

HYPERTENSION AND ITS VARIANT SYNDROME X: EVOLVING CONCEPTS AND MANAGEMENT

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Abstract

Most scientists agree to a common instigator of the cardiac pathology that anatomically leads to hypertension. The pathological issues can only be resolved by the medical intervention. All conventional treatments provide the symptomatic resolution only. Very little work has been done on the understanding of the Neuro-cardiac pathology induced by herpes family of viruses. The electrical voltage transitional activity can be seen changed, under the influence of the electrolyte imbalance of the myocardium cells i.e. high/ Low Na⁺ or high/ Low Potassium or Calcium. The pathogenesis of primary hypertension is multifactorial. However, the sympathetic nervous system plays an important role in circulatory and metabolic control and has clearly been established as a major contributor to the development of hypertension, with blood pressure elevation being initiated and sustained by elevated sympathetic nervous activity. Increased sympathetic outflow to the heart resulting in increased cardiac output and neurally mediated vasoconstriction of peripheral blood vessels are obvious examples of neural pathophysiological pathways leading to elevated blood pressure.

The consequences of increased sympathetic outflow to the kidneys, perhaps most important in this context are sodium and water retention, increased renin release and alterations of renal blood flow, effects that contribute substantially to blood pressure elevations both acutely and in the long term.

Accordingly, targeting the sympathetic nervous system directly appears to be a logical therapeutic approach for the treatment of hypertension.

However, Sympathetic nervous system is not immune against the viral infection susceptibility. Therefore, we have studied pathological issues driven by the Neuroinvasive viruses that give rise to the anatomical and physiological dysfunctions. That collectively results into the hypertension and other cardiac dysfunctions.

Keywords: Hypertension, Cardiac Neuro Virology, Cardiac Neuritis, Perkinjie Network Lesions Rheumatic Heart Disease, Sheikh's Syndrome

Introduction:

For a long time, Syndrome "X" has been regarded as a root cause of primary & secondary Hypertension¹. The prevailing cause of this ghost syndrome has never been documented, defined or eradicated as yet. However, the patients worldwide are being maintained with a variety of Neuro-electrical (Voltage & Charge, Neurodynamics) & Vascular (Hemodynamic) manipulative medicine and radically performing the "Renal Sympathetic Ablation", to sustain a normal blood pressure.

Great many possible parameters have been established to view the existence of the hypertension, Bacterial, Viral³⁵, Hormonal and anatomical³. In the recent years, scientists are looking closely at the Neuroinvasive viruses and their role in the cardiovascular disease. ^{3, 22-35}

Most scientists agree to a common instigator of the cardiac pathology that anatomically leads to hypertension.³The pathological issues can only be resolved by the medical intervention. All conventional treatments provide the symptomatic resolution only. Very little work has been commenced to understand the Neuro-cardiac pathology induced by herpes family of viruses²⁸⁻³⁵.

The electrical voltage transitional activity can be seen changed, under the influence of the electrolyte imbalance of the myocardium cells i.e. high/ Low Na⁺ or high/ Low Potassium or Calcium.

Therefore, conventionally there is an extensive use of Beta Blockers. Ace Inhibitors, Calcium channel Blockers. Whereas, to influence the hemodynamics, antiplatelets, anticoagulants and diuretics are the drugs of choice being used internationally.

So far, it has worked to halt the transient cardiac neuro pathology instigated by the neuroinvasive viral RNA proliferation that disrupts the nervous system of the heart; the management of the Cardiac Neurodynamics. As the drugs are metabolized, the ailment prevails again. Therefore, a lifetime dependency has been established to keep the hypertension in check.

The pathogenesis of primary hypertension is multifactorial. However, the sympathetic nervous system plays an important role in circulatory and metabolic control and has clearly been established as a major contributor to the development of hypertension, with blood pressure elevation being initiated and sustained by elevated sympathetic nervous activity. Increased sympathetic outflow to the heart resulting in increased cardiac output and neurally mediated vasoconstriction of peripheral blood vessel are obvious examples of neural pathophysiological pathways leading to elevated blood pressure. The consequences of increased sympathetic outflow to the kidneys, perhaps most important in this context are sodium and water retention, increased renin release and alterations of renal blood flow, effects that contribute substantially to blood pressure elevations both acutely and in the long term.

Accordingly, targeting the sympathetic nervous system directly appears to be a logical therapeutic approach for the treatment of hypertension.⁶ This substantiates the neuropathology of the sympathetic nervous system.

However, Sympathetic nervous system is not immune against the viral infection susceptibility; it is only one component of a greater picture of multi focal cardiac neuritis.

Therefore, we have studied pathological issues driven by the Neuroinvasive viruses that give rise to the anatomical and physiological dysfunctions.²⁻²³

That collectively results into the hypertension and other cardiac dysfunctions. ^{24 - 35}.

Worldwide scientists ultimately came to an understanding of the dysfunctions of the cardiac

“Electrophysiology” and have defined the following defects but without identifying the underlying cause:

1. Atrial Fibrillation

2. Atrioventricular Block
3. Right Bundle Branch Block
4. Left Bundle Branch Block
5. Ventricular Tachycardia
6. Ventricular Fibrillation
7. Cardiac Arrest

The world of Cardiology has developed the following instrumentation to the standards of the latest technology to intervene in these challenges.

- Pacemakers
- Automatic Implantable Cardiac Defibrillators (AICD) □ E.R. Defibrillators See figure 1.

However, the root cause of the hypertension remained unknown. Syndrome X is still considered as the guiding light to base the treatment of hypertension worldwide.

Currently Known Pathophysiology:

The physiological change to take place, there has to be a series of processes to take place before a complete defect can be understood. So far, the defects have been identified and defined as the following:

- Biochemical Change
- Electrolytes Imbalance
- Hormonal Imbalance (Adrenal etc)
- Hemodynamic Changes
- Platelet Aggregation
- Atherosclerotic intervention
- Electrical Change
- Electrical Transition Inadequacy
- Electrical Blocks
- Sick Nerves
- Electrical Misfiring
- Sick Nerves
- Anatomical Change
- Myocardial Hypertrophy
- Myocardial Ischemia
- Myocardial Infarction
- Myocardial Infection (Coxsackie Virus)
- Valvular Insufficiencies
- Collateral Symptomatology Resulting Hypertension
- Indigestion
- Kidney Dysfunction

- Renal Sympathetic Anomalies
- High Level of mental stress
- Psychological/ Behavioral
- Physical stress
- Social Stress
- Other

We are still at the crossroads with incomplete knowledge of the heart and its ailments. The hypertension does not exist due to the coronary artery disease only. Majority of the cardiac patients that are not a high risk with the coronary artery disease, that have developed the hypertension, mostly due to the cardiac Neuroviral proliferation. These patients must be categorically identified as the Neuro-cardiac patients with a sub specialty “Neurocardiology”.

I. Materials & Methods:

Twenty Five Patients, 9 women and 16 men with the average age of females 32 and males 45 were tested Positive or Equivocal above normal with the HSV 1 & 2, IgG and /or IgM.

Patients were recorded with the average Blood Pressure ranging 140/90 mmHg to 160/100 mmHg.

See Graph 1.

Neuronalitis of the “cardiac center” and “myocardial neuritis” was suspected to have been in effect. As the documentation of Enhanced cytomegalovirus infection in atherosclerotic human blood vessels, causing hypertension has been concluded.³⁵

And the Detection of human cytomegalovirus DNA, RNA, and antibodies in normal donor blood.³⁵ Antiviral infusions with the neuronal complimentary drugs were administered twice a day for 10 days. Vitals were recorded every 4 hours.

The following Medication Protocol was administered that helped deter the Cardiac Neuroviral Pathology. Average dose of selected antiviral, measured between 5mg to 10 mg/kg was injected through intravenous infusion allowing 30 to 60 drops per minute. N/S 0.9%, Selective Antiviral, Selective Steroidal support (Only non-Diabetic Pt.), Prevalent Vitamins for Neuronal-aid, antihistamine, Analgesic, Heparin (measured dosage for Ischemic CVA patients).

Results:

Thirty Patients, 9 women and 16 men with the average age of females 32 and males 45 were studied with the average Blood Pressure ranging 160/100 mmHg.

After 72 hours of continuous patient management, the blood pressure of the patients seen reducing to 120/70 mmHg.

Males 80% and females 70% responded to the treatment rest of the males 20% and females 30% were put on oral intake of the following regiment.

1. Antiviral 400 mg tid next 10 days.

2. Prednisone 5 mg twice a day for 8 days.

This helped reduced the residual viral load off the neurons and the nerves.

No antihypertensive drugs were used. Patients were discharged with the Neuro complimentary prescription and were followed up every 30 days for one year.

The patients were found to have resolved the root cause of the hypertension and lived a very normal life. The patients that took a break for even a day in the infusion therapy were seen to have regained the blood pressure to where they started. However, the total numbers of infusions remain the same for all the patients to have resolved the hypertension.

See Graph 2.

Discussion

It is the neuro-network of the heart which is constantly conducting the electrical impulses to perform the contractility of the myocardium cells.

The diseases of the nerves (Cardiac Neuritis) of the heart had never been addressed, other than few electrophysiological dysfunctions.

A new scientific revelation has come to light; Herpes family of viruses is highly Neuroinvasive and only affects the nerves and neurons. ⁴. Herpes family of viruses is now divided into three categories. Alpha, Beta and gamma herpesvirinae. There are 48 other members of the herpes virus that are not categorized yet.

The Cardiac Neuro-Viral Pathology:

The most important defect has been grossly ignored or left out of focus, is the Neuroviral-Pathology of The heart. It is a paradigm shift.

By the time we begin to understand the Cardiac Neuro viral Patho-physiology, we will have possible answers for the most of the following cardiac conditions along with the Hypertension.

- All Electrical Dysfunctions
- Atrial Fibrillation
- AV Blocks
- Bundle Branches Blocks
- Bradycardia & Tachycardia
- V-Tachycardia
- Cardiac Neuro Anomalies
- Hypo & Hypertension

It has been already established that the disease of the nerve is instigated by the herpes family of viruses.²

⁴. As the chickenpox appears on various parts of the body, it is first revealed as a small lesion on one side of the localized body.

It may be herpes sore on one side of the lip line or arm or leg.

Herpes External Lesion: See Picture 1 & 2

However, on the other end is also a lesion in the corresponding neuron. See Picture 3.

The Latency Associated Herpes Viral RNA proliferation⁴ at the nucleus level out-numbers the Domestic RNA's that do not synchronize with the nucleus and cellular function. Increased Viral RNA's reflect as an increase in the IgG above normal in serological testing.

Therefore, the primary disease process begins. There is no cell in the heart that is not innervated by a nerve. The only function of the nerve understood is to carry out the electrical impulse and distribute to each cell in a timely fashion, from the intra-uterine development to the life of the heart until the death.

Along the axon the viral RNA's continue to transcribe² down to the end organ and the cellular level of the controlled organ.

See Figure 2.

Axonal Lesion

When an external herpes sore is realized, the resulting pain testifies the involvement of the components of the peripheral and central nervous system. This compromise the function of the whole organ involved. See figure 3.

Therefore, the neural lesions within the myocardial arena are realized. These lesions are like any other lesions depicted by the herpes virus and painful in nature.

When the cardiac neuro-network is inflicted with this Neuroinvasive viral proliferation, anatomical & physiological integrity is compromised. The normal function of the heart is bound to be compromised resulting in a variety of dysfunctions including hypertension. See figure 1.

The external lesions are observed and diagnosed readily. The internal nerve lesions are not visible. It can only be traced if the IgG & IgM of the herpes serology is detected above normal. Higher value of IgG substantiates a continuous war of the antibodies against the Latency Associated Viral RNA proliferation. Cardiac neuro physiology is compromised resulting into Hypertension and other ailments.

Dr. Clyde Crumpacker, professor of medicine at Harvard Medical School, states discovering that CMV can cause hypertension has the potential to lead to new ways of treating high blood pressure, including antiviral medication.³

Heart is also rich in the local neuro network that has ability to manage the voltage threshold and its timely transition through various parts of the heart. Once the nerves or neurons exposed to this virus, after the resolution of the external body infection, The "Latency Associated Viral RNA Transcriptioning continues for life".²

The viral RNA's attachment to the glial cells, schwan cells down the axon and S.A. node, AV node, Bundle of His, Bundle Branches and Perkinjie Network and their proliferation, is responsible for cardiac malfunctioning.

Viral RNA proliferation in the nucleus of the cardiac controlling Neurons or the end nerves (Perkinjie Network) is bound to disrupt the intensity, frequency and the delivery of the electrical impulse. The HSV based sick cardiac nerves respond variably. (See pictureNo.3)

Antibodies to these highly Neuro invasive viruses, Human Herpes Virus 1 & 2, Cytomegalovirus HH5 are found to be the most prevalent and weak.⁴⁻²³

Cytomegalovirus-infected endothelial cells recruit neutrophils by the secretion of C-X-C chemokines and transmit virus by direct neutrophil-endothelial cell contact during neutrophil transendothelial migration.³² Therefore, the neuro viral pathology of the heart disrupts the performance of the myocardium and lead to the hypertension and other electrophysiological dysfunctions.

The neuro network of the heart is not immune against it.

As the Hypertension is the unpredictable episode, it is noted, high blood pressure is brought down by slowing down the transition and the intensity of the electrical impulse and reducing the amount of fluid to pump. We studied the impact of the Neuroinvasive viral RNA's latency associated transcriptioning that had evidenced a variety of physiological & cardiac dysfunctions that were possibly induced by its proliferation, including Hypertension.

Conclusion:

Neuroinvasive viral RNA's latency associated transcriptioning at the proximity of the trigeminal ganglia in the cardiac center and its related nerve fibers to the cardiac Neuro network disrupts the normal physiology of the cardiac center and cardiac Neuro network system. This Neuro invasive viral induced cardiac neuropathy is indeed has been the "Syndrome X" that is re-defined now as the "Sheikh's Syndrome" that is responsible for the following and multisystem pathology.

1. Primary Hypertension
2. Secondary Hypertension
3. Sudden Tachycardia
4. Sudden Bradycardia

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