

# World Record of Hyperlipidemia Deterrence in Shortest time Hyperlipidemia is a direct result of Neuro-Invasive viral Induced Neuronalitis

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## Abstract

**Objectives: Find and treat the root causes of Hyperlipidemia.**

### **Introduction:**

The innervated nerve fibers into the cellular structure of the Liver are not immune to the neuro-invasive viral disease, when the Auto-immune disorder or Neuro immune disorder takes effect.

Human Herpes Virus 1 & 2 and Cytomegalovirus HH6 are found to be the most Neuroinvasive hosted by trigeminal ganglia.

Post infection resolution, the Latency Associated Neuroinvasive Viral RNA Transcriptioning continues for life<sup>12</sup>.

As the viral RNA's out number the cellular domestic RNA's the cellular dysfunction begins and the regulatory genes mute.

Since the hepatic nerve originates at the brain stem in the proximity of the trigeminal ganglia, the transmission of the transcribing RNA's in to the hepatic controlling neurons is very prevalent. Malfunctioning of the neurons under the influence of the viral RNA's causes the hepatic dysfunctions that result into the Dyslipidemia, Hyperlipidemia and other affiliated liver disorders.

### **Materials & Methods:**

A 55 year old woman with right hemispheric post CVA was seen as an out patient, presented with compromised mobility, primary hypertension and Neuro Immune Dysfunction Syndrome. HSV & CMV IgG was found reactive with high values.

- |                  |            |              |           |            |          |              |
|------------------|------------|--------------|-----------|------------|----------|--------------|
| • Total Lipids:  | 1537 mg/dl | Cholesterol: | 565 mg/dl | HDL:       | 39 mg/dl |              |
| • Triglycerides: | 307 mg/dl  | LDL:         | 465mg/dl  | VLDL:      | 61 mg/dl |              |
| • HSV Ab IgG     | 2.1        | Cut Off: 1.0 | Reactive  | HSV Ab IgM | 0.31     | Cut Off: 1.1 |
| • CMV Ab IgG     | 1.8        | Cut Off: 0.9 | Reactive  | CMV Ab IgM | 0.41     | Cut Off: 1.0 |

Thymidine kinase infusions therapy with the neuronal complimentary drugs and Ezetimibe/Simvastatin combination were administered for 10 days. Vitals were recorded every 4 hours.

### **Results:**

As the viral RNA load was reduced 33.33% HSV and 11% CMV respectively, a new world record of 82% LDL reduction was seen in 10 consecutive days of treatment. Whereas, all other components of cholesterol were noted normalized impressively. The patient was found to have resolved the root cause of the Hyperlipidemia and lived a very normal life.

### **Conclusion:**

The overload of viral RNA's of the controlling neurons and the nerve fibers have been eradicated mainly by the thymidine kinase, the regulating genes were turned on again. Therefore, the restoration of the cellular homeostasis was re-instated. A new world record in the Hyperlipidemia deterrence of 82% in 10 days was established.

**Objectives: Find and treat the root causes of Hyperlipidemia**

## Introduction:

Since 1529 many nations have identified epidemics and viral outbreaks that have led to the same clinical symptomatology as in today's world known as Neuro Immune Dysfunction Syndrome N.I.D.S. caused by the Human Herpes Virus (HHV). Many healthcare providers to date believe that, HHV is only sexually transmitted and has been classified as HSV1 and HSV2.

In 1991, the U.S. military in the Middle East (Gulf war), encountered a very unique epidemic challenge. The servicemen and women presented with neurological symptoms those were close to have been the reactivations of herpes virus. It was conveniently labeled as "Gulf War Syndrome", due to the lack of understanding of its clinical manifestation or to avoid an institutional panic.

These and many other misconceptions need to be clarified because it carries a serious social stigma in many societies and cultures around the world.

Due to the Neuro invasive nature of this virus, there has been a great quest for understanding its role in the pathophysiology. Researchers have identified multiple types of herpes virus and their modes of operation in the human body. The family of herpes virus has been identified as Human Herpes Virus 1,2,3,4,5,6,7 and 8.

And all of them are not necessarily sexually transmitted. In today's world, we are dealing with enormous health challenges that stem from these roots.

The innervated nerve fibers into the cellular structure of the Liver are not immune to the neuro-invasive viral disease, when the Auto-immune disorder or Neuro immune disorder takes effect.

Human Herpes Virus 1 & 2 and Cytomegalovirus HH5 are found to be the most Neuroinvasive hosted by trigeminal ganglia. Post infection resolution, the Latency Associated Neuroinvasive Viral RNA Transcription continues for life<sup>10</sup>.

As the viral RNA's outnumber the cellular domestic RNA's the cellular dysfunction begins and the regulatory genes mute. Since the hepatic nerve originates at the brain stem in the proximity of the trigeminal ganglia, the transmission of the transcribing RNA's in to the hepatic controlling neurons is very prevalent. Malfunctioning of the neurons under the influence of the viral RNA's causes the hepatic dysfunctions that result into the Dyslipidemia, Hyperlipidemia and other affiliated liver disorders.

## Materials & Methods

A 55 year female patient with right hemispheric post CVA was seen as an outpatient, presented with compromised mobility, primary hypertension and Neuro Immune Dysfunction Syndrome. HSV & CMV IgG was found reactive with high values.

Total Lipids	1537 mg/dl	Cholesterol	565 mg/dl
Triglycerides	307 mg/dl	LDL	465mg/dl
HDL	39 mg/dl	VLDL	61

HSV Ab IgG	2.1	Cut Off:	1.0 Reactive
CMV Ab IgG	1.8	Cut Off:	0.9 Reactive
HSV Ab IgM	0.31	Cut Off:	1.1
CMV Ab IgM	0.41	Cut Off:	1

The patient was started on aggressive selective antiviral infusion to deter the viral transcription and passive neuronal Cytopathy conservatively on E/S, 10/10 every morning, along with diet restrictions and medication to control hypertension, hyperglycemia and other ailments.

In first 48 hours the **hyperglycemia and hypertension** was successfully controlled. Medications for the secondary ailments were further adjusted. Daily monitoring of hypertension, hyperglycemia, temperature were instituted and data was collected.

### Results:

Patient responded very well to the overall treatment. Definitive improvements were gauged and documented. Clinical symptomatology was observed relieving the patient with progress in general wellness, regain of limbic mobility, normoglycemia and normal blood pressure. Therapy was continued for 10 consecutive days. Another lab test was sent to maintain the consistency of the testing methodology.

As the viral RNA load was reduced 33.33% HSV and 11% CMV respectively, a new world record of 82% LDL reduction was seen in 10 consecutive days of treatment. Whereas, all other components of cholesterol were noted normalized impressively.

Therapy commencement Sep 14, through Sep 23.

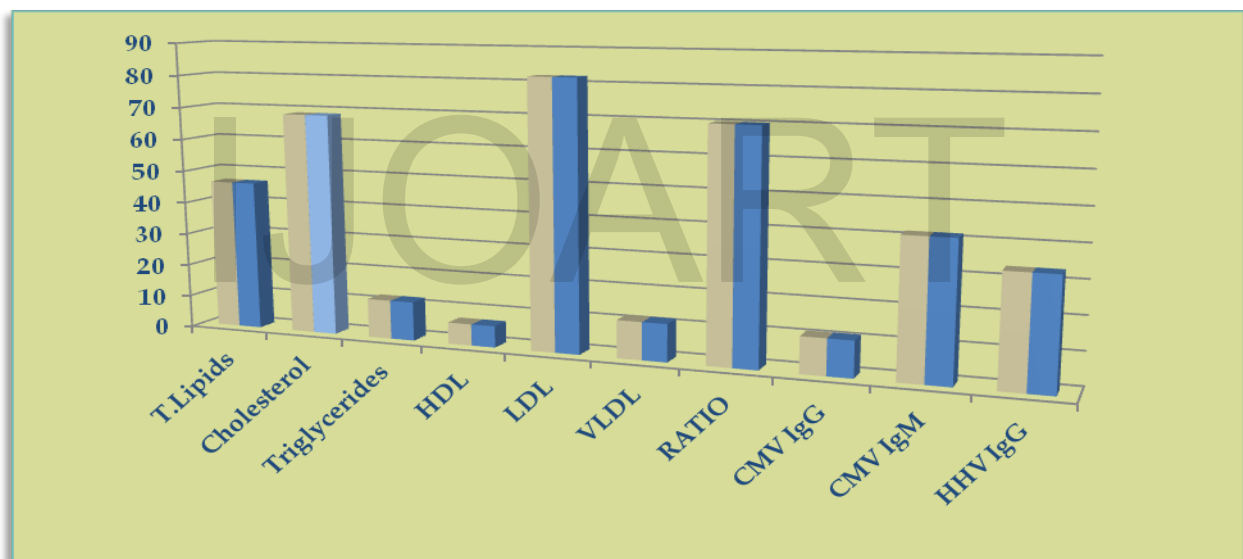
	TEST I		TEST II		
	Sep 11		Sep 25		
Test	Pre-Treatment	Normal	Post Treatment	% age	Actual Change
Total Lipids	1537 mg/dl	500-900	826 mg/dl	46.3 ↓	711 mg/dl
Cholesterol	565 mg/dl	< 200	178 mg/dl	68.5 ↓	387 mg/dl
Triglycerides	307 mg/dl	50 -150	270 mg/dl	12.0 ↓	37 mg/dl

HDL	39 mg/dl	> 45	40 mg/dl	6.6 ↑	01 mg/dl
LDL	465 mg/dl	< 100	84 mg/dl	<b>81.9</b> ↓	381 mg/dl
VLDL	61 mg/dl	< 25	54 mg/dl	11.5 ↓	07 mg/dl
Ratio	14.5	4	4.5	70 ↓	10
HbA1c	7.20%	4.0-6.0	Not Tested	N.A.	
CMV Ab IgG	1.8	0.9	1.6	11.00↓	0.2
CMV Ab IgM	0.41	1	0.24	41.46↓	0.17
Herpes Ab IgG	2.1	1	1.4	33.33↓	0.7
Herpes Ab IgM	0.31	1.1/ 1.2	0.38	0	0

TEST.I: Hormone Lab Jail Road Lahore # 658  
 TEST.II: Hormone Lab Jail Road Lahore # 1971

The patient was found to have resolved the root cause of the Hyperlipidemia and lived a very normal life.

## PERCENTAGE



## DISCUSSION

Patient had a history of 2 year post CVA, hypertension, and hyperglycemia with compromised limbic mobility of right side of the body. Previous lab data was available only for the hyperglycemic pattern.

Pre-treatment lab data revealed a higher average of hyperglycemia through HbA1c, which is a clear indication of poor management of hyperglycemia as well as Lipidemia. Hypertension was marginally controlled after the stroke, patient was found inclining towards secondary hypertension. Patient was suspected to have been suffering from the Neuro Immune Dysfunction Syndrome (NIDS).

Lab tests for post viral infection resolution; residual viral load of Human Herpes Virus 5 in particular was requested for evaluation. The lab results substantiated the CMV IgG values much more than the cut off values.

This only confirmed our diagnosis based on Dr. Wagner's research, **"During the Latent Phase, productive cycle genes are generally transcriptionally and functionally quiescent and only the latency associated transcript (LAT) is expressed."**<sup>10</sup>

Patients with Hyperlipidemia/ hypercholesterolemia have been found positive for the HHV5 (CMV Ab IgG reactive).

It can only be rationalized as following.

1. Endoplasmic Reticulum synthesizes the lipoproteins where the Host RNAs perform their primary function.
2. A continuous Viral RNA transcription and gene expression do not synchronize with the host Neuronal RNA activities.
3. Overburdening of the host neuron with Latency Associated viral RNA Transcription.
4.  $\alpha$ -mRNAs detach from the viral genome and transcribe to  $\alpha$  Protein to re-attach to the viral genome that give rise to  $\beta$ -mRNAs.
5.  $\beta$ -mRNAs detach from the viral genome and transcribe to  $\beta$  protein to re-attach with viral genome.
6. This gives rise to the Viral DNA replication and structural RNAs.<sup>10</sup>
7. Energy for the process in step 3A & 3B is drawn preferably from the free fatty acids and low density lipoproteins readily available in the neuronal cytoplasm.
8.  $\beta$  protein influencing the host neuron's own DNA strand, this in turn, causes the resident DNA mutation on the LDL receptor.
9. This instigates a higher demand of the LDL and free fatty acid production by the liver; therefore, the state of Hyperlipidemia prevails.

When combated with antiviral and E/S dosage together, every 1 (one) fold deterrence of CMV viral RNA transcription helped lowered 8 folds of LDL-C; every 1 fold reduction in the Herpes IgG lowered 3 folds of LDL-C as compared with the Feldman study's first 6 weeks outcome on E/S, 10/10 alone.

Patient therapy was further adjusted to keep the homeostasis in these challenges. E10/ S10 had proven itself to be the blessing in disguise for the scientists to win the war against Hyperlipidemia. Provided, the post infection viral RNA transcription is deterred. E/S, 10/10 administered alone to deter Hyperlipidemia reported to have lowered 47% LDL-C. Whereas, when the CMV viral RNA transcription interrupted, consequently, LDL-C was lowered 81.9% in 10 days treatment. Total cholesterol was lowered by 68% and Total lipids lowered by 46%.

There is a strong correlation between hypercholesterolemia and coronary heart disease<sup>2</sup>, transient ischemic attacks and Cerebrovascular accidents. Primary hyperlipidemia is the most common form of cholesterol disorder and is confirmed when a total cholesterol level is seen between 240 to 350 mg/dl.

Studies have shown that aggressive treatments to lower LDL levels can improve morbidity and mortality and can often reduce the severity of adverse coronary events.<sup>4-6</sup>

Lowering lipids level has been shown to decrease the risk of coronary artery disease and to help prevent additional coronary events. Our continuing improvement in understanding the neuro-invasive pathology has allowed us to develop increasingly effective treatment options.

This process lays a milestone of a primary disease, now identified as “Sheikh’s Syndrome”. As a final product of the viral RNA ridden neurons, the molecular composition of its enzyme or stimuli produced is altered from its original state and carries a grave influence of the invading RNA’s molecular configuration; therefore, the secreting enzyme or the stimulant orders issued to the end tissue/ organs, compromises the normal physiological process. Therefore, the pathophysiology gives rise to some of the following secondary diseases. The chronicity of the viral infection, establishes the root cause.

ADD	Encephalitis	Meningitis
Allergies	Endocrine Disorders	Metabolic Disorder
Alzheimer	Gastric Ulcer	Neoplastic-Tumors
Amenorrhea	Gastroenteritis	Nephropathy
Anemia	Gynecomastia	Neuralgia
Aneurysms	Hyperlipidemia	Neuropathy
Atherosclerosis	Hypo/ hyperthyroidism	Parkinson’s
Bell’s & Cerebral Palsy	Hypohemoglobinemia	Pituitary Dysfunctions
CAD	Hypothalamic Dysf.	Recurrence of Minor Infections
Cancer	IBS	Respiratory Failure
CFS	Impotence	Stroke
Chromosomal Disorder	Infertility	Sudden Tachycardia
Cognitive Disorder	Liver Dysf.	Teenage Drug Addiction
Diabetes Mellitus	M.S.	TIA

## Conclusion:

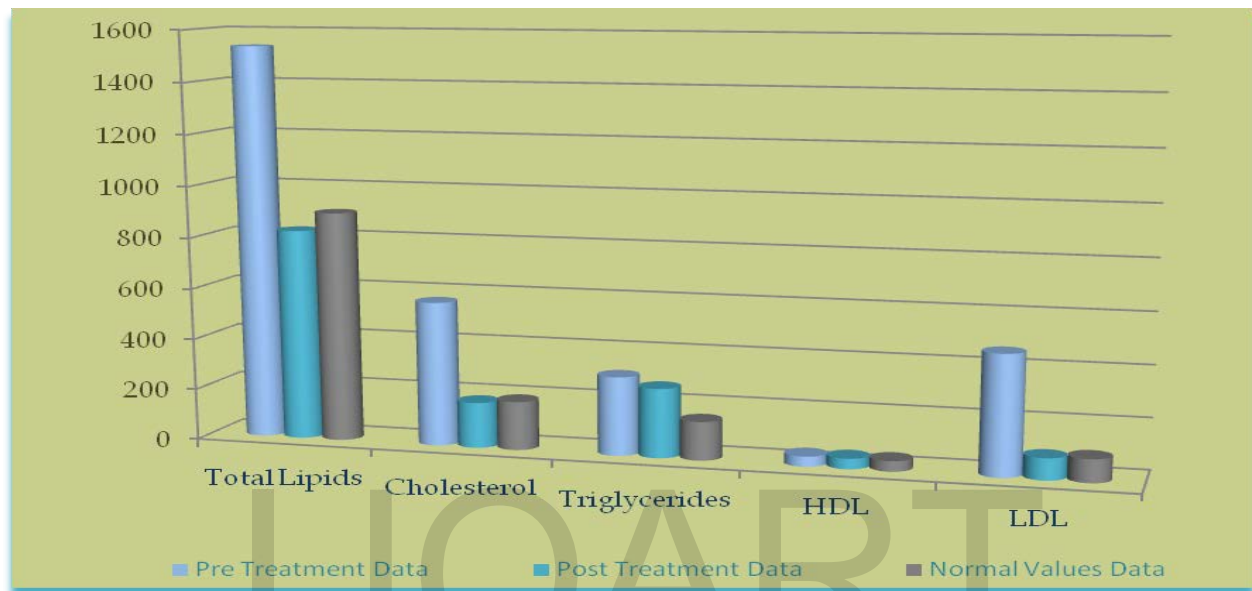
The overload of viral RNA’s of the controlling neurons and the sensory/Motor nerve fibers have been eradicated mainly by the thymidine kinase, the regulating genes were turned on again to prevail the homeostasis of the neurons axonal fiber and the liver. Therefore, the restoration of the cellular homeostasis was re-instated. A new world record in the Hyperlipidemia deterrence of 82% in 10 days is established.

To combat and disrupt the viral RNA transcriptioning, concurrently treating and alleviation of the secondary diseases is the most crucial strategy for the patients with anatomical and/ or physiological multiple disorders. The designed treatment plan was well tolerated by the patient and brought extremely appreciable clinical results. Patient prognoses are excellent. It proves that if the brain is healthy, the body is going to be healthy, no matter which physiological disorder it is, the ultimate control center indeed is the CNS. Ezetimibe/Simvastatin, on the other hand has proven to be an effective and a well-tolerated option for the expeditious management of hyperlipidemia when administered together with the antiviral regiment to deter the viral RNA transcriptioning.

**It has helped us set a new world record on deterring the hyperlipidemia in the shortest possible time.**

The combined action of this medication has allowed individuals in our and many other studies meet LDL-C goals without the high-dose side-effect profile associated with Simvastatin.

E/S is an efficacious and a pharmoeconomically desirable form of therapy that can help reduce hyperlipidemia, total cholesterol and in turn the risk of coronary events<sup>1</sup>.



## REFERENCES

1. Ezetimibe/Simvastatin (Vytorin®): A Dual Approach to Treating Hyperlipidemia
2. Katrina Touchstone, PharmD Candidate, Laura Stark, PharmD Candidate, Marlon Honeywell, PharmD, Marvin Scott, PharmD, and Evans Branch III, PharmD, DRUG FORECAST: 322 P&T® • June 2005 • Vol. 30 No. 6
3. Latts L. Assessing the results: Phase 1 hyperlipidemia outcomes in 27 health plans.
4. Am J Med 2001; 110(6A):17S-23S.
5. Dipiro J, Talbert R, Yee G, et al (eds). Pharmacotherapy: A Pathophysiologic Approach, 5th ed. New York: McGraw-Hill; 2002.
6. Feldman T, Koren M, Insull W, et al. Treatment of high-risk patients with ezetimibe plus simvastatin co-administration versus simvastatin alone to attain National Cholesterol Education Program Adult Treatment Panel III low-density lipoprotein cholesterol goals. Am J Cardiol 2004; 93:1481-1486.

7. Davidson M, Ballantyne C, Kerzner, et al. Efficacy and safety of ezetimibe co-administered with statins: Randomized, placebo-controlled, blinded experience in 2382 patients with primary hypercholesterolemia. *Int J Clin Pract* 2004; 58: 746-755.
8. Grundy S, Cleeman J, Bairey C, et al. Implications of recent clinical trials for the National Cholesterol Education Program Adult Treatment Panel III Guidelines. *Circulation* 2004; 110:227-239.
9. Bays H, Ose L, Fraser N, et al. A Multicenter, randomized, double-blind, placebo- controlled, factorial design study to evaluate the lipid-altering efficacy and safety profile of the ezetimibe/simvastatin tablet compared with ezetimibe and simvastatin monotherapy in patients with primary hypercholesterolemia. *Clin Ther* 2004; 26:1758-1773.
10. Vaughan C, Gotto A. Update on statins: 2003. *Circulation* 2004;110:886-892.
11. Ballantyne C, Blazing M, King T, et al. Efficacy and safety of ezetimibe co-administered with simvastatin compared with atorvastatin in adults with hypercholesterolemia. *Am J Cardiol* 2004; 93: 1487-1494.
12. <http://darwin.bio.uci.edu/~faculty/wagner/hsv2f.html>
13. <http://darwin.bio.uci.edu/~faculty/wagner/rnatrans.html>

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