

# On the Diagnosis and Management of Neurocutaneous Syndrome (NCS), A Toxicity Disorder from Dental Sealants

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## Abstract

*Neurocutaneous syndrome (NCS), a newly discovered toxicity disorder, is characterized by neurological sensations, pain, depleted energy and memory loss as well as itchy cutaneous lesions which may invite various opportunistic infections. Components in the calcium hydroxide dental sealants Dycal, Life and Sealapex have been identified as sources of the observed symptoms. Sulfonamide and neurological toxicity issues are discussed and three case histories are presented. Additional notes on zinc oxide, Fynal, IRM and Sultan U/P sealers are also included. Diagnostic and management protocols at the Parasitology Center, Inc. (PCI) are proposed.*

## Introduction

The original description of the neurocutaneous syndrome (NCS)<sup>1</sup> was “introductory in nature.”<sup>1</sup> Examination of many NCS patients and a careful study of their symptoms, exposures, clinical conditions and histories made it possible to identify the underlying cause of the syndrome and proceed with its management.

## Materials and Methods

Patients were personally evaluated and their clinical history, records, symptomology and exposures carefully examined. Specimens provided or collected at the Parasitology Center, Inc. (PCI) were studied. An NCS status was only determined based on symptoms and determination that one or more of the suspect sealers have been used on prior dates. Sensitivity to sulfa and elevated levels of sulfa in the blood were used as a confirmation of sulfonamide toxicity. Continuing patients follow our recommendations for dental rehabilitation, extraction of suspect liner(s), and replacement with ethyltoluene sulfonamide (ETS) and zinc oxide free sealants. A list of vitamin/mineral supplements for patient use during the transitional period and another

list of substitute sealants are provided. Patients are followed up to monitor and insure the resolution of symptoms.

## Results and Discussion

### The Neurocutaneous Syndrome

The disorder is double faceted with dermatological and neurological symptoms compatible with classical sulfa toxicity. The latter is characterized by changes in blood values, photosensitive reactions, allergic vasculitis sores, bacterial flora changes, and redness of the skin, which may lead to liver and kidney failure.<sup>2</sup> The neurological aspects are characterized by pin-prick and/or creeping, painful and irritating movement sensations, often interpreted as parasite movements in various body tissues and/or cavities. Movement sensations are either unipolar or bipolar and may proceed horizontally or vertically. They may manifest as variably shaped bruises or waves of elevated ripples or channels. In no case was the movement sensation related to parasites.<sup>1</sup> Neurological symptoms may also include loss of memory, brain fog, lack of concentration and control of voluntary movements.

The cutaneous aspects include small itchy sores (*see Fig. 1*), inflamed often elevated pimples (*see Figs. 2, 3*), and fully inflamed and painful open/amorphous mucoid lesions that often enlarge and coalesce (*see Fig. 4*). Histopathological sections of lesions (*see Fig. 5*) show superficial and deep perivascular infiltrate of lymphocytes, accompanied by interstitial deposits of granular mucin material. Eosinophils are usually present within the inflammatory infiltrate and foci of epidermolytic hyperkeratosis are often identified within the epidermis (*see Fig. 5*). Lesions may also be on the scalp where they may be associated with infestation of springtails (*Collembola*).<sup>1</sup> In many cases, lesions are associated with edematous reaction usually in the arms and legs (*see Fig.*

1. Amin OM. Neuro-cutaneous Syndrome (NCS); a new disorder. *Explore* 2001; 10: 55-56. <sup>2</sup>Ockert K. Filling caused serious reactions. *Trandlakartidningen* 1994; 86: 470. (in Swedish). <sup>3</sup>Garcia LS. *Diagnostic Medical Parasitology*. Wash, DC: Am Soc Microbiol Press, 2001.

6). Blood vessels may also become enlarged and elevated, and head may become hot and turn red. The gum tissue and the teeth and oral mucoid secretions may turn gray and become compromised first and stay compromised the longest. The above creeping sensation is clearly distinguished from these caused by nematodes such as *Toxocara canis*<sup>3</sup> or *Diocetophyme sp.*<sup>4</sup>

General symptoms usually include fatigue, compromised immune system, psychological trauma and loss of self-esteem. The depressed immune status in most patients appear to pre-empt them for opportunistic infections.

### Compounding Factors

While NCS itself is not a contagious condition, superimposed opportunistic infections on open sores may be. Initial infection with fungus or bacteria appear to attract subsequent infestations with many arthropod species, especially springtails (*Collembola: Insecta*).<sup>1,5,6,7</sup> Black specks associated with such infections appear to be metabolic waste (*fecal elements*) of these organisms or mycelial masses of certain fungal species. *Staphylococcus aureus*, *S. haemolyticus*, *Streptomyces* spp., *Candida albicans* and *Madurella* spp. among others, have been identified from cultured swabs taken from sores of various NCS patients. These opportunistic infections have been shown to aggravate the cutaneous symptoms of NCS patients. The *Madurella* infections are usually associated with black grains of mycelial masses that may be related to the black specks and fibers observed by some NCS patients. The healing of certain patient's lesions<sup>9</sup> was observed to be proportional to the exit of remaining fibers from lesions.<sup>3</sup> Patients experiencing complete remission remain susceptible to fungal promoting conditions in damp, shaded, moldy places.

Arthropods identified from sores include fleas, caterpillars, wasps, ants, beetles, winged flies, midges, thrips, ticks, mites, spiders, and springtails.<sup>1,4</sup> Springtails may have close association with sores in many NCS patients but they, and other opportunistic infections, are not causal factors of NCS sores.

### The Sealants

The three major calcium hydroxide sealants causing NCS (*Dycal, Life and Sealapex*) considered<sup>9</sup> include only about 50% calcium hydroxide in the catalyst (*Table 1*). Of the components common to all three sealants, ethyl-toluene sulfonamide as well as zinc oxide are considered most toxic. Toluene is a known potent nerve toxin.<sup>10</sup> The sulfonamide component of this compound causes a sensitivity allergic-toxic reaction ultimately manifesting as the vascular mucoid sores characteristic of the NCS, especially in sulfa sensitive patients.

Zinc oxide was shown to be genotoxic,<sup>11</sup> cytotoxic,<sup>12,13</sup> killing microphages,<sup>14</sup> and causing chronic and fibrous inflammatory reaction<sup>15,16</sup> ulcerations<sup>16</sup> and osteosclerosis.<sup>17</sup> Additionally, the toxic effects of zinc oxide and calcium hydroxide were shown to be similar.<sup>18,19</sup> Calcium hydroxide was shown to cause periapical inflammation, typical granuloma and partial lack of healing.<sup>20</sup> Titanium dioxide and Barium ions (*Table 1*) were also shown to provoke strong foreign body and bio-incompatible reactions in live tissue.<sup>21,22</sup>

Cytotoxicity of Dycal, Life and Sealapex was clearly demonstrated *in vivo* and *in vitro* in various tissues.<sup>23</sup> Sealapex was shown to cause severe inflammatory infiltration<sup>15,24,25</sup> and edema<sup>25</sup> accompanied by subcutaneous tissue necrosis<sup>15,26</sup> and progressive differentiation and reaction of monocytes, macrophages and epithelial cells.<sup>27</sup> The final phase of the inflammation is characterized by an intense granulomatous reaction especially in epithelial cells causing various intensities of irritation.<sup>28</sup> The cytotoxicity<sup>29,30</sup> and neurotoxicity<sup>31</sup> of Sealapex was well demonstrated in various mammalian systems.

As with Sealapex, Dycal was also shown to cause hemorrhage and acute to consistent inflammatory cells<sup>16,32,33</sup> necrosis,<sup>16,32,33</sup> tissue loss,<sup>33</sup> karyorrhexis,<sup>16</sup> neurotoxicity.<sup>34</sup> and formation of serous exudates.<sup>16</sup> Life has been the least researched sealants. It, however, has the same toxic ingredients, i.e., ethyltoluene sulfonamide and zinc oxide, as Sealapex and Dycal and has been associated with classical NCS symptoms in some of our patients, e.g., DB (*Fig. 6*) and MM (*Fig. 4*).

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Sealants not containing ethyltoluene sulfonamide but including zinc oxide and eugenol have also been associated with NCS cases. These include Fynal (>75% zinc oxide), IRM and Sultan U/P (<50% zinc oxide). Fynal was associated with the cases of MM (Fig. 4). Similarly, IRM (by *Dentsply caulik*) and Sultan U/P (by *Sultan Chemists*) were associated with classical NCS symptoms in some of our patients.

## Case Histories

### Case #1

ME is a Swedish female born in 1951. In 1985 she underwent dental repairs which included the use of Dycal in 20 teeth. ME is allergic to sulfonamides, with IGE values reaching 5000. Every dental treatment was followed by aggressive skin reactions of allergic and toxicological nature (Fig. 3). All tests for parasites were negative. Her symptoms culminated into full blown typical sulfa toxicity reactions including oozing skin and nasal sores with bloody scabs and smelly discharge and an infection with *S. aureus* (Fig. 7). Other symptoms included loss of memory, kidney pain and urgency, sensitivity to light and electricity fields, pin-prick and moving sensations under the skin, and swelling. After each treatment, ME felt totally knocked out with breathing and talking difficulties. She subsequently developed intestinal problems and her skin sores flared up with unbearable and unresolved itching. Photosensitive reactions presented as blotchy skin (Fig. 7) with severe burning sensations in the face, throat and chest.

Dycal was removed in 1991-1992 and initially replaced with Harvard cement. ME was confined to bed with whole body musculo- skeletal system pain, bowel disturbances and signs of polyneuropathy. Shortly after the removal of the Dycal in February, 1992, most of her sores and rashes disappeared and she could tolerate sunlight (Fig. 8).

### Case # 2

Born in Chicago in 1965, JM was a healthy active Caucasian woman until she started experiencing her first symptoms in 1991. By then, she already had 17 fillings. No sealants were used in one filling; Dycal was used in the other 16. Her earliest symptoms appeared as skin break outs on the face and neck, which was recurrent over the following 9 years, accompanied by body tremors, sleeplessness and joint pain with occasional vomiting of black bile. Thrush appeared in the mouth and around the lips. Pain at the teeth roots persisted throughout the nineties associated with rapid major decay. A sensation of prickling pain with a pressure and movement under the skin, urticaria and skin ulcerations would last for weeks or months. JM's body showed random swelling with red marks in serpentine-like shapes. The swellings eventually bottle-necked at the knees and ankles. The chest burned and hurt with strange fits of coughing. JM then started losing hair as she experienced night fevers and sweats, and peeling of the skin.

During the early 1990s JM was medicated with various antibiotics, antiparasitics and herbal remedies. She experienced some anti-inflammatory relief and occasional temporary clearing of ulcers after which ulcers returned and lasted longer. In 1998, massive ulcers appeared on JM's face at the nasiolobial area and on the skin (Fig. 9). A CBC in 1999 was unremarkable except for a high level of Alpha 1- Globulin of 0.5 (normal range 0.2-0.4) and low levels of IgA of 99 (normal range 60-400) and IgG of 724 (normal range 700-1500). The right ocular cavity was severely painful and JM was beginning to lose her eyesight.

A major dental repair was completed in 2001 when Dycal was removed from all 16 teeth. Initially, JM experienced a few episodes of sickness, sweats, and vomiting. After the fourth visit, her eyebrow area had a dramatic reduction in swelling, sensation of movement and in the red hot congestion of her face. JM's teeth were subsequently rebuilt with gold onlays section by section. By the end of the total repair, November, 2001, JM has regained her normal skin (Fig. 10) with no movement sensations or pain anywhere in her body. This state of total resolution has lasted to date without regression or relapses.

### Case #3

LG, a medium-built white American born in 1957, was in perfect health until September 18, 1998, when she had a filling in her tooth no. 18 using Dycal as a liner. She experienced severe headache within 2 hours. By 6:00 p.m. she was vomiting and delirious with the headache persisting. Her blood pressure then was monitored at 169/108 and remained high for the following three years despite repeated attempts to control it with Atenolol and Diazide. LG never experienced high blood pressure or headaches before. An MRI scan was negative. In 1999 LG's health deteriorated progressively with arthritis-like symptoms in her back, heart palpitations, mitral valve prolapse, fatigue, abnormal pap-smears including precancerous cell abnormalities, night sweats, missed periods, and severe depression. By March, 2001, LG, who normally weighed 120 lbs., has lost 20 lbs.

In April, 2001, lesions started appearing on LG's face, which quickly became red hot. Her legs became swollen and painfully burning. By May, 2001, LG had several open lesions (6 mm to 2 cm in diameter) with some surrounding erythema, on her face and scalp. Her cheek pulsed as the facial lesions seemed to track to the chin (Fig. 11) where the most fulminating lesion was; nearest to her teeth. The face was burning hot. Springtails (*Collembola*) and fibers were recovered from these sites. At that time, she showed low lymphocytes of 15.0% (normal 20-43%), high granulocytes of 77.1% (normal 51-74%) and high rheumatoid factor of 22.6 (normal <20 IU/ml). She also tested negative for all communicable diseases then. Her weight dropped to 92 lbs. as she started experiencing movement sensations under the skin of her arms, face

and scalp. Grayish pustular secretions oozed and moved down from the bloody lesions on the scalp and face. The lesion then extended to her legs.

In January, 2002, LG was diagnosed with NCS by OMA. She was allergic to sulfa and sulfonamide compounds. Following our protocol, LG had the filling and the Dycal liner removed from tooth #18 in April, 2002. These were replaced with Starflow and Aria (*a combination of Bisgma, Tegdma, Lidma and catalysts*). Our recommended vitamin supplementation program was initiated then. By May, 2002, all symptoms were resolved (Fig. 12). Constitutional and neurological functions as well as psychological, emotional and energy levels were restored to normalcy.

## Conclusion

*The toxicity of Dycal, Life and Sealapex has been well demonstrated in invivo and invitro studies of various animal and human models by many workers. The toxicity assumed cytotoxic, genotoxic, neurotoxic, phototoxic, necrotic, and inflammatory manifestations compatible with the pathology and symptoms observed in NCS patients. Ethyltoluene sulfonamide, common to all three sealants, is considered the primary cause of the NCS. The toluene component, a known nerve toxin, is believed to be responsible, at least in part, for the neurological symptoms. Neurological abnormalities are related to nerve damage associated with vasomotoric reactions due to a direct influence on the peripheral nerve endings.<sup>35</sup> The sulfonamide component is the cause of the cutaneous symptoms, especially in sulfa-sensitive patients who usually had elevated sulfonamid/sulfa levels in blood tests and allergy to sulfa in skin sensitivity tests. The relationship between sulfonamide and phototoxicity has been well established.<sup>29</sup> Resolving the symptoms (effect) by removing the sealants (cause) in patients undergoing treatments, confirms this cause-effect relationship.*

*The nature of causation of NCS precludes contagious transmission. Any similarities of symptoms among partners within the same household are traceable to the transmission of opportunistic infections, especially fungi.*

*It is recommended not to rehabilitate more than two or three teeth per month. The patient is given a list of vitamins and other supplements to take during the procedure and for the following few weeks until symptoms are completely resolved. After reaching the state of normalcy, the patient may still retain some sensitivity to moldy places lacking sun and fresh air circulation.*

*After additional test results become available and a satisfactory diagnosis of an NCS case is made at the Parasitology Center, Inc. (PCI), arrangements for dental rehabilitation are made and patient prognosis is monitored.*

## Acknowledgment

I am grateful to Marie Erixon, Nordea, Sweden for her contributions to the better understanding of issues related to NCS. ♦

**Table 1.** Components in catalysts (C) and bases (B) of Dycal, Life and Sealapex.

Material	Dycal*	Life*	Sealapex
Calcium hydroxide	51% (C)	51% (B)	NG (B)**
Zinc oxide	9.23% (C)	13.75% (B)	NG (B)
Zinc stearate	0.29% (C)	0.25% (B)	_____
Ethyltoluene sulfonamide	39.48% (C)	34% (B)	NG (B)
Titanium dioxide pigment	_____	10.0% (C)	NG (C)
Pigment	0.1% (B)	0.1% (C)	_____
Calcium phosphate	31.0% (B)	_____	_____
Barium sulphate	_____	37.90% (C)	NG (C)
Zinc oxide	9.0% (B)	_____	_____
Methyl silicate	_____	12.0% (C)	_____
Silicon dioxide NG (C)	_____	_____	NG (C)
Calcium tungstate	17.0% (B)	_____	_____
Butylene glycol disalicylate	43% (B)	_____	_____
Polymethylene mythyl salicylate	_____	38.0% (C)	_____
Isobutyl salicylate	_____	_____	NG (C)

\* See Draheim and Murrey.9

\*\* NG = Percentages not given in the manufacturer's (Kerr Corp.) Material Safety Data Sheet published July 28, 2000.

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**Figs. 1-6.** Cutaneous symptoms in NCS patients.

**Fig. 1.** Early NCS sores on the thigh of KM. She was born in 1964, poisoned with Dycal in two teeth in 1982 and in one tooth in 2002. Neurological symptoms in upper quadrant started in 1997. Cutaneous symptoms began in Spring 2002 preceded by extensive treatment with topical sulfa preparations for possible "mite infestation." Dycal was removed in December, 2002 and recovery is in progress.

**Fig. 2.** Elevated sores on the forehead of KM (Fig.1); note the hot red color of the skin.

**Fig. 3.** Diffuse NCS sores covering the whole body of ME poisoned by Dycal in 1985 (case no.1).

**Fig. 4.** Mucoid NCS/lesions on the face of MM. She was born in 1950, poisoned with Fynal in six teeth in 1981 and in one tooth in 1986 as well as with Life in two teeth in 1985 and 1988.

**Fig. 5.** Histopathological section of one of the roughly 300 sores covering the body of SK. She was born in 1956 and reacted with typical NCS symptoms to a zinc oxide cement (combined with Durelon) underneath a total veneer job in 1982. The section shows hyperkeratosis – like perivascular dermatitis with eosinophils.

**Fig. 6.** Cutaneous sores and swelling in the right hand and arm of DB. Born in 1965, DB had 10 amalgam restorations in 1982 and 1983 using Life. She started experiencing symptoms including ulcerated rash all over the body, unilateral edema and pin-prick and subcutaneous movement sensations in 2001-2002. Life is being removed and recovery is in progress.

**Figs. 7-12.** Facial appearance of the three presented cases before and after recovery from NCS.

**Figs. 7,8** (ME; case no.1); note the hot red face (Fig.7).

**Figs. 9,10** (JM; case no.2); note the lesion on the right cheek and the hot red face (Fig.9).

**Figs. 11,12** (LG; case no.3); note the return of the natural "baby" skin back (Fig.12) after the healing of all facial lesions (Fig.11).

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