– Original Research –

The Anti-Inflammatory and Antiplatelet Effects of Boluoke (Lumbrokinase) in Cancer Patients

by Dr. James A. Kholos

Abstract

Boluoke (lumbrokinase) was administered to a group of 16 participants, formerly diagnosed with either cancer or a serious cardiovascular condition, to monitor the antiplatelet effect in their blood and urine. A control group with normal medical histories was dosed aspirin for comparative analysis. Boluoke decreased hypercoagulability in all patients who typically do not respond to aspirin. Similarly, a smaller decrease in platelet aggregation and coagulability levels was observed in the control group.

Introduction

Hypercoagulable blood conditions are associated with several chronic and debilitating illnesses, from cancer to heart disease, fibromyalgia, and chronic fatigue syndrome. Recent research conducted by Dr. David Berg of Hemex Laboratory has uncovered that the hypercoagulable state can result in increased plasma viscosity and fibrin deposition on capillary walls, impeding oxygen and nutrient delivery to the tissues and slowing cellular waste removal from the circulatory system. Researchers have demonstrated that excess platelet fibrin production is associated with greater risks for myocardial infarction, congestive heart failure, and even death. Furthermore, platelet dysfunction remains a challenge to practitioners when monitoring and screening coagulopathy with useful tests.[1]

The key points:

- It is a bad thing when blood clots form in the body when and where they should not. Heart attack and stroke, pulmonary embolus, and thrombophlebitis result. It is a bad thing when clots do not form when and where they should. Bleeding, bruising, and hemorrhaging result.
- Antiplatelet agents prevent (some) thromboses from forming by preventing platelets from clumping and then setting of a cascade of events leading to plaque/clot formation.
- Aspirin is the prototypical antiplatelet agent. There are many others. A significant percentage of people do not respond to aspirin fully. Other antiplatelet agents might be useful for them.
- There are groups of people that are at high risk for inappropriate blood clotting. Many of those with cancer and cardiovascular conditions are examples.
- Medline literature references lumbrokinase as an antiplatelet agent that works, but it does not appear to be understood how it does so compared with aspirin. There are articles that speculate about clinical uses in humans of lumbrokinase with other existing agents that have antiplatelet effects.
- The following study is limited in the current number of participants; a larger study, appropriately blinded and followed long term, is recommended.
- This study looks at the effect of lumbrokinase on measures of inflammation, platelet aggregation, and platelet function.

Platelet Dysfunction and Aspirin Therapy

The value of low-dose aspirin in the prevention of myocardial infarction and apoplexy (stroke) has been demonstrated conclusively, and this resulted in increasing numbers of patients presenting for surgery while receiving aspirin.[2] Sonoclot analysis measured a declivity in the instruments' recording signature in patients receiving aspirin compared with those not receiving it. Correspondingly, cancer patients had more hypercoagulability than cardiac patients. Sonoclot analysis is based on changes of blood viscosity as a basis for a test of coagulation function. The Sonoclot analyzer is a device that measures the changing impedance to movement imposed by the developing clot on a small probe vibrating at an ultrasonic frequency in a coagulating blood sample.[3]

The present study is focused on examining platelet aggregation and blood viscosities, including infection and inflammation, in the volunteers taking Boluoke to assess their circulatory health. Selection was based

on patients' confirmed diagnosis with cancer or heart disease. A normal control group was compared for the purpose of determining differences between the findings of the two groups. The method of qualification was darkfield micrographs taken from fresh blood, then testing C-reactive protein (CRP), measuring significant inflammation in each patient with a primary interest in individual response.

Platelet aggregation (clotting) has long been shown to cause and/or be associated with major degenerative diseases. Platelet reactivity is responsible more than any other cause for most myocardial infarctions by the formation of platelet clot aggregations. In a former study in collaboration with Dr. James R. Privitera, expert in darkfield microscopy and author of *Silent Clots: Life's Biggest Killers*, 100 charts were randomly selected from his patient population (every third chart). The study concluded that one out of every three patients had excess clotting. In a second study, 22 patients with abnormal samples were selected. The abnormal darkfield samples were compared with results from highly sensitive CRP tests. The study revealed a greater than 95% correlation between significant clotting and abnormal CRP, as observed with darkfield microscopy, where a measure of 4+ platelet aggregation (which equals four times the size of a red cell in greatest diameter of the clot) was utilized.[4] In the current study, while examining his patients' charts, we found 95% of metastatic cancer associated with platelet aggregation. The amount of clotting a patient presented was cross-referenced by three different test methods: CRP was compared with darkfield microscopy findings to achieve a consensus, factoring in the measurement of urinary 11-dehydro-thromboxane B2 (11dhTxB2) findings.

Beta-thromboglobulin is considered the most abundant platelet-specific protein associated with clotting. Privitera confirmed that platelet aggregation was the most common problem observed in both groups of patients. Many natural substances treat platelet aggregation effectively. In this study, Boluoke showed a remarkable efficacy in reducing hypercoagulation.

Study Design

Sixteen people with varied case histories from cancer to heart disease were selected. Eight normally healthy men and women without history of acute or chronic disease were chosen as a control group. Over six months, they were checked in intervals for changes or improvement in blood viscosity, inflammation, and subjective symptoms related to their diagnoses. Results were obtained in both control group and diagnosed patients through testing methods that included darkfield microscopy, CRP, and measurement of urinary 11dhTxB2 findings. All participants, except four in the normal control group, were instructed to self-administer two doses of 20-mg capsules of Boluoke 30 minutes before breakfast and 30 minutes before the evening meal (2 times per day).

The volunteers in the study were advised to moderate their use of sugar, spices, or alcohol stimulants, as well as to reduce prescription medications during the study period. Interval testing was useful in monitoring each volunteer while on the protocol. Blinded random urine samples were collected and shipped to Corgenix Laboratories for testing with its AspirinWorks Test. A portion of the control group was administered low-dose aspirin (100 mg) during the study to validate the effective reduction in urinary 11dhTxB2 in apparently healthy individuals. The AspirinWorks Test is the only FDA-cleared test that measures this metabolite of thromboxane A2, the target of aspirin therapy.

Research Considerations

Tools and Methods

Darkfield microscopy was utilized to examine a drop of blood drawn from each participant's fingertip. A magnification of 150×, which enlarges the specimen up to 1,500 times its normal size, is achieved. One of the great benefits of darkfield live blood analysis is the quality of visualization, seeing in the moment platelets and fibrin clinging to the cholesterol crystal deposits, which directly results in narrowed arteries with impaired blood flow interfering with the oxygen transport to the tissue cells. Herein, platelet and fibrin complexes are readily seen as being much larger than red blood cells.

An independent laboratory was used to analyze the blood of the 16 participants for the presence of beta-thromboglobulin, a protein secreted by abnormally sticky platelets. The results of the laboratory test to darkfield results were found to correlate 100%. The presence of significant clots, as seen by the darkfield, was confirmed by an accompanying increase of beta-thromboglobulin secretion. Using darkfield technology gives the practitioner an immediate finding as to the degree of danger of platelet aggregation.

This early warning signals a high blood viscosity, a sign of bad things to come. Although one cannot actually see the blockage of the capillaries, causing diminished oxygen, when it happens in the coronary artery the patient suffers a heart attack or, in a cerebral artery, a stroke. Dosing aspirin to render platelets nonsticky may not be the long-term safe answer.

Aspirin's antithrombotic effects have been recognized for many years, and it is widely prescribed to help prevent cardiovascular disease. Low-dose aspirin reduces cardiovascular events by as much as 25% in patients with arterial vascular disease. Similarly, in high-risk vascular patients, aspirin therapy results in a 34% reduction in nonfatal myocardial infarction, a 25% decrease in nonfatal stroke, and an 18% decrease in all-cause mortality.

Aspirin functions by irreversibly acetylating the platelet cyclooxygenase-1 (COX-1) enzyme, thus inactivating it for the life of the platelet. Aspirin also inhibits the cyclooxygenase-2 pathway (COX-2), a second cyclooxygenase isoform

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induced by inflammatory stimuli, but to a much lesser extent than COX-1. Low-dose aspirin blocks more than 95% of platelet COX-1 activity, which results in a decrease in the production of thromboxane A2 (TxA2). Unfortunately, TxA2 has a very short half-life in the blood, making it a difficult analyte to measure.

TxA2 is produced from arachidonic acid by many cells and causes irreversible platelet aggregation and vascular and bronchial smooth muscle contraction. TxA2 is rapidly hydrolyzed nonenzymatically to form thromboxane B2 (TxB2). Thromboxane B2 in the plasma is converted enzymatically into a number of metabolites, including 11dhTxB2 and 11-dehydro 2,3dinor thromboxane B2, which are cleared by the kidneys and excreted in the urine. 11dhTxB2 is the most abundant urinary metabolite of TxB2 has a relatively long circulating half-life (45 minutes), and is a very stable molecule in urine.

The utility of urinary 11dhTxB2 as a measure of thromboxane production was confirmed in a study of TxB2 metabolism. In these experiments, urinary levels of 11dhTxB2 provided a more accurate indication of in vivo thromboxane metabolism than TxB2 measured in the blood, the latter method being confounded by technical difficulties encountered in the blood collection process itself. Thus, quantitation of urinary 11dhTxB2 may serve as the optimum measure for TxA2 production by platelets and an accurate index of in vivo platelet activation.

The AspirinWorks Test measures urinary 11dhTxB2 and can assist health care practitioners in the optimal utilization of traditional and/or alternative therapeutic measures to improve patient care. The 11dhTxB2 metabolite has proven to be an independent predictor of risk for heart attack, stroke, or cardiac death and may be used to identify individuals at risk for these adverse clinical events.

The AspirinWorks Test was included in this study based upon its ability to monitor a reduction in systemic thromboxane production. Because clot formation is multifactorial, involving not only platelets but coagulation proteins, the need to evaluate platelet hyperreactivity was apparent. It has been suggested that Boluoke functions by means of the lytic system. It is a combination of proteolytic enzymes that function like tissue plasminogen activator (t-PA) promoting the clot or fibrin breakdown, or lysis. The suggested benefit of lumbrokinase over t-PA is that lumbrokinase only acts in the presence of fibrin, which would indicate active clot formation or fibrin deposition. Under the conditions of active clot formation or fibrin deposition, the associated bleeding risk would theoretically be reduced. T-PA has been demonstrated to activate platelets; therefore, it is necessary to investigate if platelet activity is increased due to function of lumbrokinase, which may indicate the need for an individualized approach to combinational therapy that could treat both the enzymatic and the platelet components of thrombus formation or fibrin deposition.

Thromboxane is a powerful chemical that causes the blood vessels to constrict and platelets to become sticky, forming a fibrin clot to stop bleeding. However, in the cases reviewed, conditions such as inflammation from arteriosclerosis, high blood pressure, elevated cholesterol, and stress from patients affected by previously diagnosed excessive thromboxane levels were found the most vulnerable. This insight offers a profound perspective of the risk factors for heart disease and stroke. Contrasting volunteers taking Boluoke reduced their risk of clots by 68%, tested in weekly intervals under darkfield microscopy. By establishing a baseline using 11dhTxB2, the practitioner can monitor the effectiveness of therapy and keep the patients motivated when positive results are produced.

In contrast, the CRP is for systemic inflammation used in cardiac evaluation studies that have demonstrated that baseline highly sensitive CRP concentrations can be predictive of future cardiovascular events.[5]

Studied together with darkfield analysis, observing platelet and fibrin complexes allows the practitioner to identify a diseased patient who can be helped before suffering a clinical event. In the quest to clarify Boluoke as an adequate adjunctive protocol to offset the clotting epidemic seen worldwide, the findings below taken from this study, utilizing science and technology, may not have established irrefutable agreement; however, the gravity of these results is compelling enough as proof toward the elimination of at least one cause of cardiovascular disease.

Data measurements have been quantified and means have been compared using 11dhTxB2, taking into consideration standard deviation. A one-tail P(T = t) value has been computed according to the following null hypothesis: Mean 11dhTxB2 measurement levels for groups equal at the end of the six-month period.

Quantitative Analysis

Table 1: 11dhTxB2 Results Pre and Post Boluoke

N=16	Baseline	Post Boluoke	P value
Mean	2156	2778	0.275 NS
Range	3638	17813	
Min	320	570	
Max	3958	18383	

Table 2: Percent Change in 11dhTxB2 by Group

Group	% Change
Controls	–77
All diseases	+ 45
Heart Disease	+ 3
Cancer	+ 77
Noncancer Diseases	+ 2

Results of Measurements

11 Dehydro-Thromboxane B2 Test Results

Measurements of 11dhTxB2 from all 16 participants remaining after the six-month period of the study were averaged at 2778 and compared with the initial baseline averaging 2156. The most significant change was found in the most stricken condition, cancer: +77; compared with the control group: −77. The P(T<=t) one-tail test revealed a 97.35% confidence that the variances were not comparably similar. Results reject the null hypothesis and support the original hypothesis that Boluoke demonstrated greater antiplatelet effect in the long term than aspirin.

Results of Darkfield Microscopy

Darkfield findings were recorded utilizing a 5-point scale: 1 means no clotting, 2 mild clotting, 3 moderate, 4 severe, and 5 highest risk for a cardiac event. Positive clotting of 4+ points revealed increased platelet aggregations and morphological alterations in 70% of those studied. All participants' initial darkfield examinations revealed an increased number of lymphocytes, indicating inflammation and infection. In the second screening, a 2-point reduction in clotting was noted.

C-Reactive Protein (CRP) Findings

The cardiovascular and peripheral vascular disease risk assessment indicated each group prior to protocol to have levels of high vascular-disease progression from unstable plaque buildup in the arteries. Although CRP has been found to predict adverse cardiac events, none in either group experienced any symptom of clinical significance. CRP was used primarily as a marker of systemic inflammation. According to a recent study published in the *Medical Laboratory Observer*, "CRP concentrations can be predictive of future cardiovascular events. Baseline CRP has been found to be useful in predicting rates of recurrent ischemic episodes. A variety of cardiovascular risk factors, such as smoking, hyperlipidemia, diabetes mellitus, and obesity are all associated with increased CRP and may confound the clinical significance of baseline CRP measurements for cardiovascular risk stratification."[6] This important finding may account for the patient variability in clinical histories that reported, on average, 2.9 to 3.5 CRP findings. The table below shows the CRP scale.

Table 3: hsCRP Scale

Risk	hsCRP (mg/dL)
Low scale	<1.0
Average	1.0–3.0
High	>3.0

Summary

Measurements from all assays concluded to confirm the statistical significance demonstrated of the antiplatelet effect from participants' taking Boluoke, contrasting with the small group on aspirin. It can be assumed that after one month, individuals taking Boluoke have fewer side effects and greater antiplatelet activity.

The elimination of blood clots prevents the buildup of toxins, free radicals, and waste products that cause death and disease. Healing from inside out from applied Boluoke (enzymatic therapy) with timely monitoring could change the course of a patient's life. Boluoke works by reducing deposits within the vessels from excessive cholesterol fibrin buildups as prevention for vascular or arterial dysfunction. The observed benefit demonstrates improved clinical progress without side effects, a positive treatment outcome. Over a period of six months, monitoring each volunteer established a closer doctor-patient relationship and generated goodwill and patient referrals.

The measured data were an approximate means applying accepted laboratory techniques to evaluate endothelial function. Most heart attacks and stokes occur from abnormal platelet activation and endothelial dysfunction (from circulatory clots and underlying cholesterol buildup within the walls of arteries). It is evident under darkfield examination; the changing pathologies may be visualized as a predictive window into the future, as these findings showing toxicity and free-radical waste indicate disease.

Discussion

Practitioners who have not had training in darkfield technique may find the foregoing collaboration correlating CRP testing with Boluoke an exciting opportunity to take a first step toward this type of biomedical research. Identifying cardiovascular and peripheral vascular disease risk assessment constitutes an advanced tool applied in preventive medicine. Darkfield microscopy offers the practitioner immediate findings to visualize the clotting mechanism. The primary biomarker for inflammation in both cancer and heart patients is hypercoagulability of the blood. Collectively, highly sensitive CRP and darkfield microscopy are credible and worthwhile assessment tools, extremely important when used in evaluating inflammatory mediators in chronic disease. In future research, a larger population would demonstrate greater results than the above sample. However, with the apparent trend demonstrated, the next step is to redesign a clinical study to include people who practice a calorie-restrictive diet. The answers that we do not know define what we are certain about. In this study, typical expectations have, in fact, revealed the profound conclusion that we are on the right path.

Comments from Experts

"Boluoke in place of Coumadin may assist recovery from thrombosis, platelet fibrin complexes and clots. Traditional use of blood thinners has proven to shorten lives as it calcifies arteries, and many claim it is the most dangerous drug that doctors prescribe."

– Garry F. Gordon, MD

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Notes

1. Hett et al.
2. AspirinWorks.com.
3. Hett et al.
4. Privitera J. The clotting epidemic.
5. Kazmierczak.
6. Kazmierczak.

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