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Irena Vasileva Velcheva *, Nadia Mladenova Antonova *, Tsocho Kmetski

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Review

Significance of Whole Blood Viscosity in Acute Ischemic Stroke

Irena Velcheva [1,*], Nadia Antonova [2] and Tsocho Kmetski [1]

[1] Department of Neurology, University Hospital Acibadem City Clinic, Okolovrasten Pat Str.127, 1407 Sofia, Bulgaria

[2] Department of Biomechanics, Institute of Mechanics to the Bulgarian Academy of Sciences, Akad. G.Bonchev str., Bl.4, 1113 Sofia, Bulgaria

* Correspondence: velchevairena@gmail.com (I.V.); antonova@imbm.bas.bg (N.A.)

Abstract

The paper provides a comprehensive review of the relationship between whole blood viscosity (WBV) and acute ischemic stroke (AIS). A significant increase of diastolic blood viscosity (DBV) at the onset of AIS was established in the small artery occlusion stroke subtype. In patients with atherothrombotic cause of AIS systolic (SBV) and DBV values were higher than in those with an embolic cause. The higher WBV at low shear rates on hospital admission is associated with an increased risk of early neurological deterioration and disease progression in the patients with AIS. Most studies reveal association of the increased at the stroke onset WBV with poor functional outcome after applying intravenous thrombolysis or endovascular thrombectomy. However significant reduction of the blood viscosity variables after the combined use of these therapeutic methods in AIS patients was observed. Whole blood viscosity has an obvious effect on the risk of AIS, its clinical severity and outcome. Further research is needed due to the different methods of WBV measurement, the different time intervals of the patients` examination and the different associations of WBV with some of the applied treatment strategies.

Keywords: acute ischemic stroke (AIS); whole blood viscosity (WBV); WBV measurements

1. Introduction

Stroke is the second leading cause of death globally [1] and ischemic stroke represents 85% of all stroke cases. In 2021 the global burden of ischemic stroke is presented with 69.9 million prevalent and 7.8 million incident ischemic strokes [2]. Among the pathophysiological factors for cerebral ischemia, whole blood viscosity (WBV) has a certain significance for the development of ischemic stroke. This is accomplished through its influence on cerebral blood flow and cerebral perfusion [3] and its role in atherosclerosis and thrombogenesis.

The study aims to provide a comprehensive overview of changes in blood viscosity in acute ischemic stroke (AIS), as well as to analyze these changes depending on the methods and measuring devices used. The analysis of the effects of WBV on the risk of stroke, its etiology, imaging diagnostics and treatment strategies is of essential importance for the development and outcome of the disease.

2. Whole Blood Viscosity and Cerebral Ischemia

Viscosity quantifies the internal frictional force between adjacent layers of fluid that are in relative motion. Whole blood viscosity is the resistance to flow in the blood vessels, and its major determinants are hematocrit (Hct), plasma viscosity (PV), erythrocyte aggregation (EA) and erythrocyte deformability (ED). Blood behaves as a non-Newtonian fluid in the circulation, and it

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changes in a non-linear relationship depending on the shear rate [4]. In the blood vessels, WBV causes changes to blood flow and leads to endothelial dysfunction [5,6]. The wall shear stress, the friction force exerted parallel to the vessel wall, is affected by WBV and appears to be the primary determinant of the endothelial cell function [7,8]. The changes of wall shear stress regulate the nitric oxide-dependent flow mediated vasodilatation and thus influence the tissue perfusion [9]. It induces endothelial remodelling and alters vascular physiological responses, while at high shear mechanism direct injury of the vessel wall can be provoked [5]. The interaction of hyperviscous blood may cause structural and biochemical alterations in the capillary endothelium [10]. Whole blood viscosity also influences the basal cerebral perfusion, and this was reported by Gyawali et al. [3] who found a significant correlation of the WBV at 20 sec^{-1} with the CT perfusion parameters and the MRT DWI volume.

The involvement of WBV in thrombosis and hemostasis is accomplished mainly through its determinants: erythrocytes and fibrinogen (Fib), and their interaction. The hemorheological effects related to thrombosis include a rise in hematocrit (Hct), an increase in erythrocyte aggregation, mediated mainly by fibrinogen and immunoglobulins and a decrease in erythrocyte deformability with alteration of the resistance to flow [11]. In the microcirculation, fibrinogen stabilizes erythrocyte aggregation and leads to an increase in WBV [12]. Erythrocytes also have effects on platelet reactivity; they interact with the vessel wall and take part in the structure and properties of clots [11]. So, in patients with AIS parallel alterations of the hemorheological and coagulatory parameters are observed [13].

3. Whole Blood Viscosity and the Risk of Stroke

A significant association of blood viscosity and hematocrit with stroke appearance was established in a prospective 5-year follow-up study of a population sample by G.D.O. Lowe et al. [14]. The authors also found a significant association between plasma viscosity and fibrinogen for total cardiovascular events and stroke. In another study of 378,210 individuals, a Mendelian randomisation study did not reveal a causal effect of WBV on ischemic stroke [15].

The risk factors for stroke are closely related to WBV. In a population-based cohort, a linear positive association was established between WBV and blood pressure [16]. In patients with heart failure, the higher value of WBV is associated with a higher occurrence of cardiovascular diseases and lower survival rates [17], so in these patients, WBV predicts AIS [18]. Also, in patients with nonvalvular atrial fibrillation, increased WBV at high and low shear rates shows significant correlations with elevated CHA2DS-VA and CHA2DS2-VASc scores as indicators of higher thromboembolic risk [19]. Another closely related disease to AIS risk is diabetes mellitus, and it is characterised by increased WBV and reduced blood flow, especially in the microcirculation. This is due to the association with the induced by the high WBV oxidative stress [20]. The increased WBV and plasma viscosity in patients with transient ischemic attacks correlate with increased blood pressure values, age, leucocyte count, fibrinogen and cholesterol [21].

When discussing WBV and the stroke risk, we should note the observations of Tsuda et al [22] and Li et al. [23], who reported an increase of WBV at low shear rates in subjects with silent cerebral infarctions with ischemic lesions on brain imaging but without clinical symptoms. The authors point out that the early measurement of WBV may help to assess the risk of stroke.

4. Whole Blood Viscosity in Acute Ischemic Stroke

4.1. WBV at the Onset of AIS and During Its Follow-Up

Most investigations concerned the significance of WBV and other hemorheological parameters in acute ischemic stroke [22,24–33] (Table 1).

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Table 1. Key observations on Whole Blood Viscosity (WBV) changes in Ischemic stroke groups.

Authors	Ischemic stroke groups	Hemo-rheo-logical tests	Venepuncture and measurement time after AIS onset	WBV Measurement method/ Device Shear rate	Key observations
Coull B. et al. [25]	AIS,TIAs, stroke risk group	WBV PV	≤ 1 h after venepunctute with EDTA	Contraves LS viscometer; Ostwald microviscometer 0.145-124 s^{-1} at 25 ^{0}C	Increased WBV and PV Correlation with elevated FIB and albumin/globulin ratio
Fisher M. et al. [24]	AIS: large vessel, lacunar, cardiogenic	WBV, PV EA, ED	Within 72h with EDTA and 2 mos later Up to 5 hrs testing	Wells-Brookfield Micro Cone-Plate Viscometer 75 s^{-1} – 1500 s^{-1}, at 25^{0}C WBV at 40% Hct Zeta sedimentation ratio Centrifugal deformability technique	Increased WBV, PV and EA Increased WBV for cardiogenic and lacunar AIS with a trend for decrease after 2 months
WongWJ.[26]	AIS	WBV, PV, Ht,EA,ED	After 3 wks and 3 mos	Brookfield Cone-Plate viscometer at 35^{0}C	Increased BV, PV and EA, persisting on 3rd wk and 3 mo
Tsuda Y. et al. [22]	SI; AIS – lacunar Chronic lacunar -12.5 mos after AIS	WBV, PV	Within 3 days of onset and after 1 month	Cone-plate viscometer WBV corrected to 45% Ht, 22.5 s^{-1}-225.0 s^{-1}	Increased WBV and PV in acute LI; increased WBV after 1 mo and in chronic LI
Kowal P. et al. [27]	AIS Chronic IS – 3-6 mos after AIS	PV, Rel. BV=WBV/PV, Shear stress	Within 12h after onset with EDTA Up to 5min testing	Rotational-oscilatory reometer Contraves LS40 at 37^{0}C 0.01 s^{-1}– 100 s^{-1}	Increased relative BV in AIS and less pronounced in chronic IS. Increased PV in both groups.
Tikhomirova I et al. [28]	AIS	WBV, PV EA,ED	4h after venepunctue in heparinized tubes	Capillary viscometer with optoelectronical detection of flow, Microscope with digital camera, Micropore filtration system	Increased BV, PV and EA, decreased ED. BV correlated with the microcirculatory parameters of the upper forearm.

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Furukawa K. et al. [29]	AIS: CE, LAA, SAO	WBV	0.3 ml sample with EDTA on admission day, after 1wk, 2wks	Electromagnetic spinning sphere viscometer (EMS) at 37^{0}C, 100 s^{-1}	Significantly increased BV in SAO.The increased BV in SAO, CE and LAA is reduced on the 1st wk and increased on the 2nd wk (contribution of dehydration).
Song SH et al. [30]	AIS: CE,LAA, SAO, Cryp, stroke mimic	SBV DBV	3ml sample with EDTA within 3 days of onset after 1wk, 5wks Up to 24h testing	Scanning capillary tube viscometer (BVD-PRO1) 1 s^{-1} 300 s^{-1}	DBV highest in SAO. It decreased on the 1wk and increased on the 5wk (contribution of dehydration). DBV correlated with the number of chronic lacunes on MRI.
Hashem S. et al. [40]	AIS	BV Hct	Within 24h from onset before IV infusion	Ostwald glass capillary viscometer	BV was not a significant predictor of AIS outcomes BV correlated with the size of cerebral infarction on MRI
Han S. et al. [43]	LI	SBV DBV	Within 5 days of AIS onset before IV infusion Up to 24h testing	Scanning capillary viscometer (Hemovister) 1 s^{-1} 300 s^{-1}	Higher DBV at admission is associated with increased risk of progressive stroke in men.
Guawali Pet al. [3]	AIS: CE,LAA, SVO, UD	WBV	5 ml EDTA sample before treatment Up to 2 h testing	Brookfield DVII viscometer with CP40 spindle at 37^{0}C; WBV adjusted to 40% Ht; 20s^{-1}	Higher WBV in CE and UD AIS. Correlation of WBV with CT perfusion parameters and MRI DWI volume.
Lee H. et al. [41]	LI	SBV DBV	Sample with EDTA within 24h	Scanning capillary viscometer (Hemovister) 5 s^{-1} 300 s^{-1}	Increased DBV is associated with early neurological deterioration of LI in the anterior circulation.
Kang J. et al. [31]	AIS before and after IV fluid,hemor-rhagic stroke, stroke mimics	WBV	2 ml sample without anticoagulants Up to 3 min testing	Parallel plate rheometer 1, 5, 10 rad/sec (oscillation)	Increased WBV when compared to stroke mimic group and AIS after IV fluid
Oh J. et al. [38]	Undetermined AIS : thrombotic (UND-AT) or embolic (UND-E)	SBV DBV	Before hydration therapy Up to 24h testing	Scanning capillary viscometer (Hemovister) 1 s^{-1} 300 s^{-1}	Association of increased SBV and DBV with UD-AT

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Woo HG et al. [39]	AISwith >50% stenosis of the MCA and in situ thromboocclusion (IST), artery to artery embolism (AAE),local branch occlusion (LBO) according to MRI DWI topography	HSV LSV	6 ml with EDTA within 24h with EDTA Up to 24h testing	Scanning capillary viscometer (Hemovister) 5 s^{-1} - 300 s^{-1}	Blood viscosity was highest in patients with MCA - IST, followed by MCA-AAE and MCA-LBO. Patients with early neurological deteriorarion (END) had higher LSV and HSV. The association between END and LSV was higher in patients with MCA-LBO.
Lee M. et al. [42]	AIS: CE, LAA, SVO, OD	SBV DBV	3ml sample with EDTA prior to IV infusion Up to 24h testing	Scanning capillary tube viscometer (BVD-PRO1) at 36±0.5 ^{0}C 1 s^{-1} 300 s^{-1}	Increased DBV is associated with poor 3-mo functional outcome
Uygun G. et al. [32]	AIS: CE, LAA, SVO, UND	WBV, PV EA, ED	Sample with EDTA within 3 days after onset 4h to 24h testing	Brookfield DVIII viscometer at 37^{0}C 4.5 s^{-1} - 450 s^{-1} Laser ektacytometer (LORRCA)	No changes of BV and PV. Increased EA.
Noh S.-M. [33]	AIS: CE, LAA, SVO, UND	SBV DBV	3ml sample with EDTA before hydration therapy	Scanning capillary viscometer (Hemovister) 5 s^{-1} 300 s^{-1}	Higher DBV in SVO and in old LI and microbleeds on MRI. Association of DBV with SBV, age, C-reactive protein and hypertension.
Okumura M. et al.[36]	AIS:wake-up stroke CE, LAA, SVO, OD, UD	BV Hct	Blood samples within 72h from onset	Calculation of BV using Ht values	Higher BV was associated with wake-up stroke in elderly (>65years) in the SVO group.

Abbreviations: AIS, acute ischemic stroke; BV, blood viscosity; CE, cardioembolic; Cryp, cryptogenic; DBV, diastolic blood viscosity; EA, erythrocyte aggregation; ED, erythrocyte deformability; EDTA, ethylendiaminetetraacetic acid; Ht, hematocrit; IS, ischemic stroke; IV, intravenous; LAA, large artery atherosclerosis; LI, lacunar infarction; MRI DWI, diffusion-weighted magnetic resonance imaging; OD, other determined; SAO, small artery occlusion; SBV, systolic blood viscosity; SI, silent infarction; SVO, small vessel occlusion; TIAs, transient ischemic attacks; UND, undetermined; WBV, whole blood viscosity.

The measurement of WBV was usually performed within 12 to 72 hours after the stroke symptom onset and the following examinations were within 5 days to 3 months after the acute stroke treatment. Some of the studies included patients with transient ischemic attacks (TIAs) [24,34,35]. In addition to WBV other hemorheological variables like plasma viscosity, erythrocyte aggregation and erythrocyte deformability were examined [24,25,32] and their association with the fibrinogen values was estimated [24–26,30,34].

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In a great number of the acute stroke cases the WBV values during the onset of stroke were significantly increased in comparison to the control groups [22,24–31] except for the studies of Uygun et al. [32] and Noh et al. [33]. In all studies where erythrocyte aggregation and erythrocyte deformability were estimated significant increase of erythrocyte aggregation was detected [24,26,28,32]. Regardless of the time of the follow-up measurement, which was on the 5th day [32], the 1st week [30], the 3rd week [26], the 1st month [22], 2nd month [24] or 3rd month [5,26] a trend to normalization or significant decrease of the initial BV values was observed. Only the study of Wong et al. [26] revealed persisting increase of WBV after 3 months. Song et al. [30] who found subsequent increase of WBV on the 5th week after the initial decrease and they supposed that the decrease of WBV during the 1st week might be related to applied hydration therapy. Some authors pay special attention to the existing dehydration in part of the patients at the onset of AIS and its association with increased WBV [29,30,36] and they propose the investigation of the blood urea nitrogen/creatinine ratio as a marker of the dehydration state.

Most of the authors described the examined hematological and biochemical variables in the patients with AIS. However, few of them were mentioned to correlate with WBV: fibrinogen values and albumin/globulin ratio [25], WBC count, platelet count and HbA1c [29], C-reactive protein [33].

4.2. Association of WBV with Stroke Risk, Etiology and Imaging

In the clinical studies of the patients with AIS the frequency of the risk factors was presented. However, only Noh SM [33] reported significant association of diastolic and systolic blood viscosity with hypertension and age.

An attempt was made to compare WBV values in the etiological subgroups of acute ischemic stroke according to the TOAST classification [37]. When estimating the hemorheological variables in the different etiologic subtypes of stroke predominant increase of WBV in the group with small vessel occlusion (SVO) was reported [22,24,29,30,33,36], while other authors found it higher in stroke of cardioembolic (CE) and cryptogenic or undetermined (UND) etiology [6,24]. Also increased erythrocyte aggregation in the large-artery atherosclerosis (LAA) subgroups (24, 32] was found. Oh et al. [38] divided patients with stroke of undetermined etiology into groups with potential atherothrombosis, where systolic and diastolic blood viscosity values were higher than those in the group with possible embolism. Association of increased viscosity with imaging derived stroke mechanism in middle cerebral artery infarctions was revealed by Woo H. et al. [39] and it concerned the in situ thrombo-occlusion. As for the relationship of WBV with imaging biomarkers, DBV was found to be significantly higher in patients with old LI and microbleeds on MRI or CT [33] and to correlate with the number of chronic lacunes [30]. Also, significant correlation of WBV with the size of AIS [40] and with the CT perfusion parameters and MRI DWI was established [3].

4.3. WBV and AIS Prognosis

When estimating the prognostic value of WBV it was found that low-shear viscosity was related to early neurological deterioration in patients with in situ thrombo-occlusion and in those with artery-to-artery embolism and local branch occlusion of the middle cerebral artery [39]. Lee et al. [41] announced occurrence of early neurological deterioration was observed in 1.5% of patients, admitted for lacunar infarction. In them diastolic blood viscosity was significantly higher compared to patients without early neurological deterioration. In the same patients` group systolic and diastolic blood viscosity were associated with worsening of NIHSS score and progression of stroke [43]. Diastolic blood viscosity was also found to predict poor 3-month functional outcome in AIS patients [42]. Hashem et al. [40] failed to find relationship between blood viscosity and stroke outcome; however, they found significant correlation between WBV and the size of the cerebral infarction.

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5. WBV Measurement in AIS

During the years the changes of WBV in acute ischemic stroke were estimated with different viscometers and in some of the studies WBV was calculated by the De Simone formula [44] using the values of hematocrit and total protein. Earlier measurements were performed with rotational cone-plate [24–26] and recent studies by scanning capillary-tube viscometers [30,33,38,39,41,42] and parallel plate rheometer [31]. The reason is the need for point-of-care measurement after the patients` admission with small whole blood sample and rapid performance. The investigation in patients with AIS could help to assess the severity of the disease, predict the stroke outcome, to make decision for the treatment and estimate its effectiveness.

Formula-based viscosity (De Simone) offers quick, cost-effective estimation, but it assumes uniformity and doesn't reflect dynamic shear-thinning behavior of real blood (Table 2). Viscometer-based methods (rotational, capillary, EMS, oscillatory) provide actual viscosity values across shear rates, which is critical for clinical accuracy, especially for stroke management. No study in this text directly compared calculated vs. measured viscosity side-by-side in the same patient cohort, but such comparisons would be clinically valuable for validating formulas.

Table 2. Formula-Based vs. Instrument-Measured Blood Viscosity.

Aspect	**Formula-Based (e.g., De Simone)**	**Instrument-Based (e.g., Viscometer)**
Basis	Estimated using hematocrit (Hct) and total protein (TP)	Directly measured from whole blood using physical devices
Common Formula	HSR: (0.12 × Hct) + 0.17 × (TP − 2.07) LSR: (1.89 × Hct) + 3.76 × (TP − 78.42)	Not applicable
Sample Requirements	Only standard lab values (Hct, TP)	2–6 mL of fresh whole blood; sometimes anticoagulated (EDTA)
Time to Result	Immediate (once lab values available)	3–30 minutes depending on device and setup
Shear Rate Consideration	Static (208 s^{-1} or 0.5 s^{-1})	Dynamic; full shear profiles (e.g., 1–1000 s^{-1}, oscillatory modes)
Sensitivity to Pathology	Limited to changes in Hct & TP	Sensitive to RBC deformability, aggregation, temperature, real-time changes
Cost / Equipment Needs	Very low; no additional equipment	Medium to high; requires specialized viscometers (e.g., SCTV, EMS, Brookfield)
Clinical Use Case	Rapid estimation when viscometers unavailable	Diagnostic confirmation, stroke mimic differentiation, therapy monitoring
Limitations	Cannot capture non-Newtonian properties of blood	May be limited by device accuracy, operator variability, and processing time

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6. Whole Blood Viscosity and AIS treatment

Since several studies confirm the adverse effect of WBV in patients with cerebral ischemia stroke a question arises about its relationship with the ischemic stroke treatment modalities. Having in mind the contemporary guidelines for the treatment of acute ischemic stroke [45] the changes of WBV during different treatment strategies have been investigated.

6.1. WBV and Thrombolytic Therapy

The association between WBV and its determinants with thrombolytic therapy was investigated in the experimental study by M. Hitosugi et al. [46], who measured it in blood samples before and after mixing with recombinant tissue plasminogen activator (alteplase) at different concentrations. The author established that WBV`s initial increase was followed by a decrease and stabilised at a level below the initial values. A study of Rasyd AL.et al. [47] found an increase of WBV in 88.6% of the examined patients with AIS on admission. These patients had poorer neurological deficits on day 7 after intravenous thrombolysis and poor outcomes on day 30. This poor outcome was more pronounced in patients aged >65y old, dehydrated state and partial anterior circulation infarct. When estimating the markers of dehydration status, Li S. et al. [48] found that it is an independent risk factor for long-term prognosis of thrombolysed patients with acute ischemic stroke. The increased fibrinogen, determinant of WBV, was also shown to be associated with poor response in AIS at 24h [49] and 14 days [50] after intravenous thrombolysis. After adjustment for other stroke risk factors increased fibrinogen was identified as an independent factor for a poor response to thrombolysis [49]. The initial neutrophil-to-lymphocyte ratio also influenced this poor response.

6.2. WBV and AIS Endovascular Therapy

The outcome of another contemporary treatment option for AIS – endovascular thrombectomy is also associated with the initial WBV values in AIS patients with large cerebral artery occlusion. The elevated WBV at both high and low shear rates, calculated using De Simone`s formula, proved to be independent predictor for poor clinical outcome on day 90 after thrombectomy [51]. Also, the estimation of WBV was shown to influence the performance and hence the outcome after thrombectomy. In AIS patients with large artery occlusion high values of initial diastolic blood viscosity were associated with failure of first-pass reperfusion and need for more passages [52]. Another recent study in AIS patients` group did not reveal correlation between WBV at high and low shear rates and functional outcome of mechanical thrombectomy [53]. Similar to the WBV effects on the AIS clinical outcome after endovascular thrombectomy. increased fibrinogen on admission is associated with poor 3 -month outcome [54]. When following the examination of WBV, plasma viscosity, fibrinogen and hematocrit before and 3 months after combined treatment of AIS with intravenous thrombolysis and endovascular thrombectomy, Wu L. et al. [55] observed a significant reduction of their values as compared to those after intravenous thrombolysis alone. In patients with acute ischemic stroke receiving combined treatment with intravenous thrombolysis and endovascular thrombectomy the red blood cell fraction within the retrieved thrombi was proven to affect the thrombolytic response [56].

6.3. WBV and AIS Drug Treatment

Among the interactions of WBV with various therapies in AIS, its relationship with antithrombotic treatment in non-valvular atrial fibrillation is of special interest. When estimating the impact of prior antithrombotic use (antiplatelets, vitamin K antagonists and new oral anticoagulants) on WBV in cardioembolic stroke with non-valvular atrial fibrillation Jung et al. [57] indicated their significant association with decreased systolic and diastolic blood viscosity parallel with hematocrit values. Similar were the results of the same scientific team in a group of AIS and TIA patients with predominating lacunar stroke [58], where antithrombotic treatment included aspirin, clopidogrel and warfarin. When comparing the effect of the different antithrombotic drugs on WBV in acute

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cardioembolic stroke, warfarin showed greater reduction of WBV at all shear rates than aspirin [59]. The effect of the new oral anticoagulants did not show a difference compared to warfarin [57], and this is particularly favourable because of their convenience of use and high efficacy in reducing intracranial hemorrhage [60]. As for aspirin, its association with WBV showed different responses. Lee C. et al. [59]and Rosenson R. et al. [61] did not show changes of WBV after aspirin treatment. The response to aspirin was better when it was combined with clopidogrel [62], and this combination could provoke a significant reduction of WBV in AIS [57,58]. A positive influence of clopidogrel as an inhibitor of platelet function on the hemorheological profile was indicated [62,63]. In patients with ischemic stroke or transient ischemic attacks, clopidogrel was related to a decrease in systolic blood viscosity depending on their CYP2C19 genotype status [64] and lowering of blood viscosity could be due to the increase of adenosine and cyclic adenosine monophosphate plasma concentration [63].

During the years, the pharmacological agents aimed at influencing whole blood viscosity and improving cerebral blood flow are nimodipine, vinpocetine and pentoxifylline, among them pentoxifylline being the most extensively researched. Its properties to reduce fibrinogen, plasma and whole blood viscosity, to diminish erythrocyte and platelet aggregation and to increase blood filterability led to improvement of blood flow in the microcirculation [65]. A meta-analysis of studies in patients with acute stroke receiving intravenous and oral pentoxifylline revealed a strong trend toward reduction of early mortality [66]. Parallel decrease of blood viscosity with improved clinical outcome was reported by Rasyd A. et al [67], but the data again did not reach statistical significance. Another drug agent with possible hemorheological effect is vinpocetine, which has been shown to increase the elasticity of the erythrocyte membranes and their deformability parallel with lowering the degree of disability and cognitive functions in acute stroke [68]. Despite its promising effects there was not enough evidence to suggest it could reduce case fatality and its routing administration in patients with acute ischemic stroke was not recommended [69].

Regarding the calcium antagonist nimodipine, an experimental study revealed an improvement in brain energy metabolism and blood rheology with a significant decrease in WBV at high shear rate during ischemia and reperfusion in the gerbil brain [70]. A similar beneficial effect of nimodipine on plasma fibrinogen concentration and blood viscosity at low shear 3 weeks after acute ischemic stroke was found by Ameriso et al. [71]. In addition to its hemorrhagic effects, nimodipine has been discussed for reducing vasospasm and microthromboembolism, increasing fibrinolytic activity, and neuroprotection; however, it has not been proposed for the treatment of acute stroke due to the drug-induced significant decrease in systolic and diastolic blood pressure with subsequent poor outcome [72].

7. Conclusion

The analysis of the data on the changes of WBV in patients with AIS confirm its significance for the development and the outcome of the disease. Due to the different methods of measurement of WBV, the different time intervals of the patients`examination and the various associations of WBV with some of the applied treatment strategies, further investigations are necessary.

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Conflicts of Interest: The authors declare no conflict of interest

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