SYSTEMATIC REVIEW OF AETIOLOGY AND TREATMENT OF POST-STROKE HAND OEDEMA AND SHOULDER–HAND SYNDROME

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Studies on the aetiology and treatment of post-stroke hand oedema and shoulder–hand syndrome (SHS) published from January 1973 until August 1998 were identified. Eleven studies were included with at least some control for confounding. These were evaluated on 11 methodological criteria and by standardized effect sizes. There were five aetiological studies: four cohort studies and one study consisting of two case series using a within-subjects design. The matters investigated included lymph scintigraphy in hand oedema, bone scintigraphy, putative risk factors and the existence of autonomic dysregulation and peripheral nerve lesions in SHS. There were six therapeutic studies: one randomized controlled trial, one non-randomized controlled trial, one cohort study and three case series, of which two studies used a within-subjects design. These studies investigated continuous passive motion and neuromuscular stimulation in hand oedema as well as oral corticosteroids, intramuscular calcitonin and trauma prevention in SHS. A great diversity of pathophysiological and therapeutic insight was found. Based on systematic analysis of the literature, the following conclusions seem justified: (i) the shoulder is involved in only half of the cases with painful swelling of wrist and hand, suggesting a "wrist–hand syndrome" between simple hand oedema and SHS; (ii) hand oedema is not lymphoedema; (iii) SHS usually coincides with increased arterial blood flow; (iv) trauma causes aseptic joint inflammations in SHS; (v) no specific treatment has yet proven its advantage over other physical methods for reducing hand oedema; and (vi) oral corticosteroids are the most effective treatment for SHS.

Key words: stroke, cerebrovascular accident, hemiplegia, hand oedema, shoulder–hand syndrome, reflex sympathetic dystrophy syndrome, aetiology, treatment.

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INTRODUCTION

Oedema of the paretic hand is a problem frequently encountered by (para)medical professionals involved in the rehabilitation of stroke patients. Hand oedema may occur as an isolated problem or as part of shoulder–hand syndrome (SHS) (1). The diagnosis of definite, probable and possible SHS in stroke patients can be made according to the guidelines described in Table I. Symptoms of the hand and wrist are crucial for discriminating SHS from a painful hemiparetic shoulder. Shoulder pain usually develops in the first or second month, whereas SHS may develop between the second and fourth month after the onset of hemiparesis (2, 3). The reported incidences of isolated hand oedema after stroke vary greatly. Exton-Smith & Crockett (4) found an incidence of 16%, whereas Tepperman et al. (1) reported an incidence of 82.8%. Reported incidences of SHS also vary from 12.5% (3) to 61% (5). This substantial variation can probably be explained by differences in definition, base population and rehabilitation measures.

The aetiology of post-stroke hand oedema and SHS remains unclear. Several authors assume that prolonged dependency and immobility of the paretic arm cause hand oedema (6, 7). Especially in SHS, (repeated minor) trauma is believed to play a causal role (2, 8, 9). In this perspective, higher-order perceptual deficits such as hemi-inattention may be indirectly associated with the development of hand oedema and shoulder pain (10). Because SHS is often regarded as a clinical variant of the reflex sympathetic dystrophy (RSD) syndrome (11, 12), central sympathetic dysregulation and neurogenic inflammatory reactions may contribute to the development of SHS following stroke (13, 14).

Various treatment regimes for post-stroke hand oedema and SHS have been advocated, including many different physical measures such as elevation, splinting, compression therapy, cryotherapy, massage and active or passive motion exercises (15, 16). Relatively new concepts include continuous passive motion (CPM) (6, 7) and neuromuscular stimulation (NMS) (17). As for SHS, oral corticosteroids (2, 3), intramuscular calcitonin (18) and steroid injections in the shoulder joint (9) have been recommended. There is, however, no consensus on the preferred treatment strategy. Yet, when hand oedema and SHS are not treated in an early phase, contractures develop in the hand, wrist and shoulder leading to permanent loss of upper extremity function, even when motor recovery occurs (19). In view of the clinical relevance and the great diversity of pathophysiological and therapeutic insights, a systematic review of the aetiology and treatment of post-stroke hand oedema and SHS was conducted.

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Table I. *Criteria for post-stroke shoulder–hand syndrome (SHS)*

Site	Symptoms and signs[a]
Shoulder	Loss of ROM, especially abduction and external rotation; pain and tenderness elicited by these motions or in rest (severe cases)
Elbow	Usually free of signs and symptoms
Wrist	Considerable pain on (limited) extension; tenderness to deep palpation and dorsal oedema over carpal bones
Hand	Relatively little spontaneous pain or tenderness; oedema overlying metacarpals
Digits	Considerable pain on (limited) flexion of metacarpalphalangeal and interphalangeal joints; moderate fusiform oedema and loss of dorsal skin lines; changes in hair and nail growth; vasomotor and sudomotor lability (changes in temperature, colour and hidrosis)

Definite SHS[b]: all shoulder–wrist–hand criteria
Probable SHS[b]: all wrist–hand criteria
Possible SHS[b]: wrist–hand tenderness and swelling
Absent SHS[b]: only wrist–hand swelling

[a] Criteria adapted from Davis et al. (3).
[b] Criteria by Tepperman et al. (1).

METHODS

Selection of studies

Material for the review (published until August 1998) was collected by a systematic search in Medline (from January 1973), Current Contents (from January 1996) and Online Contents (from September 1992) using the following keywords: stroke, cerebrovascular accident/disorder, hemiplegia/hemiparesis, hand oedema, shoulder–hand syndrome, reflex sympathetic dystrophy syndrome and causalgia. In addition, the data bank of the Dutch Paramedical Institute was consulted. Checking the references of the selected articles extended the search. Only patient-related studies aimed at the aetiology and/or treatment of hand oedema and/or SHS and published in the English, German, Dutch or French languages were included.

Methodological evaluation

Both the internal validity (V) and the methods of data extraction (D) were assessed. We established adapted V and D criteria, based on a system that was originally developed for evaluating randomized controlled trials (RCT) (20), that could be used for evaluating both aetiological and therapeutic studies. Each criterion was scored according to three levels: sufficient [+], moderate [(+)], or insufficient [−]. When a specific criterion was not applicable, it was scored as such [0]. All selected studies were independently analysed by 3 referees (A.G., B.V. and J.v.L.). In the case of discrepancies, consensus was pursued in the second instance.

Internal validity. V1: The homogeneity of the study sample by means of definition was tested. An indication of the severity of the clinical problem was always required. A comment on the absence of shoulder involvement was required for studies on hand oedema. As for SHS, the diagnostic criteria listed in Table I were taken as a reference.

V2: The homogeneity of the study sample on putative causal factors was tested. Based on the literature, three relevant aetiological factors were identified: (i) stroke (either cerebral infarction or haemorrhage), (ii) degree of upper extremity sensory loss and paresis and (iii) the presence of higher-order perceptual deficits.

V3: The selected study design was judged in relation to the study aim. For therapy evaluation, a (randomized) controlled trial or case-reference design with random allocation of interventions was considered appropriate, as was any other design with the ability to control for confounding, e.g. a case series using a within-subjects design. For aetiological studies, a cohort or a case-reference design was considered appropriate.

V4: This criterion tested whether there had indeed been sufficient control for potential confounders before exposure to aetiological or therapeutic events. In this view, the duration of and time relation between the clinical problems (stroke and its primary consequences, hand oedema, SHS) were considered relevant, because pathophysiological mechanisms may change over time and, thus, so may the susceptibility to aetiological or therapeutic factors. Also, comorbidity with a possible effect on hand oedema or SHS (e.g. heart or renal failure, malignant or rheumatic disease) and related use of medication (e.g. diuretics, corticosteroids) were considered relevant confounders.

V5: The relevance of the selected effect parameters was assessed.

Data extraction. D1: This criterion tested whether the inclusion and exclusion criteria were given (e.g. first stroke, absence of prior SHS, absence of other neuropsychiatric disease), and whether the base population was identified from which the study sample was selected.

D2: It was judged whether effects were adequately reported both in terms of statistical (e.g. *F*- and *p*-values) and quantitative measures (absolute or relative differences).

D3: This criterion judged the length of the follow-up period in relation to the clinical problem.

D4: As for loss to follow-up, 5% or less was regarded as fair, whereas 20% or more was considered unacceptable.

D5: Intention-to-treat analysis was assessed in the case of therapy evaluation when there was loss to follow-up.

D6: The adequacy of the sample size was calculated by means of power analysis based on the reported (non)significant (group) differences.

Evaluation of clinical significance

Standardized effect sizes (Z-scores) were calculated for the most relevant parameters to compare (group) differences as a function of the pooled standard deviation independent of unity of measurement (21):

$$\text{Z-score} = (x_a - x_b)/\text{PSD}$$

where x_a and x_b = means of samples a and b, respectively, and PSD = pooled standard deviation.

By convention, the cut-off criterion for considering a particular effect clinically relevant was a (group) difference of at least one standard score ($Z \geqslant 1.0$).

RESULTS

The search resulted in 13 articles, of which 4 articles were excluded after a preliminary assessment because they were based on designs lacking any control for confounding (6, 9, 13, 16). Of the 9 definitively selected articles, only the work by Braus et al. (2) was aimed at both aetiological (risk factors) and therapeutic aspects (medication and prevention). Each of these three aspects was regarded as an individual study. Consequently, 11 studies were further analysed, 5 studies with a focus on aetiology and 6 therapy evaluation studies. The results of the assessment of both V and D criteria are given in Tables II and III, respectively. Z-scores of the most relevant effect parameters are presented in Table IV. The main clinical

Table II. *Assessment of internal validity (V criteria)*

First author	V1	V2	V3	V4	V5	Sample
Aetiology						
Werner (22)	(+)	−	+	(+)	+	Cohort
Greyson (8)	+	−	+	+	+	Cohort
Tepperman (1)	+	−	+	+	+	Cohort
Braus (2)	+	+	+	−	+	Cohort
Hesse (14)	+	(+)	+	+	+	Two case series
Treatment						
Faghri (17)	(+)	(+)	+	(+)	+	Case series
Giudice (7)	(+)	(+)	+	+	+	Case series
Braus (2) (medic.)	+	(+)	+	−	+	RCT + crossover CT
Braus (2) (prev.)	+	−	+	−	+	Cohort
Davis (3)	(+)	−	−	−	(+)	Case series
Hamamci (18)	+	(+)	+	+	+	Controlled trial

Note: See Methods for explanation of criteria and symbols.

outcomes of the aetiological and therapeutic studies are summarized in Tables V and VI, respectively. For reasons of brevity, only the weaker methodological aspects of the selected studies will be elaborated.

Etiology

Werner et al. (22) performed lymph scintigraphy of the paretic arms with hand oedema and compared the results to the non-paretic arms of the same patients as well as to the paretic arms of patients without hand oedema. It was a cohort study with consecutively hospitalized hemiplegic patients approached as a cross-sectional sample. The presence or absence of shoulder pain was not clearly indicated. The authors also included non-stroke patients and did not control for the degree of sensorimotor or higher-order perceptual deficits. Control for potential confounders was moderate; although other possible causes of oedema were excluded, the duration of hand oedema was not indicated. As for data extraction, no exact quantitative comparisons were made.

Greyson & Tepperman (8) and Tepperman et al. (1) reported two studies concerning one cohort of hospitalized stroke patients approached as a cross-sectional sample. They performed three-phase bone scintigraphy in both SHS-positive and -negative stroke patients and compared the paretic and non-paretic arms. The degree of primary sensorimotor or higher-order perceptual deficits was not taken into account, although Greyson & Tepperman (8) discussed the influence of subluxation on increased bone uptake in the affected shoulder. They merely provided descriptive data, although some formal quantitative analysis would have been possible. Tepperman et al. (1) reported sensitivity, specificity and positive predictive values of several clinical symptoms and signs without providing confidence intervals.

Part of the work by Braus et al. (2) was a cohort study to determine risk factors for SHS following stroke. Insufficient information was given on the possible influence of systemic comorbidity or medication. The quantitative and statistical report was marginal: a specially designed SHS score (pain, oedema, ROM) was dichotomously (0–7 vs. 8–14) related to possible risk factors presented in two sets of histograms with merely one *p*-value. Time relations between exposure and clinical symptoms were not considered. Hence, associated factors but no true risk factors could be determined.

Table III. *Assessment of data extraction (D criteria)*

First author	D1	D2	D3	D4	D5	D6	Sample (*n*)
Aetiology							
Werner (22)	+	(+)	0	0	0	+	80
Greyson (8)	+	−	0	0	0	+	85
Tepperman (1)	+	(+)	0	0	0	+	85
Braus (2)	+	(+)	0	0	0	+	132
Hesse (14)	−	+	0	0	0	+	78
Treatment							
Faghri (17)	−	−	+	+	0	+	8
Giudice (7)	(+)	(+)	+	+	0	+	11
Braus (2) (medic.)	+	−	+	+	0	+	34
Braus (2) (prev.)	+	(+)	−	+	0	+	218
Davis (3)	+	−	+	+	0	+	68
Hamamci (18)	+	+	+	+	0	+	41

Note: See Methods for explanation of criteria and symbols.

Table IV. *Standardized effect scores of main parameters*

First author	Parameter	Z-score
Werner (22)	Paretic vs. non-paretic arms:	
	Abnormal lymphatic flow	10.8[a]
Tepperman (1)	Scan-positive vs. negative patients:	
	Swelling in wrist and hand	2.6[a]
	MCP tenderness	High[b]
Braus (2)	SHS score 0–7 vs. 8–14 patients:	
	Paresis MRC grade 0–2	7.3[a]
	Shoulder subluxation	10.0[a]
	Visual–perceptual deficits	10.3[a]
Hesse (14)	Paretic vs. non-paretic arms (SHS group):	
	Skin temperature	1.8
	SSR amplitude/area	2.4/2.7
	F/M ratio (median nerve)	3.2
Faghri (17)	NMS vs. arm elevation:	
	Hand volume	3.7
Giudice (7)	CPM + arm elevation vs. arm elevation:	
	Hand volume	0.73
	Finger stiffness (passive ROM)	0.85
Braus (2) (medic.)	Before vs. 6 months after methylprednisolone:	
	SHS score	8.3
Braus (2) (prev.)	Trauma prevention vs. no prevention:	
	Frequency SHS score 8–14	7.5[a]
Hamamci (18)	Calcitonin vs. placebo:	
	Pain	4.9[a]
	Shoulder exorotation	1.9
	MCP extension	1.3

[a] Dichotomous variables give relatively high Z-scores.
[b] No Z-score could be calculated because there were no scan-negative patients with MCP tenderness.
Note: Studies by Greyson & Tepperman (8) and Davis et al. (3) gave insufficient data to calculate Z-scores.
MCP: metacarpalphalangeal; SHS: shoulder–hand syndrome; NMS: neuromuscular stimulation; CPM: continuous passive motion.

Table V. *Main clinical findings of aetiological studies*

First author	Outcome
Werner (22)	Lymphatic flow is increased in 83% of paretic compared to 15% of non-paretic arms in the same patients. No evidence of lymphoedema.
Greyson (8)	Of the 25% stroke patients with scintigraphic evidence of RSD (i.e. increased delayed bone uptake in hand and wrist), 62% showed increased blood flow, whereas 38% showed decreased blood flow. A shoulder–hand distribution was seen in 52%, whereas 48% showed involvement of the wrist and hand alone ("wrist–hand syndrome").
Tepperman (1)	See Greyson. Tenderness of the MCP joints to compression is the most valuable clinical sign for diagnosing post-stroke SHS: positive predictive value 100%, negative predictive value 96%. Swelling of the wrist and hand has a good negative predictive value (92%), but its positive predictive value is low (27%) indicating the existence of simple hand oedema.
Braus (2)	The occurrence of post-stroke SHS is associated with the severity of paresis and shoulder subluxation of the paretic arm as well as with deficits in confrontation visual field testing.
Hesse (14)	The paretic extremity in stroke patients with SHS is warmer, shows enhanced sympathetic skin responses and prolonged proximal nerve latencies compared to the non-paretic side.

RSD: reflex sympathetic dystrophy; MCP: metacarpalphalangeal; SHS: shoulder–hand syndrome.

Hesse et al. (14) compared skin temperature, sympathetic skin responses and nerve latencies of paretic and non-paretic arms in two case series of stroke patients with and without SHS using a within-subjects analysis. Although the influence of the degree of paresis was controlled for by an equal motricity index in either group, the influence of higher-order perceptual deficits was unclear. Qualitative differences between the two groups were adequately addressed with the exception of the influence of shoulder subluxation. Information on the eligibility of stroke patients was unsatisfactory.

Treatment

Two comparable therapy evaluation studies comprised a pre- and post-assessment of hand oedema in a case series of stroke patients using a within-subjects analysis. Giudice (7) investigated the use of CPM and arm elevation versus elevation alone, whereas Faghri (17) studied the use of NMS versus arm elevation. Each subject underwent both an experimental and a conventional treatment on two consecutive days. Only Giudice (7) analysed the influence of the severity of hand oedema on treatment outcome, whereas only Faghri (17) specifically excluded SHS. Neither study convincingly controlled for the degree of paresis or perceptual deficits. Faghri (17) did not investigate the influence of the duration of hand oedema on the treatment effect. The base population and the selection criteria were moderately indicated by Giudice (7) and insufficiently by Faghri (17). Giudice (7) provided some statistical information, whereas the statistics reported by Faghri (17) were insufficient and at specific points erroneous.

Braus et al. (2) used the stroke patients in their original cohort with a high SHS score (8–14) to examine prospectively the effect of oral corticosteroids versus placebo medication in a RCT. After a 4-week period, non-responders were still treated with the same steroid regime in a crossover clinical trial. Despite the relatively small sample sizes and known putative causal factors, no post-hoc test was done to confirm an equal distribution of these factors over the groups. Insufficient information was given about the duration of SHS before treatment as well as about systemic comorbidity and comedication. The quantitative report was insufficient because only one set of histograms with SHS scores was given for each group at several times and without statistical analysis.

In a second prospective trial, Braus et al. (2) took special measures of trauma prevention in patients with low SHS scores (0–7) to compare the occurrence of SHS in this group with the frequency of SHS in their original cohort. The authors' statement that this second cohort was clinically comparable to the first seems questionable because the susceptibility to developing SHS may have changed due to selection and time effects. In addition, the influence of potential confounders

Table VI. *Main clinical findings of therapeutic studies*

First author	Outcome
Faghri (17)	Neuromuscular stimulation has a better short-term effect than limb elevation on post-stroke hand oedema.
Giudice (7)	Continuous passive motion with limb elevation has a better short-term effect than limb elevation alone on post-stroke hand oedema.
Braus (2) (medic.)	32 mg/day oral methylprednisolone tapering over 4 weeks has a good long-term effect on reducing symptoms and signs of post-stroke SHS within 2 weeks compared to placebo medication.
Braus (2) (prev.)	Trauma prevention may reduce the frequency of post-stroke SHS from 27% to 8%.
Davis (3)	16 mg/day oral triamcinolone diacetate tapering over 4 weeks gives almost complete and long-term relief of pain and improvement of joint mobility in post-stroke SHS within 3 weeks.
Hamamci (18)	100 IU/day intramuscular calcitonin for 4 weeks is more effective than placebo treatment for reducing pain, tenderness, and improving joint mobility in post-stroke SHS within 4 weeks.

SHS: shoulder–hand syndrome.

remained unclear. Although the difference in occurrence of SHS was tested statistically by two proportions, the exact relative risk was not calculated. The follow-up period of this study was not indicated.

Davis et al. (3) reported a case series of stroke patients with SHS to test the effect of oral corticosteroids. Basically, this study had an inappropriate design due to insufficient control of causal and confounding factors. It was still included because it was the first major effect study on SHS and yielded impressive results, i.e. all patients became pain-free within 3 weeks. However, only global changes in pain and joint mobility were used as effect parameters. No statistical analysis was done, although stratification on confounding factors (e.g. degree of paresis) would have been possible.

Hamamci et al. (18) investigated the effect of intramuscular calcitonin on post-stroke SHS in a two-group placebo-controlled clinical trial. Their control group was characterized by relatively numerous right-hemisphere lesions and by a relatively high median motricity index, so that the chance of trauma may have been balanced. Although no randomization took place, both groups were comparable with regard to the duration of stroke and SHS, comorbidity, and pre-treatment levels of pain, oedema, joint mobility, motor function, spasticity and shoulder subluxation.

DISCUSSION

For several aetiological studies, it remains uncertain to what extent the degree of primary sensorimotor or higher-order perceptual deficits may have influenced measurements of lymphatic flow (22), blood flow and bone uptake of ^{99m}Tc (1, 8). Yet, these scintigraphic studies seem to retain sufficient validity, because they were directed at identifying basic pathophysiological mechanisms instead of specific causal factors. Greyson & Tepperman (8) found that only about half of their patients with distal signs of RSD had a scintigraphically involved shoulder joint. Hence, in between "simple hand oedema" and "true SHS", there seems to be a painful "wrist–hand syndrome" without shoulder joint involvement. The term "possible SHS" (1) for this clinical condition seems somewhat ambiguous.

Through lymph scintigraphy it was shown that post-stroke hand swelling is not due to lymphoedema. Bone scintigraphic studies indicated that in almost two-thirds of the stroke patients with evidence of RSD there is increased arterial blood flow and hyperemia in the paretic arm. On the other hand, the same studies showed that more than one-third of the stroke patients with evidence of RSD had a decreased blood flow in the paretic arm (as in 86% of the RSD-negative cases). Possibly, post-stroke hand oedema with or without painful joint inflammations primarily coincides with an elevated filtration pressure and altered capillary permeability due to autonomic vasomotor dysregulation (14, 23). Loss of central sympathetic tone opens the vascular bed and will increase blood flow to the soft tissues as well as to the bone. However, in contrast with bone blood vessels, arterial blood flow is substantially influenced by inactivity so that it can also be decreased instead of increased (8).

Reduced tonic sympathetic outflow was also suggested by Hesse et al. (14), who found a warmer paretic extremity compared to the non-paretic side in stroke patients with SHS. At the same time, they found enhanced sympathetic skin responses in these patients on the paretic side when stimulated on the non-paretic side. Because after brain infarction sympathetic skin responses are basically symmetrically suppressed (24), enhanced sympathetic skin responses may be explained by peripheral supersensitivity to sympathetic neurotransmitters (e.g. noradrenaline) rather than by central sympathetic overactivity (25).

Hesse et al. (14) claimed evidence of somato-afferent nerve injury of the brachial plexus of the paretic arm in patients with post-stroke SHS based on an increased F/M ratio compared to the non-paretic side. Although earlier work has emphasized the absence of brachial plexus injury in stroke (26), Cheng & Hong (27) also found an association between spontaneous distal EMG activity and the occurrence of SHS following stroke, suggesting brachial plexus injury. However, these results may be confounded by rather high proportions of shoulder subluxation in the SHS groups (Hesse et al. (14): 91%; Cheng and Hong (27): 74%), which might cause minor nerve injury by prolonged traction. In general, the results by Hesse et al. (14) should be interpreted with caution, because their base population and selection criteria were poorly defined.

Braus et al. (2) suggested that shoulder subluxation by itself might be a risk factor for developing SHS following stroke, in addition to upper extremity weakness, moderate spasticity and visual–perceptual deficits. Although the influence of potential confounders was unclear, these factors seem to have in common a higher risk of traumatizing the hemiparetic arm. Repeated (minor) trauma may lead to microbleeding and aseptic inflammation of peripheral, especially synovial tissue causing traumatic arthritis. The positive effects of systemic corticosteroids seem to support this notion.

Of the six selected therapy evaluation studies, two studies were specifically aimed at the physical treatment of post-stroke hand oedema. Giudice (7) showed that 30 min of CPM and limb elevation resulted in a greater reduction of hand oedema and finger stiffness than 30 min of limb elevation alone. However, these effects were hardly clinically relevant (Z-scores <1.0). Faghri (17) claimed that 30 min of NMS was more effective in reducing hand oedema than 30 min of limb elevation, but the statistical elaboration of this study was such that its credibility is poor. Hence, no conclusions can yet be drawn about the advantage of any specific treatment over other physical methods for reducing hand oedema.

Of the four studies concerned with the treatment of SHS, three investigated the effects of systemic drugs. Braus et al. (2) compared the use of placebo medication to 32 mg methylprednisolone daily divided into 4 oral doses and given at meals along with antacid preparations for 14 days before tapering over a

further 14 days. In the second instance, non-responders in the placebo group were still treated with the same methylprednisolone regimen. All patients received physical therapy (cooling). Ultimately, 31 out of 34 patients became symptom-free within 6–14 days for up to 6 months. In one patient the treatment protocol had to be repeated. Only minor side effects were seen, such as transient increase in blood glucose levels ($n = 15$), sleeping problems ($n = 7$), reversible steroid acne ($n = 5$) and slight increases in blood pressure ($n = 2$). Similar positive effects of oral corticosteroids were earlier reported by Davis et al. (3) using 16 mg triamcinolone diacetate daily in 4 divided doses for 14–21 days before tapering over an 8-day period. At the same time, a sling was given to support the hemiparetic arm. In six patients the treatment had to be repeated before a complete and long-lasting remission was reached. No complications were seen. Although both studies lacked sufficient control for causal and confounding factors as well as sufficient statistical elaboration, the results were impressive.

Hamamci et al. (18) were able to show that salmon calcitonin in a daily intramuscular administration of 100 IU for 4 weeks gave more pain reduction and a better improvement of joint mobility (except wrist flexion and metacarpalphalangeal flexion) than placebo treatment. At the same time, conventional physical therapy was administered to all patients. There was no differential effect on hand oedema or motricity index. The authors did not comment on the pharmacology underlying the effect of calcitonin in SHS, nor were any side-effects discussed. From a clinical viewpoint, calcitonin appears less effective than corticosteroids, because patients did not become completely pain-free.

The "treatment" investigated in the third study by Braus et al. (2) actually consisted of a series of specific measures aimed at trauma prevention in stroke patients with established SHS, such as good lying and sitting positions, mobilization of the scapula before passive movements of the arm and avoidance of painful activities. A reduction in the SHS occurrence rate from 27% to 8% was achieved. Although this study too had poor control of causal and confounding factors as well as an undefined follow-up period, its main relevance is in supporting the idea that trauma may be an essential causal factor in SHS.

CONCLUSION

The literature on post-stroke hand oedema and SHS shows a great diversity of pathophysiological and therapeutic insights, suggesting a multifactorial problem. Only a few concepts are sufficiently supported by clinical epidemiological evidence. In addition to peripheral factors, such as trauma causing aseptic arthritis, central factors seem to play a pivotal role, in particular an initially decreased rather than increased sympathomotor tone. Thus, where the initiating event in traumatic RSD may be primarily peripheral with secondary centralization (28), the eliciting factor in post-stroke SHS may be primarily central. This notion might explain why SHS is more frequent in central than in peripheral paresis and possibly also why there is relatively little spontaneous distal pain in post-stroke SHS.

Based on this systematic review of post-stroke hand oedema and SHS, the following conclusions can be drawn: (i) the shoulder is involved in only half of the cases with painful swelling of wrist and hand, suggesting the existence of a "wrist–hand syndrome" between simple hand oedema and SHS; (ii) hand oedema is not lymphoedema; (iii) SHS usually coincides with increased arterial blood flow; (iv) trauma causes aseptic joint inflammations in SHS; (v) no specific treatment has yet proven its advantage over other physical methods for reducing hand oedema; and (vi) oral corticosteroids are the most effective treatment for SHS. It should be considered, however, that these conclusions are based on limited scientific evidence. In addition, because the number of studies included in this review is small, there is a risk of considerable publication bias.

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