Review

Forensic onychology of heavy metal exposure: forensic dermatology of the manifestations of heavy metal toxicity in nails

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Keywords: dermatology, exposure, forensic, heavy, lines, mees, metal, nail, onychology, toxicity

Dermatology Online Journal

Vol. 31, Issue 2, 2025

Abstract

Fingernails and toenails can be an important source of trace evidence at a crime scene investigation. Arsenic, gold, lead, mercury, selenium, silver, and thallium are heavy metals; exposure to these metals can result not only in dyschromia of the nail, but also dystrophy of the nail plate. Mees lines, either single or multiple transverse white bands on the nail, were originally described in association with arsenic exposure. Similar white horizontal bands of transverse leukonychia have also been observed in patients following exposure to selenium and thallium. A diagnostic clue for persons who investigate forensic crime scenes to the possibility of heavy metal toxicity in the victim can be changes in the fingernails and toenails. The nails can be photographed and subsequently analyzed for the presence of the causative metal when the possibility of heavy metal exposure is entertained by crime scene investigators and/or medical examiners or coroners.

Introduction

Trace evidence is evidence that is extremely small and may not be visually noticed with the naked eye; it may necessitate additional tools and techniques for recognition. Trace evidence in a crime scene investigation can include blood, hair, saliva, and semen. It also can include not only subungual material (eg, blood and tissue), but also fingernails and toenails.[1-4]

Heavy metals are metallic elements with density, atomic weight, and/or atomic numbers that are relatively high. Heavy metal exposure can be toxic to people, and certain heavy metals, such as arsenic, gold, lead, mercury, selenium, silver, and thallium, can result in morphologic changes to the nail plate. Evaluation of the nail can confirm the presence of the causative heavy metal in these individuals; indeed, the metals can be detected in the nail plate.[5-7]

Nail changes can be a diagnostic clue for individuals who investigate forensic crime scenes to the possibility of heavy metal toxicity in the victim. Some of the individuals with an exposure to a heavy metal concurrently have other mucocutaneous manifestations. The potentially associated nail changes from heavy metal exposure are discussed, other heavy metal-related dermatologic features are summarized, and their implications for forensic evaluation by crime scene investigators and medical examiners or coroners are emphasized.

Discussion

Nail plate-related changes secondary to heavy metal exposure can present with changes in the color or morphology of the nail plate; analysis of the individual's nail can document exposure to the specific metal.[8-41] Mees lines (ie, transverse white lines) were the first nail dyschromia described in association with heavy metal exposure.[8-15] The history of these white horizontal lines initially observed following arsenic exposure and subsequently noted after the exposure to other heavy metals (eg, selenium and thallium) is described. In addition, the nail findings and the mucocutaneous features discovered in individuals exposed to arsenic, gold, lead, mercury, selenium, silver, and thallium are summarized with previously published illustrative cases. The importance of crime scene investigation evaluation for nail-related findings of exposure to heavy metals is also emphasized.

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Table 1. *Mees Lines: A Historic Perspective.*

Pub year	Author	YOB -YOD	Contribution	Ref
1900	Ernest Septimus Reynolds	1861 -1926	Reynolds was an assistant physician to the Manchester Royal Infirmary in England. He observed a polyneuropathy-associated illness in the beer drinking population. Reynolds realized the cause of death was not alcohol but arsenical poisoning. He published similar descriptions of his observations in three journals between 1900 and 1901. In addition to alcohol-related nail changes in which he described transverse leukonychia and horizontal ridging, he also recorded other dermatologic findings associated with the arsenic exposure including painful red neuralgia of the extremities, crimson soles, wet palms, puffy skin, keratoses, erythemata eruptions, and pigmentation changes.	9
1901	Florence Rena Sabin	1871 -1953	Sabin's patient was admitted to the hospital November 21, 1900, a few hours after taking arsenic; she was discharged 5 days later; her feet were numb. On January 1, 1901, the foot numbness was worse, and she could not walk. On or around January 7, 1901, she was again admitted to the hospital with double foot drop and wrist drop. She had keratoses on the plantar feet and palmar hands. Her skin was dry and scaly, and a transverse white line ran across each nail of both hands. Her paper was published in 1901; the title emphasized the neurologic features of her patient: "a case of arsenical neuritis."	10
1904	Charles John Aldrich	1861 -1908	Aldrich was a neurologist; he was a lecturer on clinical neurology and anatomy of the nervous system at the College of Physicians and Surgeon in Cleveland, Ohio. He described three patients with arsenic exposure. The first had a severe arsenical neuritis; he emphasized the white streaks on her nails that extended from side to side. The second and third patient both had similar transverse white streaks on their nails and exposure to arsenic. He emphasized the nail findings in his paper that was published in 1904; the paper was titled "leukonychia striata arsenicalis transversus: with report of three cases."	11
1919	Rudolf Adriaan Mees	1873 -1964	Mees was a doctor in the provincial hospital located in Duin en Brosch, Bakkum, Castricum, in the Noord-Holland province of Netherlands. He presented three patients with polyneuropathy due to acute arsenic intoxication following a large single dose of arsenic. He described the white transverse bands on the nails, suggested the nail sign could be used not only to diagnose neuropathy caused by acute arsenic poisoning, but also in forensic analysis.	12

Abbreviations: Pub, publication; Ref, reference; YOB, year of birth; YOD, year of death

History of Mees lines

Leukonychia refers to white nails. The clinical appearance can either be total, striate (either transversal or longitudinal), and/or punctate; another presentation is incompletely white nail (ie, leukonychia partialis). In addition to the morphologic classification, leukonychia can be classified based on the anatomic site of involvement: true leukonychia (in which the abnormalities involve the intrinsic nail matrix and nail plate), apparent leukonychia (in which the pathology involves the subungual tissues), and pseudo-leukonychia (in which the whiteness only involves either the dorsal or ventral nail plate.[8]

The term *Mees lines* is established in the literature to describe the transverse longitudinal leukonychia associated with arsenic poisoning (**Table 1**).[9-12] However, Mees was not the first person to publish this observation.[13,14] Also, Mees lines have been observed in several other settings.[8,13,14]

Dr. Ernest Septimus Reynolds is to be credited with the first description of arsenic toxicity-related transverse white bands on the nails in 1900. He published the observations he had made during the prior 12 months regarding an epidemic outbreak of arsenical exposure that was occurring in beer drinkers. He published his observations in several journals, the *British Medical Journal* (in issues from November 24, 1900, December 1, 1900, and December 22, 1900), *The Lancet* (January 19, 1901), and *Medico-Chirurgical Transactions* (January 8, 1901).[9,13,14]

Dr. Reynolds stated, "In many cases the nails are affected. After the patients have stopped taking beer for some weeks the best appearances are seen, for then there is a transverse white ridge across the nail; proximal to this the nail is normal, but distal to it the nail is whiter, cracked, thin, and towards the tip almost papery and much flattened. In some cases, there have been a series of parallel transverse ridges of the nails, also suggesting a series of week-end drinking bouts. These deformed nails of course break easily."[9]

Dr. Reynolds, also described that several other dermatologic manifestations of arsenic toxicity including, painful red neuralgia of the extremities, puffy skin, red palms, wet palms, keratoses on the palms and soles, erythemata (consisting of variable red rashes), and pigmentation (not only consisting of darkening of preexisting freckles but also showing well-marked lighter spots, like "rain drops").[9]

In 1901, Dr. Florence Rena Sabin published her findings regarding a young woman; the patient had ingested about a "dram of 'Rough on Rats,'" which was a poison that contained arsenic.[10,14] Dr. Sabin had presented the woman at the Proceedings of the Johns Hopkins Medical Society, Dermatology Section in December 1900.[10] The patient had developed neurologic symptoms including numbness of her feet, double foot drop, and wrist drop.[10,14] In addition, Dr. Sabin noted that "on both hands there is a white line running transversally across each nail."[10]

Dr. Charles John Aldrich also published a paper on arsenic exposure-associated horizontal white nail plate bands. In 1904, he reported three cases and titled his paper and referred to the condition as "leukoconychia striata arsenicalis transversus."[11,13,14] He likely saw the first patient in 1988; she had been suicidal and ingested "a teaspoon of 'Rough on Rats,' which is well known to contain a large quantity of arsenic. The white streaks were about one-sixteenth of an inch in width, quite regular, with fairly sharp margins and occupying an identical position on each nail."[11]

About 12 months after seeing the woman, Dr. Aldrich saw a man who had "all the symptoms of a multiple neuritis. Inspection of his nails showed the characteristic transverse streaks…".[11] The man "confessed to having taken a large dose of white arsenic…".[11]

Dr. Aldrich's third patient was seen in May 1899. He was a 29-year-old man who "had taken a quantity of 'Rough on Rats,' with suicidal intent."[11] He also developed the "white, slightly crescentic transverse streak on each finger-nail."[11]

Dr. Rudolf Adriaan Mees published three patients with a phenomenon in polyneuritis arsenicosa in 1919 based on a presentation given at the meeting of the Association for Psychiatry and Neurology on October 11, 1918.[11-14] He described that "white transverse bands are seen running over the nails, which clearly have nothing to do with the lunula;" although Dr. Mees was aware of Dr. Reynolds papers, Dr. Mees did not acknowledge Dr. Reynolds's observation of the transverse white bands.[12] Dr. Mees also commented that this phenomenon can have forensic significance. He stated that "a case of polyneuropathy that is difficult to diagnose could then be recognized by the presence of the nail band as polyneuropathy due to acute arsenic intoxication."[12]

In summary, transverse leukonychia caused by arsenic exposure is most commonly referred to as Mees lines. In addition, transverse leukonychia caused by other heavy metals such as selenium and thallium are also designated Mees lines. Also, many investigators use the designation Mees lines to refer other etiologies resulting in transverse leukonychia, such as trauma, hematologic disorders, high altitude, infections (eg, relapsing typhoid fever), skin disease, other medications (including chemotherapy), and systemic disease (including acute rejection of a renal allograft, chronic kidney disease, congestive heart failure, and symptoms associated with the nail flag sign (**Figure 1**), eg, diabetes and leprosy).[8,13-15]

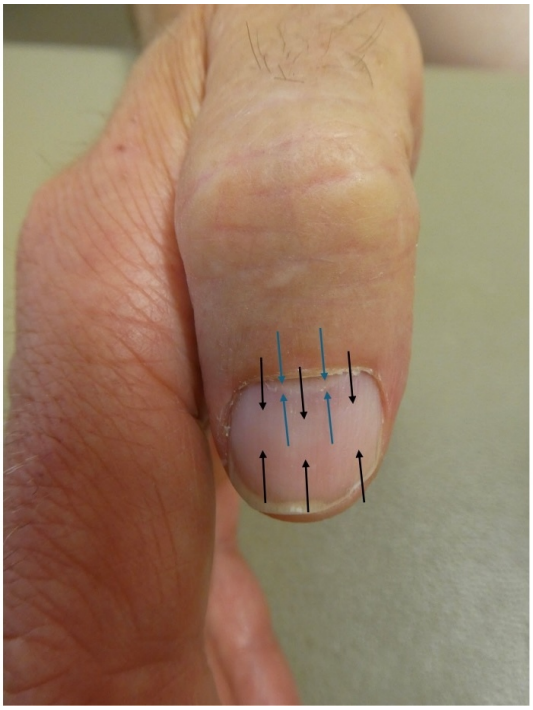

Figure 1. *Mees lines presenting as transverse leukonychia on the thumb of a 70-year-old man. The man has two horizontal white bands on his right thumb. The lunula is absent; beneath and immediately distal to the proximal nail fold and the eponychium (ie, cuticle) is a very narrow red band of nail plate. The proximal Mees line can be seen distal to the band of erythema; the proximal and distal edges of the horizontal white band is demonstrated by the blue arrows. The distal Mees line is the wider white band whose proximal and distal edges are shown by the black arrows. The man has the nail flag sign and a history of being a transplant recipient of a solid organ (ie, heart). This case has previously been reported; however, this figure has not been published before.*[15]

Heavy metals with mucocutaneous manifestations

Heavy metals are defined as those metals which have a density greater than 5g per cubic centimeter. Several heavy metals can cause toxicity. Indeed, dermatologic manifestations, including nail plate anomalies, have been observed following exposure to arsenic, gold, lead, mercury, silver, and thallium. In addition, the metalloid selenium, which has a density of 4.8g per cubic centimeter, is also often considered in this group of heavy metals.[7,16,17]

Toenails are an excellent biomarker of chronic exposure to heavy metals. There are several advantages to using toenails, instead of traditional specimens such as blood and urine, for evaluation of heavy metals. The nail plate specimen can be collected in a noninvasive manner and it is easy to store.[18-20]

In contrast to blood and urine, which can be useful for acute exposure to heavy metals, longer term exposure to the metal can be detected by the presence of the metal when evaluating a nail plate. Approximately four to 6 months of exposure to the metal is necessary before the metal can be detected in the nail plate. Processing of the nail plate to detect the heavy metal most commonly uses neutron activation analysis; other techniques that have been used include inductively coupled plasma mass spectrometry and atomic absorption spectrometry.[18,19]

Heavy metal exposure can result not only in dyschromia but also dystrophy of the nail plate (Table 2).[9-14,21-41] Transverse white bands, also known as Mees lines, are nail-associated changes that can be caused by arsenic, strontium and thallium. However, many of these metals cause diffuse changes in the appearance of the nail plate. In addition, several result in morphologic changes of the skin, mucosa, or both. Illustrative cases which include nail manifestations resulting from heavy metal exposure are described. In addition, other dermatologic features that have been observed in individuals following exposure to heavy metals that can affect the nails are summarized.[13,14,21-41]

Arsenic toxicity can be acute or chronic. Acute arsenic toxicity is characterized by dryness and burning of the upper gastrointestinal tract (including the oral cavity, esophagus, and stomach), abdominal pain, diarrhea, nausea, and protracted vomiting. Muscle spasm, restlessness, vertigo, and delirium or coma may occur; these symptoms can progress to urinary retention, shock, and death.[21-23]

Individuals with chronic arsenic toxicity have peripheral neuropathy, muscle atrophy, and sometimes hematologic abnormalities. Skin manifestations include palmar keratoses and actinic keratoses. Arsenic is also associated with skin cancer such as squamous cell carcinoma in situ. Several types of hyperpigmentation have been observed. These include diffuse macular bronze pigmentation, dark brown spots, or small areas of normal or depigmented skin within the larger areas of hyperpigmentation that have a characteristic rain drop appearance.[21-24]

The most typical dermatologic manifestation of chronic arsenic poisoning is Mees lines. The white band corresponds to the episode of arsenic exposure and typically appears 4 to 6 weeks after acute exposure. The presence of multiple bands suggests repeated attempts of acute exposure.[9-14]

Other nail anomalies associated with arsenic exposure include Beau line (ie, depressed horizontal bands), diffuse blackish-brown nail plate discoloration, longitudinal bands of brown hyperpigmentation, and onychomadesis (ie, proximal shedding of the nail plate).[25] Like nails, hair may also be a deposit for arsenic.[26]

A 16-year-old girl presented with several months of weakness, weight loss, and foot drop; she was admitted to the hospital for evaluation. All her fingernails had a transverse white line (ie, Mees line). She commented that "the lines were growing out with the nail plate."[26] Arsenic was discovered when her hair and nails were analyzed; this prompted a criminal investigation. The girl's father had recently died, and her mother's previous husband had died. Both bodies were exhumed and arsenic was found. The girl's mother disappeared. Subsequently, after several years, the mother was located; she was charged and convicted of murder and attempted murder.[26]

Medicinal gold was previously used in the treatment of arthritis, blistering conditions, lupus erythematosus, and psoriasis. The cutaneous manifestation of gold intoxication is referred to as chrysiasis. It is characterized by a permanent blue-gray discoloration primarily restricted to sun-exposed skin. The dyschromia is most pronounced around the eyes, and there is relative sparing of the skin folds.[24,27]

Yellow discoloration of the nail plate has been observed,[25,27] as has dark brown pigmentation.[25] Other changes in the nail plate have included fragility, slow nail growth, softening, and thinning.[25,27]

A 34-year-old woman with severe rheumatoid arthritis was being treated with intramuscular gold (50mg every two to four weeks) for four years. Two years after starting therapy all her toenails developed gold-yellow discoloration, and the distal two-thirds of all fingernails developed the same discoloration with loss of the lunula. In addition, other nail dystrophies occurred: nail plate thickening and onycholysis developed on both thumbnails and the right little fingernail. The gold therapy was stopped. Within three months, the yellow discoloration had partially grown out. However, after 6 months, light yellow discoloration of all 20 nails persisted.[27]

Lead intoxication, known as plumbism, can result from lead being absorbed from ingestion, inhalation, or absorption via the skin. In children, it can occur from eating lead-based paints. Lead toxicity often presents with symptoms of central neurotoxicity in children.[23,28]

In adults, work-related inhalation is the most common etiology. Occupations at greater risk for lead exposure include battery plant workers, constructions workers, crystal glass makers, firing-range operators, lead miners, metal welders, painters, and shipbuilders. Hypertension is the most common plumbism symptom in adults. Other symptoms include anemia, decreased fertility, gastric colic, muscle and joint pain, peripheral neuropathy, and renal failure.[23,28]

A study of 134 lead-acid battery workers demonstrated several mucocutaneous manifestations of plumbism. Oral changes caused from lead toxicity included gingival brown pigmentation, gingivitis, gum hypertrophy, and the lead line. In contrast to the brown pigmentation, the lead line is bluish in color and found along

Table 2. *Nail Plate Characteristics of Heavy Metal Poisoning*

Metal	CS	Nail pigmentary changes	Other nail features	Other dermatologic features	Ref
Arsenic	As	Diffuse black-brown Longitudinal melanonychia striata[a] Mees lines[b]	Beau lines[c] Onychomadesis[d] Transverse ridges	Crimson soles Erythemata[e] Keratoses: palmar and plantar Pigment changes[f] Puffy skin Wet palms	9-14, 21-26
Gold	Au	Brown (dark) Yellow (golden)	Fragility Onycholysis[g] Slow nail growth Softening Thickening Thinning	Chrysiasis[h]	24,25,27
Lead	Pb	Brown Erythronychia[i] Hyperpigmention (diffuse) Leukonychia (NOS) Leukonychia (punctate) Longitudinal melanonychia striata[a]	Nail bed hyperkeratosis Onychalgia[j] Onychomadesis[d] Pitting Washboard nails[k]	Brown gingiva Gingivitis Gum hypertrophy Lead hue (skin)[l] Lead line (gums)[m]	25,28,29
Mercury	Hg	Brown Dark hyperpigmentation Gray-brown Green-black	Fragility Loss Ridging	Acrodynia[n]: Infants and children Chronic exposure in adults[o]: Blue line (gums)[p] Mercurialitis[q] Metallic taste Skin pigmentation: slate-gray to light brown[r]	24,30-33
Selenium	Se	Erythronychia (proximal)[i] Hyperpigmentation Mees lines Yellow nails	Onycholysis[g] Shedding (complete) Subungual Hyperkeratosis Thickening: distal nail plate	Acneiform papules (scalp) Alopecia Breath odor[s] Blisters Dermatitis Pruritus Seborrhea-like scaling (scalp) Ulcers	34-36
Silver	Ag	Blue lunula Bluish-black	None	Blue-gray to slate-gray pigmentation[t] Feet: ashy-gray thick plaques[u]	24,37-39
Thallium	Tl	Brownish: diffuse or partial Mees lines	Erosions Onychorrhexis[v]	Alopecia	25,40,41

Abbreviations: CS, chemical symbol; NOS, not otherwise specified; Ref, references
[a]Longitudinal melanonychia striata are vertical hyperpigmented bands on the nail plate from the proximal nailfold to the distal tip of the nail.
[b]Mees lines are transverse white bands on the nails. The horizontal white lines can be single or multiple. In Reynolds's original description, the bands were associated with transverse ridges on the nail plate.[9] In patients with selenium exposure, the transverse white bands on the nails are referred to as Mees-like lines.
[c]Beau lines are horizontal ridges or indentations of the nail plate; they represent temporary interruption of nail matrix growth; they can occur secondary to environmental factors, infection, or injury.
[d]Onychomadesis is proximal shedding of the nail plate.
[e]Erythemata referes to acute urticaria, morbilliform rash, scarlatiniform eruptions, and vesicular eruptions.[9]
[f]Pigment changes included not only darkening of preexisting freckles in light complexioned patients, but also diffuse darkening in darker people beginning as an erythematous blush which sequentially progressed from a red to copper-color, then to bronze, and in some cases to black.[9] Also, "frequently the pigmentation shows well-marked lighter spots, like 'rain drops' ... on the face of many patients, yet on the whole it is more marked on the trunk."[9]
[g]Onycholysis is distal nail plate shedding.
[h]Chrysiasis is a permanent blue-gray discoloration of the skin; it is restricted to sun-exposed skin. The distribution of the dyschromia is most prominent in the periocular area; there is relative sparing of the skin fold.

[i]Erythronychia is red nails.
[j]Onychalgia is a painful sensation in the nail.
[k]Washboard nails have nail plates with a central depression; there is parallel, transverse ridging overlying the central area of depression.
[l]Lead hue describes the generalized skin hyperpigmentation that can occur in patients with lead exposure; it is characterized by pallor and lividity.
[m]The lead line is bluish in color; this is in contast to the brown gingival pigmentation that may be observed in patients with plumbism. The blue line is located along the border of the gum.
[n]In infants and young children, mercury exposure results in acrodynia which is also referred to as pink disease. In addition to nail dyschromia, acral sites such as the tips of the fingers and toes and nose become pink or red; other dermatologic features include "puffy, pink, painful, paresthetic, perspiring, and peeling hands."[30] The children often pull their hair out and present with alopecia.
[o]Chronic mercury exposure in adults has three symptomatic stages: a flu-like syndrome, multiorgan symptoms, and neuropsychological manifestations.
[p]A blue line on the gum, along the alveolar margin, associated with mercury exposure is like the blue line observed in patients with lead exposure.
[q]Mercurialitis describes discoloration of the ocular lens.
[r]Skin pigmentation related to mercury is slate-gray to light brown. Importantly, in contrast to the cutaneous hyperpigmentation observed in patient with gold or silver exposure, the hyperpigmentation in adults with chronic mercury exposure is increased in the skin folds such as those of the eyelids, nasolabial folds, and the neck.
[s]Selenium-associated foul breath odor has been described as "garlic odor" or "sour-milk breath."[36]
[t]Argyria-related hyperpigmentation is usually permanent and presents as a slate-gray to blue-gray discoloration; like the hyperpigmentation in patients with chrysiasis, the skin darkening in patients exposed to silver is especially prominent in sun-exposed areas and less prominent in skin folds.
[u]The plaques occurred on the feet of a man with localized cutaneous argyria after a single topical exposure to silver nitrate.
[v]Onychorrehexis is vertical ridging and longitudinal splitting of the nail plate.

the border of the gum. Skin hyperpigmentation can occur; in addition, the lead hue can develop which is a generalized change in skin color that is characterized by pallor and lividity.[28]

Several pigmentary changes of the nail plate were observed. These included brownish discoloration of the nail plate, nail staining from exogenous pigments, erythronychia (ie, red nails), punctate leukonychia, and longitudinal melanonychia (ie, vertical dark bands). Other nail abnormalities were washboard nails (which have a central depression and superimposed parallel and transverse ridging over the depressed area of the nail) and nail pitting.[26] Nail changes observed by other investigators in patients with plumbism include leukonychia, nail bed hyperkeratosis, nail plate proximal shedding (ie, onychomadesis), and painful sensation in the nail (ie, onychalgia).[25]

A 55-day-old boy presented with diffuse hyperpigmentation of his fingernails and toenails of two weeks duration without any systemic symptoms. Since birth, his parents had been applying Tao Dan powder (which has a high lead content) four times daily to his axilla, groin, neck, and perineum. The boy's urine lead level was high. Energy dispersive X-ray microanalysis documented lead in both the powder (14.1mg of lead per gram) and in the dark nail (up to 8.5%). Application of the powder was stopped. Within one month the proximal nail plate returned to normal. During the next 5 months, the dyschromia grew out and did not recur.[29]

Mercury exists in three forms: elemental or metallic, inorganic, and organic. The elemental or metallic form has consequences regarding toxicity. It can be absorbed by inhalation, ingestion, intravenous administration, or percutaneous absorption after skin exposure.[23,30]

Acrodynia (ie, pink disease) refers to mercury toxicity in infants and young children. Systemic symptoms include anorexia, listlessness, and irritability. In addition, diffuse hypotonia, pectoral and pelvic girdle muscle weakness, and photophobia can develop.

In individuals with acrodynia, the palms, soles, and nose become red; the hands can become "puffy, pink, painful, paresthetic, perspiring, and peeling."[30] The patient secretes excessive saliva, loses teeth, and develops oral mucosa ulcers. Hair changes, including alopecia secondary to the child pulling out their hair, and nail changes, such as fragility, ridging, and loss, have been observed.[30]

Mercury toxicity in adults has three symptomatic stages: 1) a flu-like syndrome with chills, dry mouth and throat, headache, fever, and myalgia); 2) multiorgan symptoms within two weeks including the central nervous system, gastrointestinal system (eg, abdominal pain, anorexia, constipation, metallic taste in mouth, nausea and vomiting, and thirst), respiratory system, and urologic system; and 3) neuropsychologic syndrome with depression, diminished self-control, drowsiness, erethism (excitability, irritability), intension tremor, and memory loss that occur over a prolonged period. Mucosal manifestations of mercury toxicity in adults can include a blue line on the gums along the alveolar margin like that seen in lead toxicity; it can also result in discoloration of the ocular lens (ie, mercurialitis). A slate-gray or light brown skin pigmentation can develop which, in contrast to argyria and chrysiasis, is increased in the skin folds such as the eyelids, nasolabial folds, and the neck.[24,30]

Chronic topical exposure to mercury-containing preparations has resulted in nail hyperpigmentation. In one study, symptoms of chronic mercury toxicity (eg, stomatitis, weight loss, hearing and sensory loss, and emotional disturbances), nail dyschromia, and alopecia occurred in patients who were applying cutaneous bleaches containing 5-6% mercury. In addition to the mercury content of the affected nails being extremely high (1720mg per liter), the urinary level of mercury (1.97mg per liter) was about 400 times above the upper limit of normal (0.005mg per liter) after treatment with dimercaprol injection.[31] In another study, 60% of the pa-

tients with scalp psoriasis who were being treated with a topical ointment containing ammoniated mercury (5%) developed brownish discoloration of the distal half or two-thirds of the nail plate of their fingernails. The researchers postulated that patients repeatedly would run their hands through their hair so that they were constantly covered with the mercury-containing ointment. However, the investigators did not evaluate the mercury levels in either the nails, serum, or urine. Therefore, the nail plate dyschromia may have only been caused by the local absorption of the mercury into the nail plate.[32]

A 56-year-old woman who was using a mercury-containing cosmetic cream for a prolonged period developed mild symptoms of chronic mercury intoxication and greenish black dyschromia of the nails. Her nail was evaluated using electron microscopy and energy-dispersive X-ray analysis, and mercury was identified in the nail plate. In addition, her serum and urine also showed high levels of mercury. She was effectively treated with 2,3-Dimercaptopropane-1-sulfonate.[33]

Selenium can be obtained from bread, cereals, eggs, meat, nuts, seafood, and vegetables; it can also be a component of oral supplements. The maximum recommended daily dietary allowance is 55mcg. In addition, topical exposure to selenium can occur from shampoos or lotions that contain selenium sulfide and are used to treat seborrheic dermatitis or tinea versicolor. Acute or chronic ingestion of excess selenium can cause toxicity.[34-36]

Chronic selenium toxicity (ie, selenosis) can present with fatigue, nausea, and vomiting. In addition, tooth decay and mottling and neurologic symptoms (eg, acroparesthesia, hyperreflexia of the tendons, motor disturbances, pain in the extremities, peripheral paresthesia, and weakness) can occur.[34-36] A unique symptom is a foul breath odor that has been described as "garlic odor" or "sour-milk breath."[36] Dermatologic manifestations of selenium toxicity include acneiform scalp papules, alopecia, blisters, dermatitis, nail changes pruritus, seborrhea-like scaling of the scalp, and ulcers.[34-36]

Mees-like transverse bands (either white or grayish-white) have been observed in selenium intoxication.[35,36] Yellow dyschromia[34,35] and proximal red (ie, erythronychia) nail discoloration[34] have also been observed. Complete shedding[35] and distal shedding (ie, onycholysis)[34] of the nail plate has occurred. In addition, subungual hyperkeratosis[34] and distal thickening[35] has been observed.

A 48-year-old woman began taking three capsules a day of a multivitamin complex (Ace plus Se); each capsule contained 20mg (26,000IU) of beta-carotene (provitamin A), 200mg of vitamin C, 200mg (200IU) of vitamin E, and 50mcg of selenium yeast. Two weeks after starting the vitamin, she developed nausea, weakness, and dermatologic symptoms. After four weeks, she developed menstrual dysregulation. She presented with generalized pruritus with itching affecting her body and scalp and scalp hair loss. Fingernail changes included subungual hyperkeratosis, onycholysis, and yellow dyschromia with proximal erythronychia. Her serum selenium level was extremely elevated (1300mcg per liter with an upper limit of normal being 1990mcg per liter). She stopped the vitamin and discontinued eating the "one or two handfuls daily of" nuts she had purchased at an open market. Within 6 months, the hair loss had stopped, and density had increased. The yellowish discoloration, subungual hyperkeratosis, and onycholysis were dramatically decreased. However, slight thickening and yellow discoloration of the nails persisted.[34]

Silver toxicity can result from topical skin contact (ie, localized cutaneous argyria) or following systemic absorption (ie, generalized argyria) from inhalation, ingestion, or contact with mucosal membranes (eg, nose drops and pharyngeal application). Systemic symptoms do not occur.[24,37-39]

The manifestations of silver exposure are dyschromia that can affect the skin, sclera, mucous membranes, and nails. The skin, especially in sun-exposed areas, develops a generalized slate-gray or blue-gray discoloration; the dyschromia is less prominent in skin folds. The acquired pigmentation is usually permanent.[24,37-39]

The entire nail is often affected. The nail plate appears either slate-gray or bluish-black. However, pigmentation can be restricted to the lunula and can be blue in color.[24, 37-39]

A 34-year-old man experienced a single episode of topical silver nitrate exposure in a work-related accident; the solution leaked onto his shoes and came in direct contact with his feet. He attempted to wash away the solution with water but three hours later his feet itched and burned. The feet developed ashy-gray thick plaques, and all his toenails were bluish-black. He was diagnosed with localized cutaneous argyria. Some of the plaques had peeled away after 15 days. However, the dyschromia of the skin and nails persisted.[38]

A 69-year-old woman presented with slate-gray discoloration of her face and dorsal hands of two-years duration; all her fingernails were slate-gray. For more than 5 years an otolaryngologist had treated her pharyngitis with topical silver protein (ie, Protein Gin) that was applied to her pharynx 5 times a week. Skin biopsy of her affected dorsal hand showed increased melanin granules in the basal layer keratinocytes and black particles in the dermis between the collagen bundles and around the skin appendages (eg, the eccrine sweat glands). Electron microscopy confirmed the presence of electron-dense granules and X-ray microanalysis established that the granules contained silver, selenium, and sulphur; a high peak of silver was seen. She was diagnosed with generalized argyria. Avoidance of sunlight was recommended and "she improved fairly."[39]

Thallium toxicity is always accompanied by alopecia; the hair loss begins withing two weeks after exposure and is usually complete within four weeks. Other symptoms of acute thallium exposure include disorientation, hypertension, hypotension, gastroenteritis, polyneuropathy, psychiatric disorders, and tachycardia.[23,40,41]

Nail manifestations of thallium toxicity include transverse leukonychia (ie, Mees lines).[40,41] Other dyschromia of the nail plate can also appear such as partial or diffuse brownish discoloration.[25] Vertical ridges and longitudinal splitting of the nail plate (ie, onychorrhexis) and erosions can develop.[25]

A 37-year-old man presented to the hospital with pain in the abdomen, chest, and lower limbs; he was treated for peripheral polyneuropathy with B complex therapy. Severe alopecia occurred after 20 days. Mees lines appeared after 27 days. Both his serum and urine thallium levels were elevated. He had intentionally drunk water mixed with thallium. He received 5 treatments with hemoperfusion and hemodialysis starting on day 21; over the next week, he began to improve. Eventually all his symptoms resolved, except for residual dysesthesia.[41]

Nail evaluation at a crime scene as a possible indication of heavy metal exposure by the victim

A crime scene can be investigated by several individuals. These can include police officers, detectives, and forensic investigators, and each of these individuals prepares a unique report of the event.[42-45] Heavy metal exposure may not result in death. Therefore, all individuals who investigate crimes might benefit from being aware that a victim's nails can provide a clue to unsuspected heavy metal toxicity.

In the case where a death has occurred, a death investigator may evaluate the scene.[46,47] The responsibility of the crime scene death investigator is to analyze the scene and the decedent. In contrast to the forensic pathologist who examines not only the external body but also the internal organs of the decedent, the crime scene death investigator focuses on the external examination of the decedent at the scene. Part of the death investigator's evaluation is a careful examination of the exposed areas of the body for scars and tattoos. This examination also includes the scalp for alopecia and the hands and the fingernails.[46,47]

An astute death investigator might notice transverse leukonychia in a decedent who has been intoxicated by arsenic, selenium, or thallium.[9-14,21-26,34-36,40,41] The investigator might not only also see blue discoloration of the lunula in a decedent with silver poisoning but also the slate-gray dyschromia of the sun-exposed face and dorsal hands.[24,37-39] Similarly, the investigator might see both the dark nails and the blue-gray discoloration of the sun-exposed skin in a decedent who had gold toxicity.[24, 25,27]

The forensic pathologist begins the autopsy with a meticulous examination of the external body.[48] A thorough examination of the skin, hair, and nails is performed. Examination of the body surface frequently provides the pathologist with the cause, the manner, and the mechanism of death.[48]

Nail changes consistent with exposure to heavy metals would prompt the coroner or medical examiner to examine the skin and mucous membranes for heavy metal toxicity-related findings. The forensic pathologist would submit the nail plate for evaluation for the presence and concentration of the associated heavy metal. In addition, the nail plate observation would prompt the pathologist to consider evaluation of other body fluids or tissues (eg, skin and hair) to confirm the identity of the suspected associated heavy metal.[26]

Conclusion

Fingernails and toenails can be an important source of trace evidence at a crime scene investigation. Heavy metal exposure to arsenic, gold, lead, mercury, selenium, silver, and thallium can result in nail plate changes. Transverse leukonychia (ie, Mees lines) of the nails was originally described in individuals with arsenic exposure; the horizontal white bands have also been observed following exposure to selenium and thallium. In addition to other nail plate discoloration and morphology changes, additional dermatologic manifestations (including pigmentary alteration) have been reported after heavy metal exposure. Evaluation of the nails in a crime scene investigation is important. Indeed, the nails can provide a clue to unsuspected heavy metal exposure. When the possibility of heavy metal toxicity is entertained, the nails can be photographed and subsequently analyzed for the presence of a heavy metal.

Potential conflicts of interest

The authors declare no conflicts of interest.

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