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SUNCT and SUNA: medical and surgical treatments

Giorgio Lambru · Manjit S. Matharu

Abstract Short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing (SUNCT) and short-lasting unilateral neuralgiform headache attacks with cranial autonomic symptoms (SUNA) are rare and often disabling primary headache disorders. Their management can be challenging. The abortive therapies are not generally useful as the attacks are relatively short lasting. A myriad of pharmacological preventive treatments have been tried in single case reports or small series in an open-label fashion. Lamotrigine, as an oral preventive treatment, and lidocaine, as an intravenous transitional treatment, seem to be the most effective therapies. For medically intractable chronic forms of SUNCT and SUNA, several surgical approaches have been tried. These include ablative procedures involving the trigeminal nerve or the Gasserian ganglion, microvascular decompression of the trigeminal nerve, and neurostimulation techniques. This review provides an overview of the current pharmacological and surgical options for SUNCT and SUNA syndromes.

Keywords SUNCT · SUNA · Trigeminal autonomic cephalalgias · Lamotrigine · Neurostimulation

G. Lambru (✉)
Clinical Research Associate Headache Group, Institute of Neurology and The National Hospital for Neurology and Neurosurgery, Queen Square, London, WC1N 3BG, UK
e-mail: giorgio.lambru.10@ucl.ac.uk

M. S. Matharu
Senior Lecturer and Honorary Consultant Neurologist, Headache Group, Institute of Neurology and The National Hospital for Neurology and Neurosurgery, Queen Square, London, WC1N 3BG, UK

Introduction

Short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing (SUNCT) and short-lasting unilateral neuralgiform headache attacks with cranial autonomic symptoms (SUNA) are trigeminal autonomic cephalalgias (TACs), characterised by repetitive short-lasting attacks of severe, unilateral, neuralgiform pain, usually experienced in the distribution of the ophthalmic and maxillary trigeminal divisions and associated with ipsilateral trigeminal autonomic phenomena. Unlike for SUNCT, a distinction between episodic and chronic forms has been proposed for SUNA syndrome. The criteria applied for the definition of episodic and chronic SUNA are identical to those of the other TACs [1].

SUNCT is believed to be rare with an estimated prevalence of 6.6 per 100,000 and an annual incidence of 1.2 per 100,000 [2]. SUNA seems to be even rarer than SUNCT, probably five times less frequent than the latter [3]. However, a preliminary report of a study presented at the 63rd American Academy of Neurology (Honolulu, USA, 9–16 April 2011), described the clinical phenotype of 61 SUNCT and 38 SUNA patients, suggesting that SUNA occurs more frequently, but is perhaps underdiagnosed [4].

In view of the trigeminal distribution pain in association with ipsilateral cranial autonomic symptoms, SUNCT has been encompassed within the TACs grouping. The activation of the posterior hypothalamus during attacks, demonstrated in functional neuroimaging studies [5, 6], has led to the hypothesis that the posterior hypothalamus plays a crucial role in the pathophysiology of SUNCT syndrome. However, besides the occurrence of ipsilateral autonomic symptoms during attacks, there are few other similarities shared between SUNCT/SUNA and the other TACs syndromes. Conversely, demographic characteristics, clinical features, duration and

frequency of attacks, erratic temporal pattern of attacks, neuroimaging findings and similar efficacious treatments underpin common pathophysiological mechanisms between SUNCT, SUNA and trigeminal neuralgia (TN).

The similarities among SUNCT, SUNA and TN have been the underpinning of many similar medical and surgical therapeutic trials for these syndromes, though there is a lack of randomized placebo-controlled clinical trials assessing therapy in SUNCT/SUNA. The evidence base is largely limited to case reports and small open-label series. This review provides an overview of medical and surgical treatments that have been reported to be useful in the management of SUNCT and SUNA.

Medical management

Abortive treatments

Since the attacks are very short lasting, attack therapy strategies are not useful in SUNCT/SUNA. There is no available abortive treatment for the individual attacks.

Preventive treatments

Lamotrigine

Before the use of lamotrigine, SUNCT was considered to be highly refractory to medical treatments. Lamotrigine acts mainly by blockade of voltage-dependent sodium channel conductance, although antifolate, antiglutamate, and antiaspartate actions have been suggested. At present, it is considered the drug of choice for the preventive treatment of SUNCT and SUNA [7]. Lamotrigine, given in an open-label manner at doses up to 300 mg daily, was reported to be highly efficacious [8]. It had a moderate-to-good effect in 68 % of SUNCT patients and 25 % of SUNA patients in an open-label study. The range of daily dose varied between 25 and 600 mg [6]. In an Australian series, the response to lamotrigine depended on the subtype of SUNCT; lamotrigine was reported to produce an excellent response in episodic SUNCT, but a poor response in those with the chronic form [2].

Topiramate

Topiramate has multiple mechanisms of action. It exerts its action through blockade of the voltage-gated sodium channels, enhancing GABA-mediated chloride influx involving GABA-A receptor and antagonism of the glutamate kainate/AMPA receptor. Topiramate was reported to be effective in five patients at doses up to 300 mg daily [9, 10]. Subsequently, 11 of 21 SUNCT patients (52 %) had a good response to topiramate in an open-label study [6].

Zonisamide

Zonisamide, which has got similar mechanisms of action to topiramate, has been tried in a SUNCT patient who did not tolerate carbamazepine with excellent results on long-term follow-up [11].

Gabapentin

SUNCT has been shown to respond to gabapentin, with complete suppression of attacks in three of nine patients treated with 800–2,700 mg daily [12–14]. When tried in an open-label fashion in 22 SUNCT and 5 SUNA patients at up to 3,600 mg daily, it was reported to be effective in 60 % of SUNA but only 45 % of SUNCT patients [6].

Carbamazepine

Carbamazepine acts by blockade of use- and frequency-dependent sodium channels, although a blockade of the *N*-methyl-D-aspartate receptor-activated sodium and calcium influx and effects on the purine, monoamine, and acetylcholine receptors have also been proposed. The therapeutic response to carbamazepine has been reported in 33 cases. In 22 of 33 patients (67 %), there was no beneficial response with the sole use of carbamazepine. Among the 11 patients with a favourable response, 8 of the 33 patients had a partial response and 3 had a complete or almost complete response [15]. In a recent open-label series of 36 SUNCT and 5 SUNA patients treated with carbamazepine, 40 % of SUNCT and 20 % of SUNA patients reported a favourable response [6].

Oxcarbazepine

There is a case report of SUNCT responding to oxcarbazepine 600 mg [16]. Another SUNCT patient was treated successfully using a combination of oxcarbazepine (600 mg) and gabapentin (400 mg) [17].

Botulinum toxin A

OnabotulinumtoxinA infiltrated at four points around the orbit, injecting 10 U at each site, was reported to be consistently effective in a SUNCT patient refractory to oral treatments after 2.5 years of follow-up [18].

Transitional treatments

There can be a lag of several days to a few weeks before the efficacy of preventive treatments becomes apparent. Transitional treatments, which produce a rapid suppression of the attacks for a limited period of time, can be used when waiting for the beneficial effect of a preventive treatment to become evident.

Intravenous lidocaine

Lidocaine mediates its effect through blockade of sodium channels. The administration of 1.3–3.3 mg/kg/h intravenous (IV) lidocaine was highly effective at completely suppressing the headaches in four patients with SUNCT syndrome [19]. Subsequently, 11 SUNCT and 4 SUNA patients reported a favourable outcome during administration of IV lidocaine at the dose of 1.5–3.5 mg/kg/h [6]. Given the quick and dramatic, but often short-lasting effect in most of SUNCT and SUNA patients, it is advisable to use IV lidocaine as a short-term treatment in patients who present in the so-called "SUNCT status" [20] and also to avoid breakthrough attacks while switching from a preventive drug to another in patients with high load of attacks. Our Headache Centre protocol consists of using IV lidocaine ranging from 1.3 to 3.3 mg/kg/h for 7–10 days, with an infusion speed that varies from 15 to 60 ml/h. Twenty-four hours ECG monitoring is mandatory during the infusion.

Greater occipital nerve blocks

A suboccipital injection of a combination of lidocaine and a steroid was beneficial in five out of eight SUNCT patients [6]. Greater occipital nerve injections (GONIs) may render the patient pain free for weeks or months, allowing the introduction and dose escalation of preventive medications.

Corticosteroids

The response to corticosteroids has been reported in 21 patients. Eleven patients were administered prednisone and eight patients were administered prednisolone or methylprednisolone. Corticosteroids were given to two patients, but the specific agent used was not stated. Six of the 11 patients taking prednisone reported a beneficial response. Six of the eight patients taking prednisolone or methylprednisolone reported a favourable effect [15, 21, 22]. In particular, oral administration of methylprednisolone for 3–6 weeks at the daily doses of $\leq$1 mg/kg was efficacious in suppressing bouts of SUNCT attacks in three patients with the episodic form of the disorder [23].

Queen Square experience

The preliminary results of an open-label study in the medical treatment of SUNCT and SUNA syndromes have been recently presented at the 15th IHS Congress (Berlin, 23–26 June 2011). The authors assessed the outcome of several preventive and transitional treatments prospectively administered in an open-label fashion in 62 SUNCT and 50 SUNA patients. No significant differences in treatment responses were noticed between SUNCT and SUNA, therefore, SUNCT and SUNA patients were considered together, when results were reported. Lamotrigine was effective in 67 % of patients treated (n = 59/88 patients), topiramate in 44.4 % (n = 24/54 patients), gabapentin in 27.8 % (n = 17/61 patients) and carbamazepine in 45.6 % (n = 26/57 patients), the latter providing a partial response in most patients who reported a benefit. Oxcarbazepine was beneficial in 59 % of patients (n = 20/34 patients), while the percentage of patients successfully treated with pregabalin was the same as those treated with gabapentin (28 %, n = 12/43 patients); mexiletine was beneficial in 55.6 % of patients (n = 5/9 patients), though the tolerability profile of this drug was poor. Remarkably, duloxetine was beneficial in 39 % of patients (n = 5/13 patients). The majority of patients treated with lamotrigine, oxcarbazepine, topiramate and duloxetine reported a good to excellent benefit. Among transitional treatments, IV lidocaine rendered 87 % of patients (n = 29/31 patients) pain free, though the pain tended to recur rapidly when treatment was stopped for most of them. A GONI using a mixture of 80 mg methylprednisolone and 2 ml of 2 % lidocaine was effective in 35.7 % (n = 20/56 patients), for a median of 14 days (range 1–150 days) [24].

Table 1 Proposed algorithm for medical treatment of SUNCT and SUNA syndromes

	Medication and dose (max dose reported to be effective)
1st line treatment	Lamotrigine (up to 600 mg/day)
2nd line treatments	Oxcarbazepine (up to 2,400 mg/day)
	Topiramate (up to 700 mg/day)
	Duloxetine (30–120 mg/day)
3rd line treatments	Carbamazepine (up to 1,600 mg/day)
	Gabapentin (up to 3,600 mg/day)
	Pregabalin (up to 600 mg/day)
	Mexiletine (up to 1,200 mg/day)
	Lidocaine patches (5 %)
Transitional treatments	IV lidocaine (1.3–3.3 mg/kg/h for 7–10 days)
	GONI (methylprednisolone 80 mg + 2 ml of 2 % lidocaine)
	Oral/IV corticosteroids

Table 1 shows a proposed algorithm of medical treatment of SUNCT and SUNA, based upon published and unpublished [24] open-label literature evidences.

Surgical management

Several surgical approaches have been tried in SUNCT syndrome. The approaches attempted can be subdivided into three main groups: ablative procedures of the trigeminal nerve, microvascular decompression (MVD) of the trigeminal nerve and neurostimulation techniques. Table 2 summarises the *status quo* of the surgical management in SUNCT and SUNA syndromes.

Ablative procedures of the trigeminal nerve

Procedures that have been tried in SUNCT syndrome include: percutaneous trigeminal ganglion compression [25], trigeminal ganglion thermocoagulation [26], retrogasserian glycerol rhizolysis [27] and gamma knife surgery [28]. Isolated case reports have suggested a potential effectiveness of these treatments. On the other hand, three patients who had poor outcome from these procedures have also been reported [19, 26]. Adverse events of these procedures include: hypoesthesia, keratitis and anaesthesia dolorosa, which can be a highly disabling complication, though very rare [29].

Microvascular decompression of the trigeminal nerve

Williams and Broadley [2], who systematically looked for trigeminal neurovascular conflict with dedicated trigeminal MRI scans, found a high proportion of ipsilateral vascular loops in contact with the trigeminal nerve in SUNCT and SUNA (88 %, $n = 15/17$). This supported the notion of MVD being a potential treatment for these conditions. To date, 10 case reports and a case series of nine patients with medically intractable SUNCT and SUNA patients, who underwent MVD of the trigeminal nerve, have been reported [26, 30–35]. After a median follow-up of 14 months (range 0.5–32 months), 12 of 19 (63 %) of cases were pain free, whereas in the remaining patients, the procedure had little or no effect. Two patients suffered from persistent complications, such as ataxia and hearing loss, whereas in five cases, transient complications were noted.

Peripheral and central neurostimulation

Occipital nerve stimulation

Lambru et al. [36] recently presented the outcome of nine medically intractable SUNCT ($n = 6$) and SUNA ($n = 3$) patients treated with occipital nerve stimulation (ONS). Data on frequency, intensity and duration of attacks were obtained from headache diaries at baseline and after implantation, along with data on disability, anxiety and depression scales administered pre- and post-implantation. At a median follow-up of 31 months (range 16–48 months), all but one patient showed substantial improvements: four patients became pain free, two almost pain free (≥95 % improvement), and two had a remarkable reduction in attack frequency and severity (≥80 % improvement). Five patients were able to discontinue preventive medications, while two were able to reduce the medications. Battery failure or voluntary stimulator switch-off were followed by recurrence or worsening of the attacks within few days in most of patients. Adverse events included new-onset hemicrania continua, lead migration, exposition of the electrode and severe muscle pain over the leads.

Deep brain stimulation of the posterior hypothalamic region

In view of the functional imaging evidence of activation of the posterior hypothalamus region being linked to attacks of SUNCT [5] and the broad experience in the use of posterior hypothalamic region deep brain stimulation (DBS) in patient with medically intractable cluster headache (CH), three patients with intractable SUNCT have been treated with DBS in the region of the posterior hypothalamus. The outcome of the three patients was promising, with a significant and sustained decrease in attack frequency, respectively, at 18-month [37], 12-month [38] and 15-month follow-up [39].

Discussion

Before the reports of the beneficial effects of lamotrigine, SUNCT was considered to be highly refractory to medical treatments. At present, lamotrigine is considered the drug of choice for SUNCT. Given the overlapping clinical features with TN, a myriad of other therapeutic trials have been tried in open-label fashion often in isolated case reports or small series of patients. Preliminary open-label data from a large series [24] provide some pointers for medical management options that may be useful in both SUNCT and SUNA.

Our group has reported that oxcarbazepine and duloxetine can be useful for the preventive treatment of SUNCT and SUNA [24]. The former could be an attractive option, given the better tolerability profile compared to carbamazepine. To our knowledge, duloxetine has never been reported for the treatment of SUNCT/SUNA. Forty percent

Table 2 Surgical treatments for SUNCT and SUNA syndromes

	Age/sex	Type	Headache duration (years)	Follow-up (months)	Outcome	Complications
Microvascular decompression of the trigeminal nerve (19 patients)	Median 54 years (28–73) 14/M 5/F	14 SUNCT 3 SUNA 2 SUNCT/SUNA 10 Chronic 2 episodic 7 md	Median 4 (1 month–26 years)	Median 14 (0.5–32)	12/19 patients: pain free	5 patients: infection, vertigo, jaw pain) 2 patients: hearing loss, ataxia
Occipital nerve stimulation (9 patients)	Median 52 years (33–74) 4/M 5/F	Chronic SUNCT Chronic SUNCT Chronic SUNCT Chronic SUNCT Chronic SUNCT Chronic SUNCT Chronic SUNA Chronic SUNA Chronic SUNA	Median 7(2–22)	Median 38 (24–55)	4 patients: pain free 4 patients: 81–99 % improvement. 1 patient: non-responder	Hemicrania continua (1 patient) Electrode migration and exposition of the electrode (1 patient) Severe muscle pain (1 patient)
Deep brain stimulation (3 patients)	73/M	SUNCT	6	15	>50 % benefit	None
	44/M	SUNCT	36	12	>50 % benefit	Erectile dysfunction
	66/F	Chronic SUNCT	14	15	>50 % benefit	Diplopia
Glycerol rhizotomy (4 patients)	80/M	Episodic SUNCT	25	87	>50 % benefit	Facial sensory loss
	72/F	Chronic SUNCT	10	90	>50 % benefit	Facial sensory loss
	52/M	Chronic SUNCT	8	7	Pain free	Md
	38/M	Chronic SUNCT	2	5	Ineffective	Hypoesthesia
Gamma knife radiosurgery (3 patients)	82/M	Episodic SUNCT	6	39	Pain free	None
	39/M	Chronic SUNCT	2	5	Mild benefit	Anaesthesia dolorosa
	28/M	SUNCT	10	2	Ineffective	Md
Gasserian ganglion balloon compression (2 patients)	66/F	SUNCT	0.1	120	Pain free	None
	68/F	Chronic SUNCT	17	18	>50 % benefit	None

of our patients reported a favourable response. If these outcome data will be confirmed in larger series of patients, duloxetine may become a new treatment option for the prophylaxis of these disorders. Duloxetine is a selective serotonin (5-HT) and norepinephrine (NE) reuptake inhibitor [40]. Duloxetine has been reported to be effective in diabetic neuropathic pain and recently in chronic orofacial pain disorders, such as burning mouth syndrome [41, 42], suggesting other mechanisms of action. Indeed, duloxetine has been shown to act on certain sodium channels, blocking persistent late Nav1.7 currents. This effect could explain its efficacy in neuropathic pain syndromes [43] and also in SUNCT/SUNA.

Several surgical treatments have been tried in SUNCT and SUNA. Surgical management of primary headaches should be offered to medically intractable cases. However,

to date, the definition of medically intractable SUNCT/SUNA is obscure. It is, therefore, difficult to compare the outcome of the various surgical treatments.

Occipital nerve stimulation seems at present the surgical option of choice for medically intractable chronic SUNCT and SUNA syndrome. The lack of the more significant side effects associated with invasive brain procedures renders this technique a very attractive option. Although a longer follow-up period would be ideal to assess the long-term efficacy of MVD for chronic medically intractable SUNCT/SUNA, it may also be considered a valuable option in patients with ipsilateral trigeminal nerve compression due to a vascular loop, though possible benefit should be weighed against operation-related risks of permanent neurological deficits. The outcome of posterior hypothalamus region DBS in chronic SUNCT is encouraging for the first three patients treated so far. Nevertheless, more data are required before hypothalamic region DBS can be routinely recommended, especially given the small risk of fatal complications [44].

Conclusion

The management of SUNCT and SUNA syndromes remains problematic. However, new studies, including large series of patients, will hopefully be able to broaden the armamentarium of effective medical and surgical treatments. Sodium channel blockers seem to be the most effective category of drugs for SUNCT and SUNA. This raises the possibility that these syndromes may be due to an underlying sodium channelopathy. The molecular characterisation of these patients to confirm this clinical finding [45], may potentially direct the research towards the production of new specific compounds.

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