WORLD JOURNAL OF PHARMACEUTICAL RESEARCH
SJIF Impact Factor 8.084
Volume 10, Issue 2, 1275-1286. Review Article ISSN 2277– 7105

VIBRATION WHITE FINGER- AN OCCUPATIONAL HAZARD

Manisha Sinha[1] and Nalini M. S.*[2]

[1]Post Graduate, Department of Periodontology, Rajarajeswari Dental College and Hospital, Bangalore.

[2]Professor, Department of Periodontology, Rajarajeswari Dental College and Hospital.

Article Received on
18 Dec. 2020,

Revised on 08 Jan. 2021,
Accepted on 28 Jan. 2021

DOI: 10.20959/wjpr20212-19746

***Corresponding Author**
Dr. Nalini MS
Professor, Department of Periodontology, Rajarajeswari Dental College and Hospital.
drnalinims79@gmail.com,

ABSTRACT

This review aims to briefly discuss the pathophysiology and diagnostic tests for the vibration induced white finger assuming its multifactorial etiology. The vibration exposure in dentistry is a possible risk in developing various vibration induced symptoms like vascular, neurological, muscular and skeletal symptoms or disorders. It could occur due to neural dysfunction, local acral vasodysregulation, shear stresses, blood viscosity and cell activation. The best available method of diagnosing Raynaud's phenomenon (RP) and vibration white finger (VWF) is a detailed medical interview by the clinician. Hand examination includes finger skin temperature, hand grip strength, pinch strength, finger vibrotactile perception threshold, cold water immersion test and nailfold capillaroscopy Regular medical check-ups should be carried out, with special attention paid to the vascular, neural and osseous systems of the upper limbs care must be taken to limit the exposure for vibration.

KEYWORDS: Vibration white finger, white finger syndrome, finger skin temperature.

Clinical relevance

- Dentists can be at a potential risk of developing vibration induced vascular changes.
- Hence, better knowledge and understanding of the symptoms can help diagnose disease at an early stage.
- It can help the clinicians to improve their clinical effectiveness in routine dental practice.

INTRODUCTION

Work related vibration is a potential health hazard and it results from transfer of mechanical vibration from the instrument to the body.[1] This vibration exposure in dentistry is a possible risk in developing various vibration induced symptoms like vascular, neurological, muscular and skeletal symptoms or disorders.[2] These symptoms can be recognised by the intensity of vibrations, the range of frequency, direction, type, point of penetration, time and kind of daily exposure as well as the total time of exposure. The vascular symptoms are referred as white fingers—an intermittent blanching of the fingers. The neurological symptoms include intermittent tingling, pins-and-needles sensations, paresthesiae and numbness (Griffin, 1990).[2] Paroxysmal circulatory disturbances in the fingertips are described as Raynaud's phenomenon, "dead fingers" or the "white finger syndrome."[1] The use of vibrating tools and high-force and repetitive hand–wrist movements are important risk factors for the development of other symptoms like pain, paresthesiae and numbness (Cannon et al., 1981); muscular weakness, decreased grip strength and symptoms in the wrists, elbows, neck, shoulders and back may also occur (Färkkilä, 1978); The symptoms may lead to a reduction in the quality of life and the ability to cope with job tasks and leisure activities.

This type of high frequency energy is absorbed by large parts of superficial tissues and tissue may lead to diagnosing an occupational disease called the vibration syndrome. Among tissue structures that can be affected in this case are the microscopic mechanoreceptors in the skin of the palm. It is known that there are four types of mechanoreceptors which are sensitive to mechanical skin displacement, among which the Pacinian corpuscle is the most sensitive (Vallbo, Johansson, 1978; Johansson, et. a1. 1982).[4] This corpuscle detects very small skin displacements, in the magnitude of one micron, especially in the frequency range of 200-300 Hz. It is presumed that the damaging effect of local vibrations occurs within the range 5-1400 Hz, the most harmful being low-frequency vibrations, i.e. those below 16 Hz. Also, the history of certain diseases, injuries of the upper limbs, drugs used, smoking and alcohol intake affects the course of this disease. Other contributing factor enhancing the syndrome is the inadequate microclimatic conditions such as low temperature, high humidity and intense movement of the air, all of which tend to refrigerate the hands.

Symptoms of VWF mostly arise after many years of regular occupational exposure to hand-transmitted vibration. Experimental studies in healthy people show that vibration of one hand provokes digital vasoconstriction, not only in the exposed hand but also in fingers of the non-

vibrated hand (e.g. Bovenzi et al. 1998, 1999, 2000; Griffin et al. 2006; Thompson and Griffin 2009).[5,23] Among the dentists, the main source of the vibration are the vibrating power-driven or air-driven instruments, such as low- and high-speed handpieces as well as ultrasonic instruments. The vibrations emitted by these machines travel directly from the handles to the operator's hand eventually leading to the development of vibration syndrome in the long term.

This review aims to briefly understand the pathophysiology and diagnostic tests for the vibration induced white finger.

Pathophysiology of vibration-induced raynaud's phenomenon[6]

Assuming its multifactorial etiology, the pathophysiology involves various components as described below.

1. Neural dysfunction

1.1. Autonomic dysbalance

Due to an overstimulation of pacinian corpuscles it leads to increase in sympathetic activity and/or parasympathetic depression. Increased vascular peripheral resistance and hypothermy, changed skin vasomotor and sudomotor reactivity demonstrate this statement. Long-lasting vibration exposure may provoke central sympathetic vasoconstrictor reflex mechanisms, which trigger primarily episodic arterial closure typical of VRP.

1.2. Receptor and Nerve ending dysfunction

Hand–arm vibration causes mechanical damage to blood vessels and vasoregulatory nerve elements as it is absorbed by skin structures. The affinity of efferent receptors becomes extreme to vasoactive substances potentiated by local cooling which suggested for the increase of the peripheral resistance of finger circulation in HAVS. Receptors and/or pain-mediating nerve fibers and sympathetic vasoconstrictor nerves or receptors in fingers are affected by hand–arm vibration.

Peripheral sensory, motor, pain-mediating nerve fibers, temperature nerve-endings, slowly adapting type I (SAI) and fast-adapting types I and II (FAI and FAII) receptors at the fingertips are disturbed due to vibration. Vibration damages local nerve endings leading to general neuronal loss, especially in those digital cutaneous perivascular nerves containing the neuropeptide with powerful vasodilator properties, i.e. calcitonin gene-related peptide (CGRP).

2. Local acral vasodysregulation

Various degrees and forms of endothelial dysfunction may derive as a result of hand–arm vibration exposure. Endothelial damage was supported by the elevated plasma level of thrombomodulin established in vibration exposed workers and in HAVS patients with and without VRP; elevated plasma levels of fibronectin in HAVS patients.

2.1. Endothelial dysregulation

Significantly lower endothelin level was found in workers exposed to vibration possibly due to a local adaptive axon-reflex resulting in vasodilation. But in HAVS patients the concentrations of endothelin-1 is found to be high, the highest being in most advanced stages. Increased production of endothelin inhibits the direct myorelaxing effects of NO on vascular smooth muscle. Furthermore, the imbalance between endothelin-1 and CGRP also contributes to the vasospastic phenomenon. The absence or dysfunction of endothelium in HAVS patients favors the occurrence of vasospasm. Existing oxidative stress in VRP also contributes to vasospastic paroxysms. Reduced blood flow responses to sodium nitroprusside (endothelium-independent vasodilator) found in VRP patients probably reflects disturbed smooth muscle response to NO.

3. Shear stresses

Patients with VRP have experienced both mechanical trauma and shear stress enough to induce endothelial damage, swelling, and even endothelial cell loss resulting in endothelial dysfunction. At the sites of arterial stenosis leukocytes and platelets are exposed to transient but extremely high shear stresses, which cause activation of these cells in vitro.

4. Blood viscosity and cell activation

4.1. Erythrocyte activation

Erythrocyte (RBC) aggregation significantly influences microcirculatory blood flow rate. An acute vibration exposure increases capillary permeability probably as a result of vibration-induced endothelial damage and causes hemoconcentration with increases in RBC count, hemoglobin concentration and plasma viscosity. It is also associated with RBC hypo deformability and higher number of degenerative and reversibly changed RBC (echinocytes and stomatocytes) found in HAVS patients. Possible mechanism could be the low oxygen partial pressure in the hands of HAVS patients and the pH reduction in their hand venous blood.

4.2. Platelet activation

Intravascular platelet aggregation is described probably as a result of vibration-induced endothelial damage. It may lead to release of mitogenic mediators as platelet-derived growth factor causing focal proliferation of smooth muscle cells found in VRP and of vasoconstricting thromboxane A (TxA) whose metabolite thromboxane B is a potent vasoconstrictor and increased thromboxane B level is described in VRP. Platelets release serotonin when they aggregate and may further induce digital vasospasm. But other data show that vibration-induced vascular injury does not seem to provide a sure stimulus sufficient to induce persistent platelet activation.

4.3. Leukocyte activation

Leukocytes (WBCs), especially polymorphonuclear leukocytes (PMNs), pass with relative difficulty through small capillaries even under normal condition. By forming rigid structures, activated or abnormal leukocytes can impair blood flow within the microcirculation aggregating to each other and adhering to the vascular wall as found in VRP patients who already have a tendency for vasospasm and thereby enhance tissue ischemia. A subpopulation of hard and poorly deformable PMNs was found in VRP patients. Because of the trapping of the less deformable PMN within the microcirculation, a significant fall in single PMN count in venous blood was revealed in VRP. But acute hand-transmitted vibration had no in vitro effect on leukocyte rheology.

Leukocytes become more adhesive after shear stress exposure. Leukotrienes, chemotactic factors and cytokines released from the damaged and ischemic endothelium can further activate the leukocytes against a background of reduced prostacyclin and NO production. These mediators also can upregulate the expression of adhesion molecules on endothelial surfaces and on PMN cells. The increased levels of leukotriene B4 and free radical production support the leukocyte activation in VRP, which may contribute to the microvascular damage. In summary, WBCs may play some role in the pathogenesis of microvascular disorders and tissue ischemia in VRP; however, it is not elucidated if this is a cause or effect of the disease.

Patients with HAVS are found to have elevated soluble intercellular adhesion molecule-1 (sICAM-1) levels involved in the adherence of WBCs to endothelium and to other WBCs, and thus decreasing blood flow in microcirculation. Significantly lower cytokine interleukin-8 (IL-8) levels are also found in HAVS patients. The impeded flow of blood cells through the

microcirculation may result in these low levels of circulating inflammatory cytokine due to its binding to erythrocytes.

Diagnostic tests

Medical Interview and Questionnaire

The best available method of diagnosing RP and VWF is a detailed medical interview by the clinician and is most often used as a method of reference. The interview includes passive or neutral questions concerning presence or absence of any possible finger symptoms, its onset, duration and the subjects themselves should describe their symptoms. The physician has to then assess if the patient has convincing history of RP.[7]

The minimum requisites for the diagnosis RP and VWF have been proposed as follows by a working group (Oslen et al 1995)

a) RP; cold-provoked episodes of well- demarcated blanching (whiteness) in one or more fingers;
b) VWF; first appearance of RP after start of professional exposure to hand-arm vibration and no other probable causes of RP.
c) Current activity of VWF; currently active VWF if episodes have been noticed during the past 2 years.

However, the diagnostic signs of finger colour have been observed by cold-provocation tests in subjects without RP, in subjects judged to cease RP, and in anamnestically non-affected phalanges in subjects with VWF.

Hand examination

The special hand examination includes measurement of finger skin temperature, finger nail capillary return time, finger vibrotactile perception threshold, hand grip strength and pinch strength and a cold water immersion test.

1. Finger skin temperature[8,9]

The finger skin temperature can be measured over the pulp of the index, middle, ring and little fingers using an infrared thermometer. The finger nail capillary return is measured for the index, middle, ring and little fingers after pressing the nail bed gently for 10 seconds.

2. Hand grip strength[8,10]

The hand grip strength can be obtained using a digital dynamometer in a standing position with both arms in a neutral position, the elbows extended and the forearms pronated at 90°. The patient is asked to grip the dynamometer as strong as possible using one hand followed by the other hand, and the procedure is repeated for 5 times.

3. Pinch strength[11,16]

The pinch strength between the thumb and index, middle and ring fingers is measured respectively using a pinch by asking the patient to pinch the gauge using the thumb pad and distal phalanx of each finger as strong as possible while preventing other fingers from assisting in the pinching effort.

4. Finger vibrotactile perception threshold[12,13]

The vibrotactile perception threshold can be measured for the index, middle, ring and little fingers using a vibration sensation meter with a vibration frequency setting of 125 Hz. During the test, the patient is asked to touch the vibrator probe (without a surround platform) with the pulp of the finger, while the other fingers, hand, wrist and arm were positioned to prevent contact with the surface of the vibration sensation meter and examination table.

The vibration level is then increased gradually at an interval of 2.5 dB from the starting intensity of −10 dB. The patient is instructed to lift up the other hand immediately when he felt the vibration sensation. The procedure is repeated three times, and the vibration intensity level with at least two out of three similar responses is considered as the correct vibration perception threshold. The interview and all test procedures are carried out in the same session. The room temperature for the examination is maintained at 21−23°C, there is an adaptation period of 30 minutes, and a hand warming procedure is performed prior to the physical examination.

Cold water immersion test[8,14]

The method generally used for the evaluation of VWF is to measure the skin temperature of the fingers of the hand immersed up to the wrist in cold water at 5 or 10°C.The cold water immersion test is adopted in the examination using a water temperature of 5°C and a one minute immersion duration. The cold water provocation test is carried out to evaluate the change in the response to skin temperature measurement and vibration perception threshold following immersion of the dominant/symptomatic/worst affected hand in the water at 5°C

for one minute.

A K-type thermistor wire temperature probe is attached to the middle finger of the test hand and masked with a paper tape. The test is carried out after an adaptation period of 30 minutes, with the subject in a sitting and relaxed posture and both hands at heart height during the settling and recovery period with the wrist in a neutral position.

The test hand is immersed in the cold water up to the wrist level. The immersed hand is gently wiped dry immediately after the cold provocation to prevent heat loss from evaporation. The skin temperature is monitored at zero, one, two and three minutes before immersion, 15, 30, 45 and 60 seconds during immersion and at every minute after immersion until 10 minutes post immersion.

The vibration perception threshold is measured at the index finger immediately post immersion and at 5 minutes and 10 minutes post immersion. Throughout the test, the water temperature is maintained at 5°C +/− 0.5°C and the room temperature is maintained between 21−23°C. Hand movement during the measurement of VPT should be kept to a minimum by keeping the wrist in a neutral position throughout the test.

Several unfavorable symptoms such as chest discomfort, headache, nausea and pain during the cold-water immersion test have been reported (Chang 1976).

Nailfold capillaroscopy[15]

Evaluating the structure of nailfold capillaries by means of nailfold capillaroscopy can be useful in diagnosing vibration white finger. It is typically hairpin in shape and are mostly parallel to the skin surface. Nailfold capillary abnormalities associated with HAVS can be recognized by structural alterations, such as degeneration of capillary density, avascular areas, appearance of enlarged capillaries, local haemorrhages and angiogenesis. Cutolo et al began their studies on capillaries, eventually detailing a capillaroscopy procedure and suggesting a set of parameters which should be taken into consideration, including the presence of enlarged and giant capillaries, haemorrhages, loss of capillaries, disorganisation of the vascular array, and ramified/bushy capillaries.

Treatment

In the progression of the day's work the dentist should notice the principle that periods of exposure to mechanical vibrations alternate with periods of harmless actions. Regular

medical check-ups should be carried out, with special attention paid to the vascular, neural and osseous systems of the upper limbs. It frequently happens that a simple examination, if carried out at the right time, leads to a timely discovery of changes caused by exposure to vibration. Also, care must be taken while purchasing new equipment to ensure that it does not emit greater noise and vibration than the old machinery.[1]

The treatment of VRP is nonspecific. More or less, drugs beneficial could be

1. Calcium-channel blockers (nifedipine, felodipine, isradipine, diltiazem, nicardipin) which can act with peripheral vasodilating, membrane-protecting and antioxidant effects;
2. Postsynaptic blockers of a-adrenoceptors with vasodilatory and hemorrheological effects (prazosin),
3. Sympatholitic drugs with microcirculatory effect (buflomedil, thymoxamine, guanethidine),
4. Prostacyclin analogues with vasodilative and platelete-antiaggregation effects (iloprost, beraprost);
5. Selective serotonin reuptake inhibitors and serotoninergic receptor antagonists (fluoxetine, ketan-serine),
6. Simple vasodilators (pentoxifylline) including topical ones (hexyl nicotinate, glyceryl trinitrate, nitric-ox-idegenerating gel);
7. Angiotensin-converting enzyme inhibitors with potent vasodilator effect (captopril);
8. Fibrinolytic drugs (stanazolol) affirm the involvement of multiple pathophysiological mechanisms in VRP.[6]

REFERENCES

1. Szymańska J. Dentist's hand symptoms and high-frequency vibration. Ann Agric Environ Med, 2001; 8: 7-10.
2. Bylund SH, Burström L, Knutsson A. A descriptive study of women injured by hand–arm vibration. Annals of occupational hygiene, 2002; 1, 46(3): 299-307.
3. Chowdhry R, Sethi V. Hand arm vibration syndrome in dentistry: A review. Current Medicine Research and Practice, 2017; 1, 7(6): 235-9.
4. Lundström R, Lindmark A. Effects of local vibration on tactile perception in the hands of dentists. Journal of Low Frequency Noise, Vibration and Active Control, 1982; 1(1): 1-1.

5. Ye Y, Mauro M, Bovenzi M, Griffin MJ. Reduction in finger blood flow induced by hand-transmitted vibration: effect of hand elevation. International archives of occupational and environmental health, 2015; 1, 88(7): 981-92.
6. Stoyneva Z, Lyapina M, Tzvetkov D, Vodenicharov E. Current pathophysiological views on vibration-induced Raynaud's phenomenon. Cardiovascular research, 2003; 1, 57(3): 615-24.
7. Olsen N. Diagnostic aspects of vibration-induced white finger. International archives of occupational and environmental health, 2002; 1, 75(1-2): 6-13.
8. Su AT, Fukumoto J, Darus A, Hoe VC, Miyai N, Isahak M, Takemura S, Bulgiba A, Yoshimasu K, Maeda S, Miyashita K. A comparison of hand-arm vibration syndrome between Malaysian and Japanese workers. Journal of occupational health, 2013; 13-0059.
9. Ye Y, Griffin MJ. Effect of room temperature on tests for diagnosing vibration-induced white finger: finger rewarming times and finger systolic blood pressures. International archives of occupational and environmental health, 2017; 1, 90(6): 527-38.
10. De S, Sengupta P, Maity P, Pal A, Dhara PC. Effect of body posture on hand grip strength in adult Bengalee population. Journal of Exercise Science and Physiotherapy, 2011; 7(2): 79.
11. Su AT, Maeda S, Fukumoto J, Darus A, Hoe VC, Miyai N, Isahak M, Takemura S, Bulgiba A, Yoshimasu K, Miyashita K. Dose–response relationship between hand-transmitted vibration and hand-arm vibration syndrome in a tropical environment. Occup Environ Med, 2013; 1, 70(7): 498-504.
12. Harazin B, Harazin-Lechowska A, Kalamarz J, Zielinski G. Measurements of vibrotactile perception thresholds at the fingertips in Poland. Industrial health, 2005; 43(3): 535-41.
13. Ye Y, Griffin MJ. Assessment of thermotactile and vibrotactile thresholds for detecting sensorineural components of the hand–arm vibration syndrome (HAVS). International archives of occupational and environmental health, 2018; 1, 91(1): 35-45.
14. Xiao B, Zhang D, Yan M, Qu H, Wen W, Zhang X, Lin H, Ye Y, Chen T, Chen Q. Cold water immersion test (10° C, 10 min) for diagnosing vibration-induced white finger among a group of polishers in a subtropical environment. International archives of occupational and environmental health, 2019; 1, 92(6): 865-72.
15. Chen Q, Chen G, Xiao B, Lin H, Qu H, Zhang D, Shi M, Lang L, Yang B, Yan M. Nailfold capillary morphological characteristics of hand-arm vibration syndrome: a cross-sectional study. BMJ open, 2016; 1: 6(11).

16. Abbas SU. Pinch Force and Workrelated Musculoskeletal Disorders in Dental Professionals.
17. Inaba R, Mirbod SM, Iwata H. Pathophysiology of Vibration-Induced White Finger and Safety Levels for Hand-Transmitted Vibration. Journal of Occupational Health, 1996; 38(1): 1-5.
18. Nasu Y, Kurozawa Y, Fujiwara Y, Honma H, Yanai T, Kido K, Ikeda T. Multicenter study on finger systolic blood pressure test for diagnosis of vibration-induced white finger. International archives of occupational and environmental health, 2008; 1, 81(5): 639-44.
19. Sauni R, Pääkkönen R, Virtema P, Toppila E, Uitti J. Dose–response relationship between exposure to hand-arm vibration and health effects among metalworkers. Annals of occupational hygiene, 2009; 1, 53(1): 55-62.
20. Hua Y, Lemerle P, Ganghoffer JF. A two scale modeling and computational framework for vibration-induced Raynaud syndrome. Journal of the mechanical behavior of biomedical materials, 2017; 1(71): 320-8.
21. Aarhus L, Stranden E, Nordby KC, Einarsdottir E, Olsen R, Ruud B, Bast-Pettersen R. Vascular component of hand-arm vibration syndrome: a 22-year follow-up study. Occupational Medicine, 2018; 11, 68(6): 384-90.
22. Shen SC, House RA. Hand-arm vibration syndrome: What family physicians should know. Canadian Family Physician, 2017; 1, 63(3): 206-10.
23. Ye Y, Mauro M, Bovenzi M, Griffin MJ. Association between vasoconstriction during and following exposure to hand-transmitted vibration. International archives of occupational and environmental health, 2014; 1, 87(1): 41-9.
24. Rytkönen E. High-frequency vibration and noise in dentistry. University of Kuopio, 2005.
25. La Rochelle NR. Work-related musculoskeletal disorders among dentists and orthodontists.
26. Poole CJ, Bovenzi M, Nilsson T, Lawson IJ, House R, Thompson A, Youakim S. International consensus criteria for diagnosing and staging hand–arm vibration syndrome. International archives of occupational and environmental health, 2019; 3, 92(1): 117-27.
27. Haghighi AB, Khosropanah H, Vahidnia F, Esmailzadeh S, Emami Z. Association of dental practice as a risk factor in the development of carpal tunnel syndrome. Journal of Dentistry, 2013; 14(1): 37.
28. Mansfield NJ. The European vibration directive–how will it affect the dental profession?. British dental journal, 2005; 199(9): 575-7.

29. Sauni R, Toivio P, Pääkkönen R, Malmström J, Uitti J. Work disability after diagnosis of hand-arm vibration syndrome. International archives of occupational and environmental health, 2015; 1, 88(8): 1061-8.
30. Bovenzi M, Pinto I, Picciolo F. Risk assessment of vascular disorders by a supplementary hand–arm vascular weighting of hand-transmitted vibration. International archives of occupational and environmental health, 2019; 3, 92(1): 129-39.