Review

Mechanism and clinical aspects of sodium hypochlorite accidents: A narrative review

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Abstract

Sodium hypochlorite (NaOCl) solution is a widely used irrigant in endodontics. However, it is highly cytotoxic and can have destructive effects on surrounding tissues when it is not confined to the root canal during irrigation. The extrusion of NaOCl beyond the confines of the root canal into the surrounding tissues or anatomical spaces is known as a NaOCl accident. The NaOCl accident is a serious iatrogenic mishap that can lead to severe tissue damage and complications, which can be life-threatening and/or cause long-term or permanent consequences with medico-legal implications. Therefore, this narrative review was conducted to provide clinicians with a comprehensive understanding of the mechanism and clinical aspects of NaOCl accidents. A literature search was conducted in various online databases using specific Medical Subject Headings (MeSH) and key search terms. The review included all categories of articles dealing with the NaOCl accident and available as full text. Additionally, a manual method of search was conducted by screening references of the included articles. Duplicate articles and articles available only as abstracts were excluded from the review. The included articles were reviewed, analyzed and discussed according to the following sections: causative factors; mechanism; clinical categorization; clinical manifestations; diagnosis, including history, clinical assessment and examination, clinical investigation, and differential diagnoses; and treatment planning of NaOCl accidents. This would enable clinicians to recognize and manage NaOCl accidents in the best possible manner and minimize their serious consequences. Future research should prioritize the identification of solutions or measures to address the challenges associated with conducting clinical or in vivo studies on NaOCl irrigation and extrusion.

Keywords: accident, endodontics, root canal irrigants, root canal therapy, sodium hypochlorite

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Highlights

- A sodium hypochlorite accident is an iatrogenic incident with the potential for severe complications and medico-legal consequences.
- Clinicians must understand the causes, mechanisms, clinical categorization, manifestations, diagnosis, and treatment of NaOCl accidents for effective management and recovery.
- Future research should investigate the dynamics of NaOCl irrigation and extrusion in clinical settings to achieve higher levels of evidence.

Introduction

Sodium hypochlorite (NaOCl) solution is one of the most widely used irrigants in endodontics due to its capacity to dissolve pulpal tissues and remove smear layer, as well as because of its antimicrobial effects, lubrication properties and low viscosity.[1–5] It is also inexpensive and easily available, with a reasonable shelf life.[2,6] Sodium hypochlorite solution is used in concentrations ranging from 0.5% to 6.0%.[7,8] Although NaOCl is an effective irrigant, its efficacy also depends on the irrigant delivery and activation techniques used. The efficacy of NaOCl has been shown to increase with mechanical or machine-assisted techniques when compared with manual or conventional techniques, despite some variations and limitations among the former.[9] Sodium hypochlorite has certain drawbacks, such as high surface tension, unpleasant odor, as well as discoloring and corrosive effects on endodontic instruments.[1,2,10–14] The corrosive effect of NaOCl can affect the mechanical properties of endodontic instruments and contribute to instrument separation, which is a concern considering the role of nickel–titanium (Ni-Ti) rotary files in modern endodontics. However, a study reported no significant effect of NaOCl on the fracture resistance of Ni-Ti rotary files despite employing a higher concentration of NaOCl (5.25%) at different temperatures.[15] The major concern with NaOCl has been its cytotoxicity, including its tendency to cause cellular (chromosomal) abnormalities at all concentrations.[1,16–21] Sodium hypochlorite is highly cytotoxic to all living tissues, with the exception of heavily keratinized epithelia.[17] The cytotoxic effects of NaOCl include severe inflammation, rapid hemolysis, ulceration, inhibition of neutrophil migration, destruction of endothelial and fibroblast cells, and degradation of cancellous bone.[17–20] This can result in severe damage to both soft and hard tissues.[17,19]

A NaOCl accident is an irrigant mishap, which mainly refers to the extrusion of NaOCl beyond the confines of a root canal into the surrounding periapical or periradicular tissues and/or tissue spaces.[11,22] It can lead to extensive tissue damage, life-threatening situations, and residual or long-term complications.[23–27] Therefore, from the perspectives of clinicians or operators, NaOCl accidents can have medico-legal implications and be associated with malpractice.[28,29] A recent clinical study by Özdemir et al. demonstrated a low rate of NaOCl extrusion and accidents during root canal irrigation with NaOCl.[30] However, it is reported that many practitioners have experienced the NaOCl accident at least once in their career.[31,32] Therefore, it is very important for a clinician or an operator to be completely aware of various aspects of NaOCl accidents to thoroughly understand, manage or prevent them and avoid their associated consequences, including potential medico-legal issues. The purpose of this article is to provide a comprehensive narrative review of the mechanism and clinical aspects of NaOCl accidents, including their causative factors, categorization, manifestations, diagnosis, and treatment planning.

Material and methods

This narrative review was performed following a thorough electronic and manual literature search. The electronic search was conducted in PubMed®/MEDLINE, Web of Science, Google Scholar, Cochrane, Scopus, LILACS, ScienceDirect, and Crossref databases using specific Medical Subject Heading (MeSH) and key search terms, which included "sodium hypochlorite", "irrigant", "irrigation", "rinse", "root canal", "apical", "periapical", "tissue", "accidental", "inadvertent", "extrusion", "extravasation", "injection", "iatrogenic", "error", "mishap", "accident", "adverse effect", "complication", "dentistry", and "endodontics". These terms were used with Boolean operators to search for articles on NaOCl accidents and irrigant-related mishaps. No language restrictions were applied and the articles published up to August 2023 were searched. All categories or types of articles, such as case reports, case series, original studies, and reviews, available as full text, were included in this review. Additionally, a manual search was performed by screening the references of the included articles. Duplicate articles and articles available only as abstracts were excluded from this review. The included articles were reviewed in depth, and relevant details were extracted, compiled, analyzed, interpreted, and discussed under the following sections: causative factors; mechanism; clinical categorization; clinical manifestations; diagnosis, including history, clinical assessment and examination, clinical investigation, and differential diagnoses; and treatment planning of NaOCl accidents.

Discussion

Causative factors for NaOCl accidents

Although a NaOCl accident is considered an iatrogenic mishap mainly caused by the clinician or operator, it occurs in the presence of various contributing or predisposing factors related to the patient (host), tooth, operator, and irrigant (NaOCl).[25,33] The major contributing factors are employing positive pressure or conventional irrigation technique (syringe and needle) and delivering NaOCl in solution form and/or in large amount or volume into the root canal through an open-ended needle or a tightly binding or wedged needle with higher irrigation force, apical irrigation pressure, or NaOCl flow rate during irrigation in a tooth with a large periapical lesion, open or immature apex, wider apex due to resorption, perforation, over-instrumentation, or greater apical patency, larger apical preparation, root defect due to resorption, perforation, or fracture, apical fenestration, close proximity to maxillary sinus, or any iatrogenic error.[25,31–34]

Presently, clinical evidence on the causes for NaOCl accidents is primarily based on case reports, case series, retrospective and observational studies, few studies in the form of surveys, and a couple of systematic reviews on the published case reports and case series.[25,28–31,34–36] Although an observational clinical study reported no statistically significant correlation of demographic characteristics of patients and the preoperative status of teeth to NaOCl extrusion,[30] female sex and non-vital teeth with periapical lesions have been identified as risk factors for NaOCl accidents.[25,31,33,34] Although the NaOCl accidents are mainly caused by the extrusion of NaOCl due to operator factors such as technique, force, pressure, and flow rate employed during NaOCl irrigation, the available clinical data on NaOCl irrigation and extrusion remains limited. Moreover, the data obtained from in vitro studies is inconsistent and not sufficiently conclusive to be extrapolated to a clinical scenario.[30,33–36] Although more clinical research is required on NaOCl irrigation and extrusion, there are many challenges related to the sample size, standardization of predisposing factors, objective measurement of irrigation factors (i.e., force or pressure), tissue back pressure, volume of extruded NaOCl, determination of the extent of tissue damage, and ethical dilemma due to the absence of an antidote to NaOCl and the dearth of data on the volume of NaOCl that could be considered safer upon extrusion.[36]

Mechanism of NaOCl accidents

The extrusion of NaOCl beyond the confines of the root canal can be categorized as either active or passive. Active extrusion occurs when NaOCl is forced under pressure and is mostly responsible for a NaOCl accident.[37–39] On the other hand, passive extrusion happens when NaOCl seeps or leaches out of the root canal without the application of force or pressure.[32,38,40–42] The likelihood of passive extrusion of NaOCl is increased in cases involving an open, immature or widened apex due to root resorption or overinstrumentation, which provides larger apical access for NaOCl seepage. Both active and passive extrusion of NaOCl can lead to a NaOCl accident, exhibiting different mechanisms and manifestations.

As a result of active extrusion, a NaOCl accident occurs following the cascade mechanism:

- extrusion of NaOCl under high force or pressure into the periapical tissues through a patent apical foramen[22,41];
- infusion of extruded NaOCl into the surrounding vasculature. This process requires intact and open blood vessels and a vascular pressure lower than the pressure of extruded NaOCl, as infusion of NaOCl takes the path of least resistance. The infusion of extruded NaOCl occurs in the veins rather than the arteries because the venous pressure is always lower than the arterial pressure in the vasculature. Thus, when the apical pressure extruding NaOCl into the periapical tissues exceeds the venous pressure in the facial vasculature, infusion of NaOCl ensues. The infusion of extruded NaOCl into the venous vasculature can occur through direct or indirect means. Hypothetically, a direct infusion of NaOCl requires intact pulpal and/or periapical vasculature. However, in the absence of such vasculature, the direct infusion of NaOCl may not occur in teeth with pulpal and/or periapical pathology or those undergoing root canal therapy (RCT). Interestingly, a direct infusion of NaOCl into the pulpal vasculature remains possible in vital teeth undergoing endodontic therapy. However, venous congestion in an inflamed pulp and/or pulp extirpation, which often precedes a major part of irrigation, can limit any such potential. Therefore, an indirect infusion of NaOCl into the vasculature occurs when extruded NaOCl reaches the intraosseous or bone marrow space, which contains a network of sinusoids acting like a sponge and a higher intraosseous blood pressure compared to the central venous pressure. Thus, extruded NaOCl is immediately absorbed into the intraosseous or bone marrow space, reaching the venous vasculature due to a higher to lower pressure gradient[22];
- draining of the infused NaOCl through the venous vasculature directly into the pterygoid plexus. However, in the presence of anatomical variations in the facial venous vasculature, the infused NaOCl reaches the facial veins instead of draining into the pterygoid plexus. Apart from releasing chemical mediators, the infused NaOCl causes damage to the vessels and increases vascular permeability. Thus, the infused NaOCl indirectly comes into contact with surrounding soft tissues, leading to a NaOCl accident with typical signs and symptoms.[22,43,44]

In accordance with the abovementioned mechanism, NaOCl accidents mainly occur in instances of active extrusion of NaOCl under force or pressure. However, it has been established that a NaOCl accident may not occur in every patient, even in cases where similar irrigation methods are employed. Two theories have been advanced to explain this phenomenon, namely the threshold theory and the compliance theory.

According to the initially proposed threshold theory, a NaOCl accident occurs only if active extrusion takes place, with the irrigation pressure near the apical foramen exceeding a threshold of back pressure offered by the surrounding tissues. Therefore, when the pressure is below this threshold, a NaOCl accident may not occur, despite forceful irrigation.[22,32,45–48] However, this theory has certain limitations which are related to the size of pressure sensors employed in the studies, lack of validity for the positive back pressure concept as the influx of fluid into an empty root canal does not occur, absence of consistency in the values of back pressure at the apical foramen, and findings reporting that conventional irrigation pressure can actually exceed the stated thresholds.[32] Therefore, the compliance theory has been proposed as an alternative concept. According to this theory, back pressure is not constant and is influenced by the differences in the anatomy of the periapical tissues.[31,32,34,42] Therefore, the compliance of the periapical tissues plays a crucial role in back pressure offered toward the extrusion of NaOCl.[32,42] Thus, a classic NaOCl accident occurs only when NaOCl is extruded with a pressure that exceeds the pressure from the surrounding tissues and the vasculature and is infused into the venous blood flow.

The aforementioned mechanism and theories explain why NaOCl accidents are less prevalent despite the common nature of RCT.[22] This mechanism also clarifies why NaOCl accidents with classic features may not occur in cases of passive extrusion of NaOCl into the periapical tissues.[22,32,42] However, in the event of passive extrusion, NaOCl can still come into contact with the periapical tissues, exerting a direct cytotoxic effect on them, even though cortical bone is generally resistant to the effects of NaOCl.[19] This situation can lead to a NaOCl accident without typical signs and symptoms. Although classic features of a NaOCl accident may not manifest, extrusion of even a small amount of NaOCl can lead to pain. Sodium hypochlorite extrusion is one of the contributing factors for postoperative or post-treatment endodontic pain. Similarly, passive or active extrusion of NaOCl into an empty or surrounding anatomical space, such as the maxillary sinus, can result in a NaOCl accident without typical manifestations. Additionally, when NaOCl is extruded into the surrounding soft tissues due to iatrogenic errors, such as perforation, or directly into a soft tissue space, such as the buccal and infraorbital spaces, through the periapical area or bone fenestration, NaOCl directly exerts its cytotoxic effect, actively destroying surrounding soft tissues and creating its own planes. This spread may occur in a haphazard or unpredictable manner, accompanied by symptomatology that is less typical of NaOCl extrusion into the periapical tissues.[31,49,50] The various types of NaOCl extrusion and various pathways for extrusion can influence the mechanism and manifestations of NaOCl accidents. These pathways have been schematically depicted in Fig. 1.

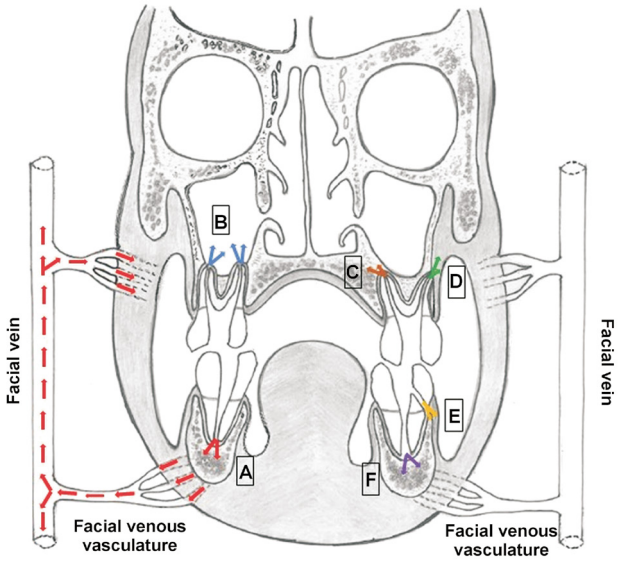

Fig. 1. Schematic illustration depicting types and pathways of sodium hypochlorite (NaOCl) extrusion that influence the mechanism and manifestations of NaOCl accidents

A – active extrusion of NaOCl via root apex into hard tissues (periapical tissues) with an indirect (hematogenous) pathway via facial vein to surrounding tissues (red arrows); B – active extrusion of NaOCl via root apex into an anatomical space (maxillary sinus) with a direct pathway to surrounding tissues (blue arrows); C – active extrusion of NaOCl via root apex into hard tissues (periapical tissues) with a direct pathway to surrounding tissues (orange arrows); D – active extrusion of NaOCl via bony defect (e.g., apical fenestration) into soft tissues or fascial spaces with a direct pathway to surrounding tissues (green arrows); E – active extrusion of NaOCl via root defect (e.g., root fracture or iatrogenic root perforation) and a direct pathway into the surrounding soft tissues (yellow arrows). Similarly, active extrusion into hard tissues (periradicular tissues) with an indirect pathway to surrounding tissues can occur if the defect is located beyond the cervical third of the root; F – passive extrusion of NaOCl via root apex (e.g., open apex) into hard tissues (periapical tissues) (purple arrows). Similar tendencies may also exist for passive extrusion of NaOCl via root apex into an anatomical space (maxillary sinus) or via root defect into soft tissues or tissue spaces.

Clinical categorization and manifestations of NaOCl accidents

Sodium hypochlorite accidents can be broadly categorized based on their manifestations and the mode and site of extrusion (Table 1).[22] Their clinical manifestations may be further categorized based on various factors (Table 2).[51]

Table 1. Clinical categorization of sodium hypochlorite (NaOCl) accidents

Basis	Category
Mode of NaOCl extrusion	• NaOCl accident due to active extrusion: – direct pathway to the surrounding tissues – indirect pathway to the surrounding tissues (hematogenous route) • NaOCl accident due to passive extrusion
Site of NaOCl extrusion	• NaOCl accident due to extrusion into the surrounding soft tissues or fascial spaces and hard tissues (such as periapical and periradicular tissues) • NaOCl accident due to extrusion into the surrounding or anatomical tissue spaces (such as the maxillary sinus)
Manifestations depending on the mode and site of NaOCl extrusion	• NaOCl accident with classic or typical manifestations • NaOCl accident with non-classic or non-typical manifestations

Table 2. Categorization of clinical manifestations of sodium hypochlorite (NaOCl) accidents

Basis	Category
Site of clinical manifestations	• local manifestations: – intraoral features – extraoral features • systemic manifestations
Duration or time required for the onset or persistence of clinical manifestations	• short-term manifestations: – immediate or early features – later or delayed features • long-term or residual manifestations
Extent of clinical manifestations	• mild manifestations • moderate manifestations • severe manifestations

NaOCl accident due to NaOCl extrusion into the surrounding tissues

Accidents involving NaOCl typically result from the extrusion of NaOCl into the surrounding soft tissues or fascial spaces and hard tissues, including periapical and periradicular tissues. Documented cases of accidents involving NaOCl under this category have been reported in both permanent and primary teeth.[25,34] However, the number of cases involving primary teeth is limited.[49,52–58] The clinical manifestations and management of NaOCl accidents do not differ significantly between permanent and primary teeth.

Local manifestations

Short-term manifestations

Short-term manifestations include both soft tissue and hard tissue manifestations with intraoral and extraoral features.

Immediate or early features occur immediately after NaOCl extrusion, although in some cases, they may manifest after few hours.[59] They are mainly due to soft tissue damage following a NaOCl accident. Sudden pain, profuse bleeding from the root canal, immediate swelling, and ecchymosis are the characteristic features of NaOCl extrusion into the periapical tissues.[25,60] The intensity of pain varies depending on the extent of the NaOCl accident, pain threshold of the patient, and whether anesthesia was administered during RCT. Some patients treated under local anesthesia (LA) may not experience pain immediately.[61] Otherwise, pain would be severe even if the patient was anesthetized.[62] With NaOCl spreading rapidly over a wide region, pain management becomes difficult because symptoms from distant anatomic structures will continue to cause discomfort. This phenomenon also explains the extreme pain experienced during NaOCl accidents, despite the implementation of adequate LA prior to treatment initiation.[31,63] The profuse bleeding from the canal, including the gingival margin,[50,64] is due to the body's reaction to NaOCl.[63] The extruded NaOCl affects vascular permeability by damaging the vessels and releasing chemical mediators. This results in interstitial hemorrhage, causing immediate swelling, severe bleeding from the canal, and ecchymosis.[43,44]

Swelling manifests as both large and diffuse, appearing within minutes to hours after the extrusion. It extends intra- and extraorally,[65] and may lead to difficulty in the opening of the eye on the affected side.[59,66,67] The distended soft tissues cause a sensation of tightness and contribute to pain.[60] Swelling may also manifest as emphysema with crepitus due to the use of positive pressure irrigation with open-ended needles to deliver NaOCl or extrusion of compressed air or hydrogen peroxide along with NaOCl,[56,68,69] as well as oxygen liberation into the tissues because of the oxidizing ability of NaOCl.[66,69]

Ecchymosis is another salient feature of NaOCl accidents and can be broadly categorized considering its location and extent (Table 3).[22] Ecchymosis typically manifests extraorally and unilaterally along the angle of the mouth and around the periorbital region of the affected side, following the course of the facial superficial venous vasculature. This is attributed to the mechanism of NaOCl accidents, where extruded and systemically absorbed NaOCl eventually reaches the facial vein, causing extraoral ecchymosis.[22] Due to the same reason, NaOCl accidents typically manifest with a similar pattern of extraoral ecchymosis, irrespective of the tooth involved.[22] Most often, extraoral ecchymosis does not manifest in the cheek or middle third of the face because the malar fat pad and zygomatic muscles cover the facial venous vasculature, concealing the area of underlying interstitial hemorrhage.[22] Occasionally, ecchymosis may manifest intraorally[22,23]

Table 3. Categorization of ecchymosis following a sodium hypochlorite (NaOCl) accident

Basis	Category
Site of involvement	• intraoral ecchymosis • extraoral ecchymosis
Side of involvement	• unilateral ecchymosis • bilateral ecchymosis
Extent of involvement	• grade 1: ecchymosis is not observed • grade 2: ecchymosis is evident, involving the angle of the mouth and the periorbital region (facial ecchymosis) • grade 3: ecchymosis is evident, involving the regions of grade 2 and extending into the neck region (facial and cervical ecchymosis) • grade 4: ecchymosis is observed, involving the regions of grade 3 and extending into the chest area (facial, cervical and mediastinal ecchymosis)

or be observed bilaterally by crossing the midline[70] or extending to the contralateral periorbital region due to any communication between left and right anterior facial veins.[49,55,69] The extent of ecchymosis depends on the amount of loose tissue present in the surrounding area.[70] However, ecchymosis does not manifest in the intraoral soft tissues around the apical area of the involved tooth, as intravenous infusion of extruded NaOCl into the local circulation is least likely due to the lack of pulpal and/or periapical blood supply in relation to the involved tooth.[22]

Later or delayed features manifest within 24–48 h or several days after the NaOCl accident. These include life-threatening airway obstruction due to Ludwig's angina,[23,24] dysphagia,[71] dysphonia, drooling saliva,[23] trismus,[23,72] purulent discharge from periapical tissues or necrotic soft tissues due to secondary infection,[60,66,73–75] ulceration,[65] formation of a fistula with ulceration,[76] hard tissue damage in the form of demineralization and necrosis of bone,[19,77] neurological complications such as altered sensation,[69] residual anesthesia, paresthesia, and loss of sensory and/or motor functions affecting speech and swallowing due to the involvement of the trigeminal, infraorbital,[78] or isolated facial nerves,[44,64–66,68,70,72–74,76,79–84] and ophthalmological complications such as eye pain, blurring of vision, blepharospasm,[78] diplopia, and corneal patchy coloration.[62,73]

Long-term or residual manifestations

Long-term or residual effects may persist for many months or years. They include neurological deficits manifesting as persistent or permanent paresthesia,[76,85,86] as well as permanent damage to mimic musculature, trigeminal nerve weakness,[78,82,87,88] ocular damage,[87] hypertrophied or excessive granulation tissue and fibrosis with or without a foreign body-like reaction,[27,44,77,89] mucosal or facial tissue scarring,[49,65,90] facial atrophy,[61,88] facial discoloration,[91] cosmetic deformity,[25,44,64,73,74] and damage to the permanent tooth follicle in case of a NaOCl accident in a primary tooth.[92,93]

NaOCl accident due to extrusion of NaOCl into the surrounding anatomical spaces

The extrusion of NaOCl into the surrounding anatomical spaces involves tissue spaces, such as the maxillary sinus. Although the risk of extrusion of NaOCl into the maxillary sinus is higher, only few cases have been documented in comparison to extrusion of NaOCl into soft and hard tissues (periapical and periradicular tissues). The literature has documented such cases only in the context of permanent teeth.[37,74,87,89,94–97] The clinical features of extrusion of NaOCl into the maxillary sinus also differ from those of extrusion of NaOCl into soft and hard tissues. The extrusion of NaOCl into the maxillary sinus generally manifests with non-classic or non-typical features.

Local manifestations

Short-term manifestations

Immediate or early features include dripping of extruded NaOCl with a taste of chlorine from the nose or throat due to its flowing from the nostrils, a burning sensation in the maxillary sinus, epistaxis, blood or blood-stained fluid dripping in the throat, breathing disturbances, and sinusitis or sinus congestion.[37,77,89,95–97] Classic or typical features such as severe pain, swelling, ecchymosis, and bleeding from the canal may not be seen, as NaOCl extruded into the maxillary sinus, which is not an enclosed space, may be evacuated from the nostrils with limited time of contact in the sinus.[25,37,68,89,94–97] Nevertheless, few cases with immediate severe pain and swelling have been reported.[77,95,97] Symptoms such as the taste of chlorine and NaOCl running down through the nose and throat can also manifest with the extrusion of NaOCl into the periapical tissues of maxillary incisors due to their proximity to the nasal cavity or floor.[83]

Later or delayed features consist of any necrosis or sequestration of surrounding or adjacent bone and associated features.[77,87]

Long-term or residual manifestations

Long-term manifestations include hypertrophy or thickening of sinus lining, neurological complications such as paresthesia due to infraorbital nerve damage, ocular complications such as dystonia of the eye and enophthalmos due to damage to the orbital floor, mobility of the tooth, and oroantral fistula or communication as an associated complication following the extraction of the involved tooth.[77,87,89]

Systemic manifestations of NaOCl accidents

NaOCl accidents with systemic involvement are rarely reported, and the tendency for systemic involvement seems to be limited to cases of grade 4 ecchymosis extending to the cervical and mediastinal areas.[22] However, a NaOCl accident can potentially lead to systemic manifestations in a medically compromised patient.[26] Barbas et al. reported the death of a patient due to systemic involvement following NaOCl irrigation during RCT in a maxillary tooth.[98] The irrigation with NaOCl was identified as the probable cause of the patient's sudden coma and subsequent death, despite their reported health status. Based on the available circumstantial evidence, the death was attributed to an acute rise in blood pressure and intracerebral hemorrhage resulting from severe pain and/or sudden trigeminal nerve stimulation following the irrigation with NaOCl, which may have been extruded through the root apex. The reported symptoms prior to the patient's unresponsiveness were indicative of a NaOCl accident.[98]

Diagnosis of NaOCl accidents

The diagnosis of NaOCl accidents involves history, clinical assessment and examination, clinical investigations, and differential diagnoses.

History

The majority of NaOCl accidents are reported during the RCT itself (under primary care). However, when such cases are referred or reported to secondary care (i.e., oral and maxillofacial surgery unit, emergency unit, hospital, or medical and/or dental specialist), they could be either an immediate or early referral with all the classic or non-classic signs and symptoms or complications, or a delayed referral, with features of residual or long-term effects.[51,77,87–89] In such cases, a comprehensive history of RCT, along with the rapid or sudden onset of salient features such as pain and swelling during the procedure, prevailing signs and symptoms, and clinical and radiological examination, would facilitate the diagnosis.

Clinical assessment and examination

The intensity of pain should be assessed using the numeric rating scale or the visual analogue scale. These instruments facilitate the categorization of the extent of a NaOCl accident and, consequently, the formulation of a treatment plan. They also serve as a baseline record to monitor the progress and management of a NaOCl accident.[51,99] Although pain evaluation using these scales is subjective, it can still be considered a useful tool for the assessment of the extent of pain.[100] In cases where patients are already experiencing significant discomfort, and where time is of the essence in managing a NaOCl accident, these scales can be instrumental in rapidly evaluating pain and mitigating further distress to the patient. They can facilitate the immediate initiation of emergency measures to control pain and other symptoms.

Vital signs must be meticulously monitored, particularly in cases where systemic involvement is observed following substantial extrusion and infusion of NaOCl into the vasculature. This is needed to monitor the patient's well-being, as systemic involvement can be life-threatening.[98]

The extraoral examination consists of facial, airway, neurological, and ophthalmologic assessment. The facial assessment involves the observation of any asymmetry, swelling or ecchymosis. The airway assessment is crucial for detecting signs such as high-pitched wheezing (stridor), hoarseness, coughing, difficulty swallowing, and labored or rapid breathing, which would necessitate emergency intervention. The neurological assessment involves the examination of cranial nerves, particularly the trigeminal and facial nerves, to assess sensory and/or motor functions in the affected area.[23,27,51,99] The examination of nerves should be performed once the anesthetic effect has worn off.[25] The ophthalmologic assessment is of critical importance, especially in cases of NaOCl accidents involving the maxillary region, to avoid ocular complications.[87,99]

The intraoral examination involves the assessment of the treated tooth, sensory and/or motor functioning of nerves in the affected area, as well as any associated swelling, ecchymosis, ulceration,[65] or necrosis of soft and hard tissues.[77] The involved tooth and the associated gingiva are usually tender to percussion.[51] Additionally, the presence of a chlorine odor in the affected area can assist in the diagnosis of patients who have been referred with a NaOCl accident.[76,79]

Clinical investigation

The clinical investigation involves the radiological assessment using periapical radiography,[65] dental panoramic tomography,[72] cone beam computed tomography (CBCT) or dental volumetric tomography (DVT),[89,101] computed tomography (CT),[49,69,77,86,87] and magnetic resonance imaging (MRI).[64] Since conventional radiographs have certain limitations,[32,71] advanced imaging techniques such as CBCT or DVT, CT, and MRI have been suggested depending on the extent of damage following a NaOCl accident.[32,51] These advanced techniques also allow for preoperative identification of predisposing factors for NaOCl accidents, three-dimensional visualization of the affected area, revealing the cause of an accident, and reduction of time spent in assessing the prognosis of the involved tooth, particularly in a referred case of a NaOCl accident.[60,101] In CBCT, the affected region shows the presence of multiple round or ovoid air-bubble-like voids and low-density areas within the soft tissues.[60] Computed

tomography and MRI are suitable for the assessment of affected areas in relation to the maxillary sinus.[77,102] Nasal endoscopy, or nasoscopy, is also recommended in cases of NaOCl extrusion into the maxillary sinus.[77,86,87,97] Medical or laboratory investigations including blood and urine analysis can be considered in cases of systemic manifestations following a NaOCl accident.[103–105]

Differential diagnoses

The following conditions must be ruled out while diagnosing NaOCl accidents, especially in referred cases:

– mucosal injury with salicylate or other chemicals or solvents, particularly in cases of intraoral ulceration.[105,106] Similarly, in the presence of features such as pain, intraoral ulceration, soft and hard tissue necrosis, and paresthesia, the exclusion of any seepage of NaOCl during RCT is imperative. The absence of immediate pain and swelling during NaOCl irrigation and extraoral ecchymosis may assist as differentiating features[107–109];
– extrusion of other irrigants such as hydrogen peroxide, chlorhexidine, ethylenediaminetetraacetic acid, and citric acid. When a regimen of multiple irrigants is employed during RCT, a similar potential for extrusion exists. Extrusion of other irrigants can also lead to signs and symptoms such as pain, swelling, ulceration, and paresthesia, which are similar to those caused by a NaOCl accident. However, features such as crepitus and the absence of facial ecchymosis may facilitate a differential diagnosis. The management of extrusion of these irrigants is based on similar principles[110–112];
– injury due to inadvertent injection or extrusion of gutta percha solvents such as xylene and chloroform, particularly in a patient undergoing endodontic retreatment[113,114];
– direct injection of NaOCl instead of LA into the surrounding tissues of the oral cavity. This iatrogenic error can manifest with signs and symptoms analogous to those associated with NaOCl accidents, including pain, swelling, ulceration, and paresthesia. However, facial ecchymosis, a salient feature of NaOCl accidents, is mostly absent. Since the LA step generally precedes the irrigation procedure during RCT, confirming this may assist in ruling out the possibility of direct injection of NaOCl. However, when LA is administered during root canal cleaning and shaping and the irrigation procedure, direct injection of NaOCl into the surrounding tissues must be considered under differential diagnoses. The protocols for managing NaOCl accidents and direct injection of NaOCl are similar[102,105,115–117];
– orofacial infection such as an acute periapical abscess with cellulitis.[51,80,81,90] However, the presence of features such as history of pain and swelling that develops suddenly during RCT, facial bruising or ecchymosis, and sensory or motor deficit help to rule out such an infection[80,81,90,118,119];
– allergic or immediate hypersensitivity reaction to household products, medications, local anesthetics, and rubber dam.[24,118,120] The absence of history of such allergic reactions, the presence of intense pain and the clinical features that are mostly seen unilaterally on the side corresponding to the treated tooth, the absence of breathing difficulties or cardiorespiratory manifestations, and the lack of itching and/or rashes on the skin facilitate in ruling out an allergic reaction[24,105,118,121];
– air or tissue emphysema. The presence of immediate and intense pain, swelling mostly confined to the affected side, and the absence of crepitus and erythema help to rule out air or tissue emphysema[121];
– hematoma formation[121];
– angioneurotic edema. Although it could be rapid in its onset, facial swelling is typically bilateral and may involve other areas of the body, manifesting as urticaria and skin rashes. The absence of these features, the absence of a history of recurring episodes of such facial swelling, and the occurrence of signs and symptoms during or following RCT would assist in ruling out angioneurotic edema[120,121];
– interappointment and post-treatment endodontic pain or flare-up. These must be considered under the differential diagnoses in the presence of iatrogenic errors, such as overinstrumentation, and when signs and symptoms, such as pain and swelling following a NaOCl accident, start to appear late.[100,122]

Treatment planning for NaOCl accidents

Following confirmation or diagnosis of a NaOCl accident, it can be clinically categorized based on its manifestations and extent. The extent of a NaOCl accident is influenced by various factors, including the host, the site of extrusion, the concentration and amount or volume of extruded NaOCl, the spatial location of the fluid introduction, and proximity to vital anatomic structures.[41,68,73,123] Sodium hypochlorite accidents can be categorized into mild, moderate and severe. The categorization and treatment planning for NaOCl accidents can be carried out in the following manner.

Mild category

Mild NaOCl accidents are characterized by pain at the lower end of the pain scale and are localized to the tooth undergoing RCT. These accidents result in swelling less than 30% relative to the contralateral side and localized ecchymosis. The management of this condition can be performed under primary care[51] or on an outpatient basis in a hospital.[84]

Moderate category

Moderate NaOCl accidents are characterized by an increase in pain, with an intensity in the mid-range on a pain scale, swelling up to 50% relative to the contralateral

side, diffuse ecchymosis, intraoral ulceration adjacent to the treated tooth, and cosmetic deformity. Such cases require management under secondary care in a maxillofacial unit[51,99] or emergency unit, or on an outpatient basis in a hospital.[84]

Severe category

The severe category of NaOCl accidents is characterized by intense pain at the higher end of the pain scale, swelling greater than 50% relative to the contralateral side, diffuse ecchymosis, intraoral ulceration and necrosis of soft tissues, compromised airway, neurological and ophthalmological complications, and cosmetic deformity. Severe cases must be managed under secondary care in a maxillofacial unit or emergency unit[51,99] or on an inpatient basis with hospitalization and intensive care.[23,24,26]

A comprehensive diagnosis involving history, clinical assessment and examination, and clinical investigations, as well as the categorization of manifestations and their extent would assist clinicians in decision-making to manage NaOCl accidents and minimize their consequences (Fig. 2). The management and prevention of NaOCl accidents will be addressed as a separate topic, given the extent of content to be reviewed and discussed.

Conclusions

A NaOCl accident is a serious iatrogenic mishap with potential medico-legal consequences. Although clinicians must implement measures to prevent it, an element of risk exists during root canal irrigation with NaOCl. Therefore, dental professionals must thoroughly understand and be aware of various aspects of NaOCl accidents. This narrative review was conducted to elucidate the mechanism and clinical aspects of NaOCl accidents. It provides clinicians with a detailed overview of the causes, mechanism, clinical categorization, clinical manifestations, diagnosis, and treatment planning of NaOCl accidents, thereby enabling them to manage NaOCl accidents in the most optimal manner and minimize or avert their serious consequences, including medico-legal implications. However, this narrative review is based on the currently available clinical evidence on NaOCl irrigation and NaOCl accidents or extrusion in the endodontic literature, which is considered to be limited. Hence, there is a need to accumulate higher-level evidence through additional clinical studies on NaOCl irrigation and extrusion. Future research should prioritize the identification of solutions or measures to address the challenges associated with conducting clinical or in vivo studies on NaOCl irrigation and extrusion.

Ethics approval and consent to participate

Not applicable.

Data availability

The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request.

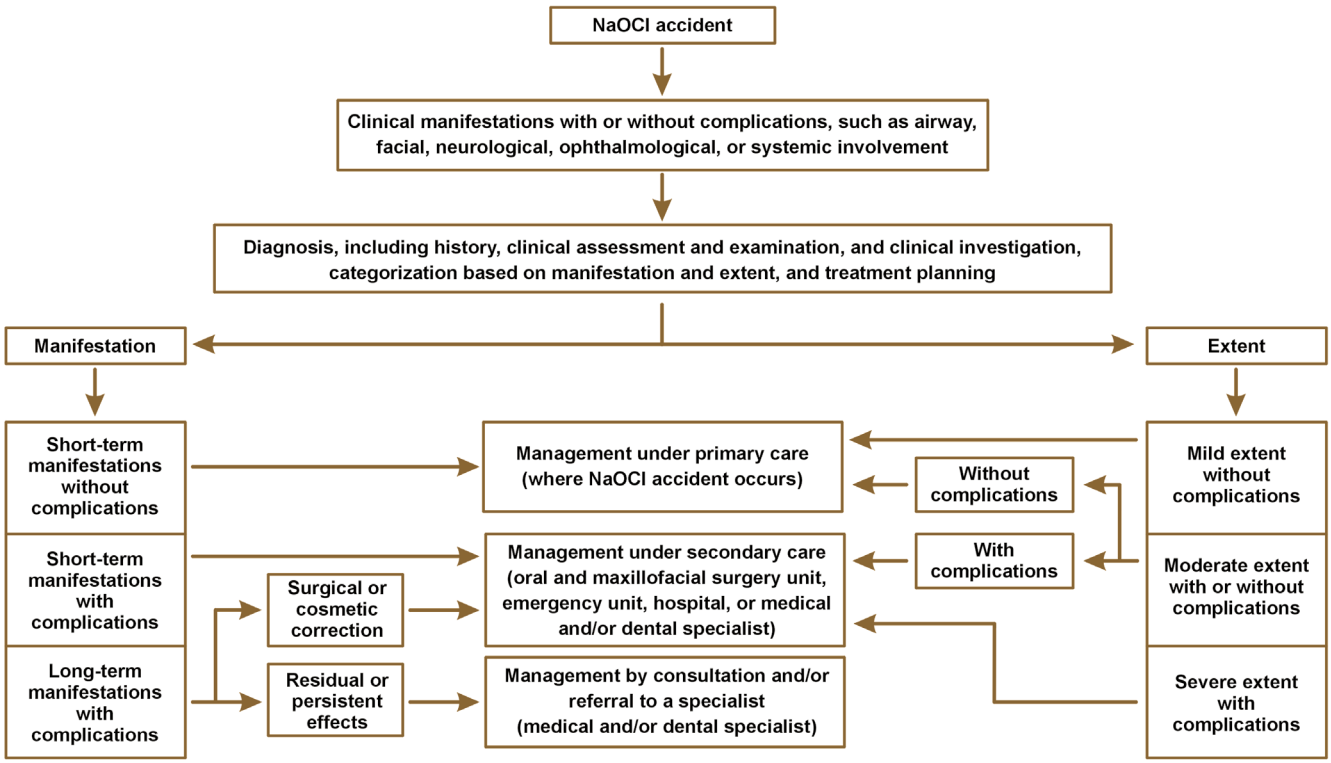

Fig. 2. Flowchart depicting the clinical decision-making process concerning treatment planning and management of sodium hypochlorite (NaOCl) accidents

Consent for publication

Not applicable.

Use of AI and AI-assisted technologies

Not applicable.

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