Possible Photobiomodulation on Swine Flu

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Possible prophylaxis and rehabilitation effects of intranasal low intensity laser therapy on influenza A (H1N1)

Abstract

Intranasal cells and tissues play an important role in the generation of local immunity to influenza infections. It has been found that chilling caused a pronounced constriction of the blood vessels in the nose so that the onset possibility of common cold symptoms increased due to chilling-induced immunosuppression, and hot drink provided immediate and sustained relief from symptoms of common cold and flu. Intranasal low intensity laser irradiation may rehabilitate intranasal cells and tissues. Many phenomena and the mechanism of intranasal low intensity laser therapy (ILILT) have been integrated to support the prophylaxis and rehabilitation effects of ILILT on the new influenza A (H1N1) (once named as swine flu) in this paper.

Key words: flu, Influenza A (H1N1), photobiomodulation, nose, laser

1 Introduction

Influenza is an acute viral disease which mainly affects the respiratory tract and occurs in all age groups with yearly epidemics during the cold season. In the United States, seasonal influenza epidemics account for > 200,000 hospitalizations and > 30,000 deaths annually. More than 90% of the deaths are in the elderly. A basic method of protecting the population against influenza, which is also the cheapest, is vaccination of as many people in the population as possible, especially those high-risk patients, but it was susceptible to failure resulting from antigenic changes. Moreover, seemingly, from one influenza season to the next, the efficacy of the leading antiviral influenza drug has been lost because of resistance. The use of laser acupuncture has been found to considerably decrease the severity of infection in acute experimental influenza infection (Savtsova et al 1990). Intranasal low intensity laser therapy (ILILT) (Liu et al 2009) is very extremely safe. Its possible prophylaxis and rehabilitation effects of on the new influenza A (H1N1) (once named as swine flu) (Centers for Disease Control and Prevention 2009) was discussed in this paper.

2 Flu pathology

The common cold is a mild self-limiting illness usually confined to the upper respiratory tract (Heikkinen et al 2003). The disease is self-diagnosed from a range of symptoms such as nasal stuffiness, sneezing, throat irritation and mild fever. Johnson et al (2005) found acute chilling of the feet causes the onset of common cold symptoms in around 10% of the chilled subjects. Chilling of the feet in cold water (12°C ± 1°C) has been previously reported to cause an intense vasoconstriction of both the cutaneous and upper airway blood vessels (Drettner 1961) and the vasoconstriction of the upper airways has been proposed as a mechanism that reduces respiratory defence against infection (Mudd et al 1919, Eccles 2002) due to stress-induced immunosuppression (Wheway et al 2005). Chilling causes a pronounced constriction of the blood vessels in the nose and shuts off the warm blood that supplies the white cells that fight infection. When common cold viruses are circulating in the community, a proportion of subjects will have sub-clinical infections, and chilling of these subjects may cause vasoconstriction in the upper airway epithelium and conversion of a sub-clinical to a clinical infection. In these cases, the subject links the causality of the common cold symptoms to the chill and does not realize that they were already infected before they 'caught' a cold. This might be one of the causes why the individuals such as people over 65 years or under 2 years old, and individuals with chronic cardiovascular, pulmonary or renal disease, diabetes mellitus or immunosuppression (Schmidt 2004) are under high risk of flu infection.

Tamura et al (1996) found that nasal Th1 cells, capable of producing the type I interferon (IFN)-gamma and mediating delayed-type hypersensitivity, a protective localized cell-mediated immune response against intracellular pathogens primarily, are involved in the type-specific acceleration of recovery from influenza after challenge in mice immunized intranasally with adjuvant-combined nucleoprotein, although the nonspecific mechanism of accelerated recovery remains to be solved. IgA is the major, if not the sole,

mediator of nasal immunity to influenza virus in immunocompetent mice (Renegar et al 1991). Tamura et al (1998) also found that virus-specific IgA antibodies, produced by IgA antibody-forming cells in the nasal-associated lymphoid tissue (NALT), play an important role in recovery from infection. Therefore, NALT plays a role in the generation of local immunity to influenza infections although it is not essential for the development of protective immunity and viral clearance in the upper respiratory tract (URT) (Wiley et al 2005).

Influenza causes a broad range of illness, from symptomless infection to various respiratory syndromes and disorders affecting the heart, brain, and other organs, to fulminant primary viral and secondary bacterial pneumonia. During influenza epidemics, hospitalizations for stroke and cardiac diseases increase, and more than half of the excess mortality during such epidemics was attributed to causes other than influenza, including cardiovascular diseases and stroke (Grau et al 2005). Bogomol'tsev et al (2003) have studied microcirculation, hemocoagulation and blood viscosity in 377 of 1033 inpatients with influenza and other acute respiratory viral infections. They found that the microcirculatory changes manifest themselves with advanced erythrocyte aggregation, activation of vascular-platelet and plasmic links of hemostasis, and high blood viscosity at low shift velocities. In the presence of concomitant pathology (ischemic heart disease, hypertensive disease, diabetes mellitus) and development of complications, especially acute pneumonia, these disturbances are still greater and tend to increase to the period of decline of clinical and toxic manifestations.

The incidence of upper respiratory infections (URIs), 38% of which are due to influenza, peaks in winter (November and December). On the other hand, both acute myocardial infarction (AMI) and stroke also have their peak incidence in winter months (Meyers 2003). AMI and atherothrombotic stroke share a common pathogenesis involving disrupted atherosclerotic plaque and intravascular thrombosis. URIs result in many biochemical, cellular, and hemostatic changes that could predispose to plaque disruption and thrombosis. Infections, particularly URIs, frequently precede AMI and stroke. Up to 16% of persons older than 60 years of age experience a URI each year. Nineteen percent of those suffering an AMI recall a URI in the 2 weeks prior to their event. Three epidemiologic and one small clinical trial suggest that influenza vaccination is associated with a 50% reduction in incidence of sudden cardiac death, AMI, and ischemic stroke. Influenza vaccine is extremely safe and has a 50% efficacy.

3 Flu prophylaxis and rehabilitation

IFN response represents one of the first lines of defense against influenza virus infections. Kugel et al (2009) have assessed the protective potential of exogenous IFN-alpha against seasonal and highly pathogenic influenza viruses in ferrets. Intranasal treatment with IFN-alpha several hours before infection with the H1N1 influenza A virus strain A/USSR/90/77 reduced viral titers in nasal washes at least 100-fold compared to mock-treated controls. IFN-treated animals developed only mild and transient respiratory symptoms, and the characteristic fever peak seen in mock-treated ferrets 2 days after infection was not observed. Repeated application of IFN-alpha substantially increased the protective effect of the cytokine treatment.

Fujisawa et al (1987) found that polymorphonuclear leukocytes (PMNs) (X-ray-sensitive, carrageenan-resistant) were the cells primarily responsible for early protection in influenza virus infection and that after infection with a high dose of the virus alveolar macrophages (X-ray-resistant, carrageenan-sensitive) also played a protective role in the early phase. Brokstad et al (2001) further found that the basal level of influenza-specific antibody-secreting cells in the mucosa of the respiratory tract may be important in the protection against influenza infection.

In mice administered Lactobacillus casei strain Shirota (LcS) intranasally, potent induction of interleukin 12, IFN-gamma, and tumor necrosis factor (TNF) alpha, which play a very important role in excluding influenza virus (IFV), was evident in mediastinal lymph node cells. Hori et al (2001) found the titers of virus in the nasal wash of mice inoculated with 200 microg of LcS for three consecutive days (LcS 200 group) before infection were significantly lower than those of mice not inoculated with LcS (control group) in this model of upper respiratory IFV infection, and the survival rate of the mice in the LcS 200 group was significantly greater than that of the mice in the control group. These findings suggest that intranasal administration of LcS enhances cellular immunity in the respiratory tract and protects against influenza virus infection.

Sanu et al (2008) have studied the effects of a hot drink on nasal airflow and symptoms of common cold and flu, and found that the hot drink provided immediate and sustained relief from symptoms of runny nose, cough, sneezing, sore throat, chilliness and tiredness , whereas the same drink at room temperature only provided relief from symptoms of runny nose, cough and sneezing.

The immunity enhancement might be mediated by sirtuins (SIRTs). Sirt 1-7 are nicotinamide adenine dinucleotide (NAD) dependent deacetylases (Haigis et al 2006). CD38 plays multi-faceted roles in immune responses and inflammation (Lund 2006). CD38 blockade in influenza HA-specific T cells inhibited

Interleukin-2 and IFN-gamma production, protein kinase C (PKC) phosphorylation at Thr538, and PKC recruitment to the immunologic synapse induced by antigen-pulsed T cell-antigen-presenting cells (Muñoz et al 2008). However, the major enzymatic activity of CD38 is the hydrolysis of NAD, in fact, CD38 will generate one molecule of cADPR for every 100 molecules of NAD hydrolyzed. Aksoy et al (2006) found CD38 is a major regulator of cellular NAD levels. The larger the ratio of NAD and its reduced form NADH, NAD/NADH, is, the higher the SIRTs level will be (Haigis et al 2006). Sequeira et al (2008) found sirt1-null mice develop an autoimmune-like condition, which observation was interpreted as consistent with a role for SIRT1 in sustaining normal immune function. Van Gool et al (2009) have shown that intracellular NAD concentration promotes TNF synthesis by activated immune cells. They have identified SIRT6 as the NAD-dependent enzyme able to regulate TNF production by acting at a post-transcriptional step. Ferrer et al (2009) found a swimming session of one hour at 75-80% of maximal capacity induced the SIRT3 mediated antioxidant defence in lymphocytes.

4 Photobiomodulation

Photobiomodulation (PBM) is a modulation of laser irradiation or monochromatic light (LI) on biosystems, which stimulates or inhibits biological functions but does not result in irreducible damage. The LI used in PBM is always low intensity LI (LIL), ~10 mW/cm2, which is denoted as LPBM. From 1989 on, many Russian groups have studied the therapeutic effects of intranasal LIL on the local inflammation in vasomotor rhinitis and acute and chronic maxillary sinusitis. In Mainland China, intranasal LIL has been studied to treat internal diseases and the special treatment was called ILILT (Liu et al 2009). Nose-mediated therapeutics in traditional Chinese medicine (TCM) has been a very old system (Gao 1994), but ILILT began in 1998. It has been applied to treat hyperlipidemia, the blood-stasis syndrome of coronary heart disease, myocardial infarction and brain diseases such as insomnia, intractable headache, Alzheimer's disease, Parkinson's disease, post-stroke depression, ache in head or face, migraine, cerebral thrombosis, diabetic peripheral neuropathy, cerebral infarction, acute ischemic cerebrovascular disease, brain lesion, schizophrenia, cerebral palsy and mild cognitive impairment (Liu et al 2009). These studies indicated that serum amyloid β protein, malformation rate of erythrocytes, plasma cholecystokinin-octapeptide, the level of viscosity at lower shear rates, hematocrit, and serum lipid decreased, respectively, while melanin production, red cell deformability, superoxidase dismutase activity and β endorphin increased, respectively, circulation was improved, and immunity was regulated after ILILT (Liu et al 2009).
The vasoconstriction induced dysfunction (Johnson et al 2005) may be improved with ILILT. Blood flow velocity and vascular diameter increased under conditions of LIL (Chertok et al 2008). Su et al (2009) have studied the therapeutic effects of ILILT on vascular diseases. 90 old patients of average age 76.1 years with coronary heart disease or cerebral infarction were randomly divided into two groups, 60 in the treatment group and 30 in the control group. The treatment group and the control group were intranasally treated with low intensity GaInP/AlGaInP diode laser irradiation at 650 nm at 3 and 0 mW for 30 minutes each time once a day ten days each session for two sessions, respectively. After the treatment, blood viscosity at high shear, plasma viscosity, red blood cell aggregation, and total cholesterol decreased in the treatment group, respectively, high-density lipoprotein cholesterol increased in the treatment group, but no significant differences occurred in the control group; low-density lipoprotein cholesterol, redox viscosity at low shear and high shear decreased in the treatment group, but increased in the control group, respectively; while blood viscosity at low shear increased in the control group, but no significant differences occurred in the treatment group. The further discussion indicated that ILILT improvement of blood lipid and hemorheologic behavior might be mediated by SIRT1 (Su et al 2009).
Moreover, there is LPBM on leukocytes (Liu et al 2009), such as its stimulating lymphocytes to produce factor(s) that can modulate endothelial cell proliferation in vitro and its modulating nitric oxide (NO) and cytokines production by leukocytes. There are two ways for PMNs to kill bacteria, phagocytosis and neutrophil extracellular traps, both of which have been found to be induced or promoted with LIL in our laboratory(Liu et al 2009). Many cellular LPBM studies provide the foundation for ILILT on immunological functions so that the lymphocyte proliferation was promoted and the CD3 and CD8 increased and CD4/CD8 decreased after ILILT (Liu et al 2009). Moreover, the low intensity helium-neon laser radiation (LHNL) can induce the INF formation from leukocytes of the donor blood (Leonova et al 1994), and induce a TNF- alpha production from isolated macrophages and an INF-gamma production from isolated macrophages and splenic lymphocytes (Novoselova et al 2006). Kucerová et al (2000) have evaluated the effect of the different frequencies of LIL (diode 670 nm and Helium-Neon 632.8 nm) on the healing process after human molar extractions, and found differences in levels of the saliva IgA and albumin were significant when comparing irradiated and nonirradiated groups.
Blood cells mediate the therapeutic effects of intranasal LPBM on the local inflammation (Liu et al 2009). The LPBM treated patients with vasomotor rhinitis showed a significant increase of T-lymphocytes and a

higher capacity of T-cells to form the migration inhibition factor. For the treatment of LHNL therapy on microcirculation of nasal mucosa in children with acute and chronic maxillary sinusitis, it was found that laser therapy produced a positive effect on microcirculation and reduced the potential of relapses. LPBM is effective in correction of microcirculatory disorders and tissue mechanisms of homeostasis in children with neurovegetative vasomotor rhinitis.
Savtsova et al (1990) have studied the influence of two schemes of laser acupuncture on some cell-mediated and humoral immunity characteristics of mice, as well as on their nonspecific antiviral resistance, in acute experimental influenza infection. The use of both schemes has been found to considerably decrease the severity of infection, enhancing the activity of lymphocytes of infected mice in the graft versus host reaction, the O2-producing activity of alveolar macrophages and modulating the ratio of antihemagglutinins and nonspecific antiviral inhibitors in the blood serum.
The rehabilitation of LPBM on immunity might be mediated by SIRTs. Function-specific homeostasis (FSH) is a negative-feedback response of a biosystem to maintain the function-specific conditions inside the biosystem so that the function is perfectly performed (Liu et al 2009). In Johnson et al (2005)'s study, the persons in the control group were in immunity-specific homeostasis (ISH) so that they had the resistance to the cold, but the persons in the chilling group were far from ISH so that their onset possibility of common cold symptoms increased. There is a FSH-specific redox potential (FSR) according to Karu (1998), and a FSH-specific NAD/NADH (FSN), and a FSH-specific SIRTs level (FSLs). There is no LPBM on the function in FSH (Liu et al 2009). The further the redox potential away from the FSR, the lower the ratio NAD/NADH below FSN and the lower the SIRTs level below the FSLs, and the stronger the LPBM will be in terms of Karu (1998). In other words, LPBM may increase the redox potential until at FSR, the ratio NAD/NADH until at FSN, and SIRTs level until at FSLs. Therefore, ILILT might increase the SIRTs level of the persons in the chilling group until they are at FSLs, respectively.

5 Possible photobiomodulation on flue

A person in ISH can resist influenza virus until the person is far from ISH. ILILT can rehabilitate the person so that there might be the prophylaxis of ILILT against flu. Chilling causes a pronounced constriction of the blood vessels in the nose and shuts off the warm blood that supplies the white cells that fight infection so that the onset possibility of common cold symptoms increased (Johnson et al 2005). As it is pointed out in the last section, the vasoconstriction induced dysfunction may be improved with ILILT. LIL increased the level of the saliva IgA (Kucerová et al 2000). IgA is the major, if not the sole, mediator of nasal immunity to influenza virus in immunocompetent mice (Renegar et al 1991, Tamura et al 1998). LHNL can induce IFN formation (Leonova et al 1994). Repeated application of IFN-alpha substantially increased the protective effect of the cytokine treatment (Kugel et al 2009). LHNL can induce a TNF-alpha production from isolated macrophages and an INF-gamma production from isolated macrophages and splenic lymphocytes (Novoselova et al 2006). The intranasal administration of LcS, potent induction of IFN-gamma and TNF-alpha, enhances cellular immunity in the respiratory tract and protects against influenza virus infection (Hori et al 2001). ILILT can rehabilitate leukocytes (Liu et al 2009). PMNs were the cells primarily responsible for early protection in influenza virus infection (Fujisawa et al 1987). The basal level of influenza-specific antibody-secreting cells in the mucosa of the respiratory tract may be important in the protection against influenza infection (Brokstad et al 2001).
The prophylaxis of ILILT against flu might hold for the high risk individuals. As the U.S. Centers for Disease Control and Prevention (CDC) (CDC 2009) has pointed out, Groups at higher risk for seasonal influenza complications include: Children less than 5 years old; Persons aged 50 years or older; Children and adolescents (aged 6 months–18 years) who are receiving long-term aspirin therapy and who might be at risk for experiencing Reye syndrome after influenza virus infection; Pregnant women; Adults and children who have chronic pulmonary, cardiovascular, hepatic, hematological, neurologic, neuromuscular, or metabolic disorders; Adults and children who have immunosuppression (including immunosuppression caused by medications or by HIV); Residents of nursing homes and other chronic-care facilities. There has been rehabilitation of ILILT on chronic cardiovascular diseases and brain diseases such as insomnia, intractable headache, Alzheimer's disease, Parkinson's disease, post-stroke depression, ache in head or face, migraine, cerebral thrombosis, diabetic peripheral neuropathy, cerebral infarction, acute ischemic cerebrovascular disease, brain lesion, schizophrenia, cerebral palsy and mild cognitive impairment (Liu et al 2009). There will be possible rehabilitation of ILILT on aging, chronic renal dysfunction, cancer and diabetes mellitus (Liu et al 2009). The other high-risk adults might be also treated with ILILT. For example, the immunosuppression except HIV might be treated with ILILT because ILILT can rehabilitate leukocytes (Liu et al 2009).
Many flu complications are far from their respective FSHs. ILILT can rehabilitate the FSHs so that there might be the rehabilitation of ILILT on those flu complications. Bogomol'tsev et al (2003) found that the microcirculatory changes with influenza and other acute respiratory viral infections manifest themselves

with advanced erythrocyte aggregation, activation of vascular-platelet and plasmic links of hemostasis, and high blood viscosity at low shift velocities. URIs result in many biochemical, cellular, and hemostatic changes that could predispose to plaque disruption and thrombosis and then resulted in AMI or stroke (Meyers 2003). The hot drink provided immediate and sustained relief from symptoms of runny nose, cough, sneezing, sore throat, chilliness and tiredness (Sanu et al 2008), which supports the ILILT rehabilitation.

6 Discussion

On April 17, 2009, CDC determined that two cases of febrile respiratory illness occurring in children who resided in adjacent counties in southern California were caused by infection with a swine influenza A (H1N1) virus (Centers for Disease Control and Prevention 2009). The viruses from the two cases are closely related genetically, resistant to amantadine and rimantadine, and contain a unique combination of gene segments that previously has not been reported among swine or human influenza viruses in the United States or elsewhere (Centers for Disease Control and Prevention 2009). Although this is not a new subtype of influenza A in humans, concern exists that this new strain of swine influenza A (H1N1) is substantially different from human influenza A (H1N1) viruses, that a large proportion of the population might be susceptible to infection, and that the seasonal influenza vaccine H1N1 strain might not provide protection(Centers for Disease Control and Prevention 2009). This never-before-seen virus could lead to a killer pandemic(Cohen et al 2009). As some exports pointed out (Cohen et al 2009), the world hasn't done nearly enough over the past 10 years to prepare for a pandemic. They worry that most countries will find themselves without access to vaccines or antiviral drugs, which could become especially dangerous if the virus causes severe disease in many people—which is still uncertain—or evolves to do so. Early on, CDC began to brew a "seed" strain for a possible vaccine against swine H1N1, and by 27 April the World Health Organization in Geneva, Switzerland, was already talking to vaccine manufacturers. One key problem is that the world's influenza vaccine production capacity—which still relies on growing the vaccine virus in chicken eggs—is limited to some 400 million vaccine doses a year and is impossible to expand quickly (Cohen et al 2009). For now, the virus is treatable with the influenza drugs oseltamivir (Tamiflu) and zanamivir (Relenza). But the drug's complex manufacturing process makes it too pricey for many poor nations(Cohen et al 2009).
There are insufficient data available at this point on the swine-origin influenza A (H1N1) virus infection. At this time, the same situation for seasonal influenza complications should also be considered for the swine-origin influenza complications. At this point, the possible prophylaxis and rehabilitation effects of ILILT on flu might be of very importance for the swine-origin influenza and cost much less money.

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