipsilateral
Yu, 1998[26]	29, M	Fear→cold/pilo→automotor (LOC) (GTC)	L temporal	L temporal	–	Initial L body, ipsilateral
Yu, 1998[26]	37, M	Cold/pilo/fear	L temporal	–	L temporal	Initial L body, ipsilateral
Baumgartner, 2001[11]	50, –	Pilo	R temporal	–	No EEG change*	R arm, ipsilateral
Roze, 2000[22]	66, M	abd→flush/warmth→pilo	–	–	L fronto-temporal	R body, contralateral
Stefan, 2001[9 24]	17, M	abd rising→cold/pilo→automotor (LOC)	L temporal	–	L temporal	L body, ipsilateral
Sa'adah, 2002[27]	26, M	Cold/pilo→LOC	L temporal	L temporal	–	Initial L leg, ipsilateral
Sa'adah, 2002[27]	23, F	Unclear aura→cold/pilo→automotor	–	L and R temporal	–	Initial L leg, epileptogenic zone not defined
Stefan, 2003[9]	41, F	(?)→pilo →cold abd	–	–	–	Initial R arm, epileptogenic zone not defined
Stefan, 2003[9]	36, F	(?)→cold →pilo	–	–	–	Initial L arm, epileptogenic zone not defined
Stefan, 2003[9]	59, F	(?)→cold →pilo nausea	L temporal (?)	–	–	R arm, contralateral
Stefan, 2003[9]	49, F	(?)→cold →pilo sweating, palpitation	L temporal	–	–	Initial L leg, ipsilateral
Seo, 2003[7]	27, M	Pilo→automotor (LOC)	R medial frontal	–	R frontal	Initial R leg, ipsilateral

→, evolves into; (), only occasionally following; abd, abdominal; EEG, electroencephalogram; F, female; GTC, generalised tonic-clonic seizure; gust, gustatory aura; L, left; LOC, loss of consciousness; M, male; olfact, olfactory aura; pilo, piloerection; R, right; somsens, somatosensory; unsp, unspecific.
*Right and left temporal seizures were recorded previously without associated piloerection.

automatisms and loss of consciousness (table 1). Only in nine cases reported previously did pilomotor seizures usually develop into automotor seizures or loss of consciousness with staring.[1 7] This may be because of the fact that piloerection may go unnoticed if there are other prominent symptoms immediately following, particularly if these symptoms are impressive and involve loss of consciousness.

The generator of ictal piloerection is most likely located in close vicinity to the central autonomic network, which includes the insula and medial prefrontal cortex, the central nucleus of the amygdala, the preoptic region, the hypothalamus, the midbrain periaquaeductal grey matter, the pontine parabrachial region, the nucleus of the solitary tract, and the intermediate reticular zone of the medulla.[32] Piloerection has been elicited by electrical or pharmacological stimulation in humans and animals at multiple sites—for example, the insula,[33] hippocampus, amygdala,[34–36] hypothalamus,[37] midbrain reticular core,[38] and medial prefrontal cortices.[34 39 40]

The insula is involved in higher order viscerotopically organised autonomic control[11] and is connected with lateral-temporal, limbic, and frontobasal cortical structures.[41] Seizures starting in the mesial temporal region can spread to the insula, thereby producing epigastric auras and autonomic seizures, including piloerection.

Direct cortical stimulation of the left parahippocampal gyrus produced an ipsilateral pilomotor response with simultaneously recorded after-discharges in one of our patients. Fish *et al*[34] found piloerection following amygdala stimulation without or only focal after-discharge. Electrical stimulation of the amygdala in unanesthetised cats produced a fear response,[36] including piloerection. However, piloerection may be secondarily mediated, and not directly elicited by mesial temporal stimulation.

In one patient, subdural EEG recordings gave evidence for ictal onset in the cingulate gyrus.[7] Additionally, electrical stimulation of the rostral cingular cortex in monkeys resulted in generalised piloerection.[39 40] However, because of a lack of documentation of after-discharges, piloerection because of spread of the initial discharge cannot be excluded in this case.[7]

Ipsilateral piloerection was also seen after electrical stimulation of the posterior hypothalamic region.[37] This area is closely connected with the hippocampus through the fornix. The study was also not controlled for after-discharges.

Because of the fact that patients are not always aware of ictal piloerection and because of the fact that not all video recordings are sensitive enough to detect ictal piloerection we may have missed patients with piloerection, which may have lead to a sampling bias in our retrospective study. Prospective investigation of this ictal phenomenon with additional monitoring devices—for example, skin resistance testing—may improve the sensitivity to pick up ictal piloerection in future studies.

In summary, ictal piloerection is rare, associated with temporal seizure onset, without clear localising value if the piloerection is bilateral. However, if the piloerection starts unilaterally, the seizure onset is most likely lateralised to the ipsilateral hemisphere. The exact location of the generator of piloerection remains unclear. It is most likely situated within the central autonomic network possibly in the insula or amygdala.

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