Evaluation of Acute Ischemic Stroke Patients Treated with Intravenous Thrombolysis; Experiences of a Stroke Center

İнtravenous Trombolitik Tedavi Verilen Akut İskemik İnme Hаstalarının Değerlendirilmesi; Bir İnme Merkezinin Deneyimleri

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Abstract

INTRODUCTION: Intravenous thrombolytic (iv-tPA) treatment is a recommended treatment in acute ischemic stroke (AIS). We aimed to evaluate the effectiveness and side effects of this treatment on our patients.

METHODS: We retrospectively evaluated the datas of patients with AIS treated with 0.9 mg/kg iv-tPA between 2018-2020.

RESULTS: Forty-nine patients were treated with iv-tPA between 2018 and 2020. NIHSS scores at 24th hour were found significantly decreased compared with at onset(8.3±4.1 at onset, 4.9±5.1 at 24th hour, p=0.000). Thirty-eight (97.7%) patients had good clinical outcome. Intracerebral hemorrhage (ICH) was spotted in 3 (6.1%) patients. Systolic blood pressure (ICH group: 141±25 mmHg, non-ICH group: 124±45 mmHg, p=0.007) and mean door-needle time (ICH group: 125±9 min., non-ICH group: 137±54 min, p=0.029) were found significantly increased in patients with ICH. Total 4 (8.2%) patients died after iv-tPA treatment.

DISCUSSION AND CONCLUSION: Our study showed that iv-tPA improves the clinical findings of AIS. Additionally high serum glucose level and systolic blood pressure may increase the risk of ICH.

Keywords: ischemic stroke, intravenous thrombolytic, alteplase

Öz

GİRİŞ ve AMAC: İntravenous trombolitik (iv-tPA) tedavi, akut iskemik inme hastalarının tedavisinde önerilen bir yöntemdir. Bu çalışmamızda amaçımız iv-tPA’nın hastalırmızdaki etkinliğini ve yan etkilerini incelermektir.

YÖNTEM ve GEREÇLER: Merkezimizde 2018-2020 yılları arasında 0,9mg/kg dozunda iv-tPA alan hastaların verileri retrospektif olarak incelendi.

BULGULAR: Merkezimizde 2018-2020 yılları arasında 49 hastanın iv-tPA ile tedavi edildiği saptandi. Iv-tPA ile tedavi edilen hastalarnın 24. saat NIHSS skorlarının tedavi öncesi NIHSS skorlarına göre belirlen dişlik olduğu izlendi(tedavi öncesi 8,3±4,1; 24. saat 4,9±5,1, p=0,000). Hasta пояlar ve (%)’de dönemde iyi klinik prognozun olduğu izlendi. % (6,1) hastada intraserebral kanama olduğu izlendi. Intraserebral kanama olan hastalarda sistolik kan basıncının(kanama olanlar: 163±59 mmHg, kanama olmayanlar: 141±25 mmHg p=0,014), serum glukoz düzeyinin(kanama olanlar: 173±117 mg/dl, kanama olmayanlar: 124±45mg/dl p=0,007) ve kapsigi-çap sürelerinin(kanama olanlar: 125±9 dk, kanama olmayanlar: 137±54 dk p=0,029) daha yüksek olduğu izlendi.

TARTIŞMA ve SONUÇ: Çalışmamız iv-tPA’nın akut iskemik inme hastalarının klinik bulgularını düzeltiltikçe gösterdi. Ayrıca yüksek serum glukoz düzeyi ve sistolik kan basıncının da intraserebral kanama riskini artırdığı izlendi.

Anahtar Kelimeler: iskemik inme, intravenous trombolitik, alteplaz
INTRODUCTION

Intravenous thrombolytic (iv-tPA) treatment is a recommended treatment in acute ischemic stroke (AIS). In 1995, National Institute of Neurological Disorders and Stroke study group reported that patients with acute ischemic stroke who were treated with alteplase 0.9mg/kg within 3 hours after onset of symptoms had better disability rates(1). ECASS III study showed that iv-tPA had benefit on patients treated 3-4.5 hours after onset of stroke symptoms(2). In American Hearth Association/American Stroke Association (AHA/ASA) guidelines, iv-tPA was recommended for patients who may be treated in 4,5 hours(3). Usage of iv-tPA in treatment of AIS spreads in our country. In this study, we evaluated the patients with acute ischemic stroke who were treated with iv-tPA.

MATERIAL AND METHODS

We retrospectively evaluated the datas of patients with AIS in Kocaeli Derince Training and Research Hospital between 2018-2020. This study was approved by Kocaeli Derince Training and Research Hospital Ethic Committee. Datas were collected from hospital records. Patients or their relatives were phoned for missing datas.

Iv-tPA treatment was applied patients who didn’t have any contraindication according to guideline of AHA/ASA (3). Iv-tPA was given as dose of 0,9mg/kg (maximum 90 mg) over 60 minutes with initial 10% given as bolus over 1 minute. All patients with acute ischemic stroke who were treated with iv-tPA didn’t have large vessel occlusion (LVO) (internal carotid artery or M1 segment of middle cerebral artery) in computarized tomography (CT) angiography were included to our study. Patients with large vessel occlusions were excluded, because they were treated with endovascular techniques.

Demographic datas, medical history (diseases, drugs,etc), laboratory findings (blood cell counts, glucose, kidney function tests, liver function tests, lipid profile, International Normalized Ratio (INR), hemoglobin A1c, etc), systolic and diastolic blood pressures at admission, symptom-needle time, symptom-door time, door-needle time, Alberta Stroke Program Early Computed Tomography Score (ASPECT)(4) on non-contrast brain CT at admission, National Institutes of Health Stroke Scale (NIHSS) score at admission and 24th hour, brain CT findings at 24th hour and modified Rankin score (mRS) at 90th day were collected. Symptom-needle time was defined as time from onset of symptoms to bolus injection of iv-tPA. Symptom-door time was defined as time from onset of symptoms to arrival to hospital. Door-needle time was defined as time from arrival to hospital to bolus injection of iv-tPA.

Intracerebral hematoma (ICH) types on brain CT at 24th hour were defined according to European Collaborative Acute Stroke Study (ECASS) classification (5). Subtypes of ischemic stroke were defined according to Trial of Org 10172 in Acute Stroke Treatment (TOAST) classification(6).

Statistical analyzes were made with SPSS 15.0. Categorical variables were expressed as frequencies and percentages. Continuous variables were expressed as mean (SD) or median (interquartilerange [IQR]) for non-normal distribution. Kolmogorov-Smirnov test was used for assessing the normality of distribution. We used Mann-Whitney U, paired T and independent T-tests for continuous data and χ² for binary and catarogical data. All p values <0,05 were considered significant.

RESULTS

We evaluated datas of 49 patients treated with iv-tPA between July 2018 and December 2020. Twenty-eight (57,1%) patients were male. Mean age of patients was found as 67,4±12,3 years (Male:68,5±12,5; female:66±12,2; p=0,985). Smoking was found high in males (p=0,04; OR:0,152, 95%CI 0,04-0,574). Hypertension (HT) was found increased in females (p=0,014; OR:11,11, 95%CI 1,292-95578). Other risk factors were found similar between male and female patients. Symptom-needle, symptom-door and door-needle times were found similar between males and females. Mean ASPECT score was found 9,6±0,7 points (Median:10, males: 9,7±0,6; females: 9,6±0,7, p=0,573). In laboratory findings, mean platelet count was increased and mean creatine level was decreased in female patients. Twenty-two (44,9%) patients had usage of antiagregants before stroke (17[34,7%] patients acetylsalicly acid (ASA), 2[4,1%] patients klopidogrel, 3[6,1%] patients ASA+klopidogrel. Atherosclerotic ischemic stroke was found high in male patients (p=0,015). Demographic, laboratory and clinical datas were shown in Table 1.

Mean NIHSS score at onset was found 8,3±4,1 points (Males: 9,1±4,6; females:7,1±3,2, p=0,131). Mean NIHSS score 24th hour was found as 4,9±5,1 points (males: 5,6±6,1, females:3,9±3,1, p=0,575). Mean NIHSS score of all patients decreased significantly at 24th hour(p=0,000, p=0,019 for males, p=0,007 for females). Changes in NIHSS scores were shown in Table 2.

ICH was seen in 3 (6,1%) patients on brain CT performed at 24th hour. One patient had petechial hemorrhage type 2 and 2 patients had parenchimal hematoma type 2. We found decreased symptom-door time (ICH group: 125±9 min., non-ICH group: 137±54 min. p=0,029) and increased door-needle time (ICH group: 73±37 min., non-ICH group: 37±9 min. p=0,009) in patients with ICH. Systolic BP was found high in patients with ICH(ICH group: 163±59 mmHg, non-ICH group: 141±25 mmHg p=0,014). In laboratory findings, only serum glucose levels were found increased in ICH group (ICH group: 173±117 mg/dl, non-ICH group: 124±45mg/dl, p=0,007).

Good clinical outcomes were seen in 38 (77,6%) of 49 patients. Distribution of mRS was shown in Table 3. Mean ASPECT score was found increased (p=0,037), and NIHSS score at 24th hour was found decreased (p=0,001) in patients with good clinical outcome. In laboratory findings, thyroxine (T4) levels were found high in patients with good clinical outcome(p=0,029). Ischemic stroke in medical history was found high in patients with poor clinical outcome (p=0,007; OR:21,143, 95%CI 2,046-218,497). Datas of patients with good and bad clinical outcome were shown in Table 4. Four (8,2%) patients died. Two (4,1%) patients died because of ICH, 1 (2%) patient died because of aspiration pneumonia and 1 patient died because of COVID-19 infection.
Table 1: Demographic, clinical, radiological and laboratory findings of male and female patients

|                         | Overall (%) | Male (%) | Female (%) | p     |
|-------------------------|-------------|----------|------------|-------|
| Patients                | 49          | 28(57,1) | 21(42,9)   |       |
| Age                     | 67.4±12.3   | 68.5±12.5| 66±12.2    | 0,985 |
| Hypertension            | 37(77,6)    | 18(64,3) | 20(95,2)   | 0,014 |
| Diabetes Mellitus       | 10(20,4)    | 4(14,3)  | 6(28,6)    | 0,291 |
| Smoking                 | 21(42.9)    | 17(60,7) | 4(19)      | 0,004 |
| Coronary Artey Disease  | 8(16,3)     | 6(21,4)  | 2(9,5)     | 0,438 |
| Hyperlipidemia          | 6(12,2)     | 3(10,7)  | 3(14,3)    | 1,000 |
| Stroke History          | 5(10,2)     | 5(17,9)  | 0(0)       | 0,062 |
| Atrial Fibrillation     | 14(28.6)    | 7(25)    | 7(33,3)    | 0,542 |
| Hearth Failure          | 3(6,1)      | 2(7,1)   | 1(4,8)     | 1,000 |
| Symptom-needle time (min)| 178±49      | 181±46   | 173±53     | 0,316 |
| Symptom-door time (min) | 135±51      | 141±45   | 128±59     | 0,143 |
| Door-needle time(min)   | 39±14       | 40±16    | 38±12      | 0,456 |
| ASPECT                  | 9,6±0,7     | 9,7±0,6  | 9,6±0,7    | 0,573 |
| NIHSS at onset          | 8,2±4,1     | 9,1±4,6  | 7,1±3,2    | 0,131 |
| NIHSS at 24th hour      | 4,9±5,1     | 5,6±6,1  | 3,9±3,1    | 0,575 |
| Cardioembolism          | 14(28.6)    | 7(25)    | 7(33)      | 0,542 |
| Atherosclerotic         | 7(14.3)     | 7(25)    | 0(0)       | 0,015 |
| Lacunar infarction      | 10(20.4)    | 5(17.9)  | 5(23.8)    | 0,726 |
| Undefined cause         | 18(36.7)    | 9(32.1)  | 9(42.9)    | 0,318 |
| WBC(/mm³)               | 8500±4109   | 9085±5050| 7719±2228  | 0,234 |
| Neutrophile %           | 62,5±14,7   | 61,8±16,8| 63,3±11,6  | 0,402 |
| Hemoglobin(gr/dl)       | 12,6±2,3    | 13,2±2,5 | 11,8±1,8   | 0,353 |
| MCV(fl)                 | 84,7±13,8   | 82,6±17  | 87,5±7,1   | 0,466 |
| Platelet(/mm³)          | 242408±74653| 225642±56179| 264761±90534| 0,029 |
| MPV(fl)                 | 8,99±0,9    | 9,1±0,95 | 8,84±0,83  | 0,647 |
| Glucose (mg/dl)         | 127±51      | 129±55   | 123±47     | 0,952 |
| Urea (mg/dl)            | 38,2±18,6   | 36,3±11,9| 40,8±25,1  | 0,505 |
| Creatine(mg/dl)         | 0,92±0,38   | 0,93±0,23| 0,90±0,53  | 0,019 |
| AST(U/L)                | 39,1±106,5  | 27,3±20,8| 54,9±161,8 | 0,169 |
| ALT(U/L)                | 28,6±73,2   | 18,4±10,3| 42,2±111,2 | 0,460 |
| GGT(U/L)                | 32,4±40,1   | 22,6±10,2| 45,3±58,3  | 0,123 |
| INR                     | 1,07±0,12   | 1,09±0,09| 1,06±0,15  | 0,294 |
| CRP(mg/l)               | 13,5±14,9   | 12,6±13,6| 14,8±16,7  | 0,418 |
| Triglyceride(mg/dl)     | 133,5±61,8  | 126,4±55,6| 143,3±70  | 0,409 |
| Total Kolesterol(mg/dl) | 189,1±42,8  | 183,6±43,1| 196,8±42,4 | 0,904 |
| HDL(mg/dl)              | 42,3±13,3   | 41,2±14,1| 43,7±12,4  | 0,827 |
| LDL(mg/dl)              | 121,3±35,9  | 118,9±38,8| 124,4±32,1| 0,364 |
| TSH(uU/ml)              | 2,78±6,23   | 3,11±7,87| 2,26±1,92  | 0,250 |
| T3(pg/ml)               | 2,57±0,46   | 2,62±0,52| 2,5±0,36   | 0,184 |
| T4(nge/ml)              | 1,22±0,22   | 1,18±0,19| 1,27±0,25  | 0,234 |
| HbA1c(%)                | 6,25±1,38   | 6,5±1,73 | 5,91±0,53  | 0,256 |
| Systolic BP(mmHg)       | 143±29      | 147±27   | 135±30     | 0,387 |
| Diastolic BP(mmHg)      | 81±15       | 83±16    | 77±14      | 0,236 |
| Antiaggregant           | 22(44,9)    | 13(46,4) | 9(42,9)    | 0,865 |
| Warfarin                | 3(6,1)      | 2(7,1)   | 1(4,8)     | 1,000 |
| ICH                     | 3(6,1)      | 3(10,7)  | 0(0)       | 0,250 |
| Good Clinical Outcome   | 38(77,6)    | 20(71,4) | 18(85,7)   | 0,311 |
| Mortality               | 4(8,2)      | 4(14,3)  | 0(0)       | 0,125 |


Table 2: NIHSS Score Changes Between at Onset and at 24th Hours

|                  | NIHSS at onset | NIHSS at 24th hour | p    |
|------------------|---------------|--------------------|------|
| Overall          | 8.2±4.1       | 4.9±5.1            | 0.000|
| Male             | 9.1±4.6       | 5.6±6.1            | 0.019|
| Female           | 7.1±3.2       | 3.9±3.1            | 0.007|

Table 3: Distribution of mRS at 90th day

| mRS | n(%)   |
|-----|--------|
| 0   | 10(20,4)|
| 1   | 18(36,7)|
| 2   | 10(20,4)|
| 3   | 6(12,2)|
| 4   | 1(2)   |
| 5   | 0(0)   |
| 6   | 4(8,2) |

**DISCUSSION**

In this study, we evaluated the data of ischemic stroke patients treated with iv-tPA who didn’t have LVO. We found that clinical findings of patients significantly recovered in 24 hours after treatment. Additionally, 77.6% of patients had functional independence at 90th day, and 8.2% of patients died due to ICH or infections. In a study, NINDS reported that 60% of patients had NIHSS≤3 points(1). In SITS-MOST study, functional independence (mRS≤2) rate of patients was found as 54.8%(7). In ECASS III study, favourable outcome was seen in 52.4% of patients treated with iv-TPA in 3-4.5 hours(2). Good clinical outcome was found as 62.7% in SITS-ISTR study(8). In a recent study, good clinical outcome at 3rd month was seen in 65% of patients in Turkey(9). Our good clinical outcome rate was found high from other trials. In AHA/ASA guideline published in 2015, endovascular treatment was considered in patients with LVO(10). In our center, patients with LVO are treated with mechanical thrombectomy. So patients with LVO were excluded from our study. But in these studies were designed before 2015, and patients with LVO were treated with only iv-tPA. But, recanalization rate was found as in 5.9% patients with distal ICA occlusion after iv-tPA alone in a trial(11). Exclusion of patients with LVO might increase the rate of good clinical outcome in our study.

Early admission of iv-tPA increases the efficacy of this treatment. Symptom-door time was found 157 minutes in SITS-ISTR trial(8). Our findings were similar to this trial. But mean symptom-door time was found low in two recent studies performed in Turkey(9, 12). Our hospital is only center treating AIS patients with iv-tPA and endovascular techniques in our region. Most of our patients came from other hospitals. Neurologists in these hospitals don’t want to treat AIS patients with iv-tPA due to not having experience it, and they send these patients to our hospital. This situation increased the symptom-door time of patients. Symptom-door time may be decrease by spreading the usage of iv-tPA by neurologists in other hospitals or transporting AIS patients to center where patient can be treated. In 2018 AHA/ASA guidelines, it’s recommended that patients with stroke findings should be transported to the closest center can administer iv-tPA(3). On the other hand, we have shorter door-needle time from other studies performed in Turkey(9,12). It’s recommended that initial bolus dose of iv-tPA should be given to ≥50% of AIS patients within <60 minutes after arriving to hospital (door-needle time <60 minutes) (3). We found mean door-needle time < 60 minute. Additionally 94% of patients had door-needle time <60 minutes. We found high door-needle time in patients with poor clinical outcome, but there wasn’t significant difference.

Our study showed that NIHSS score at 24th hour was significantly decreased in all patients. We found that NIHSS score at 24th hour decreased significantly in patients with good clinical outcome. Early neurological improvement after iv-tPA is accepted as predictor of recanalization and good clinical outcome(13). We found significant correlation between improvement in NIHSS score at 24th hour and good clinical outcome.

Our study showed that patients with good clinical outcome had high ASPECT scores. ASPECT score ≤7 points is predictor of poor clinical outcome(4). But there isn’t any information about association between ASPECT score and clinical outcome in patients treated with iv-tPA in literature. In a recent trial, Das et al reported that lower ASPECT scores were associated with increased risk of ICH(14).

ICH rate was found as 6.4% in NINDS trial(1). In SITS-MOST trial, ICH was seen in 2.2% of patients(7). Hackett et al reported that 2.4% of patients treated with iv-tPA within 3-4.5 hours had ICH in ECASS III trial(2). In a meta-analysis evaluating ATLANTIS, ECASS and NINDS trials, substantial ICH rate was found as 5.9%(15). Kutluk et al found ICH rate as 4.9% in a multicenter study performed in Turkey(9). We found similar ICH rates with literature. Patients with ICH had decreased sympathomotor and increased door-needle times. There isn’t any data about association between increased door-needle time and ICH. Cause of increased door-needle time might be prolonged treatment of high SBP levels. As known, iv-tPA is contraindicated in patient with SBP>180 mmHg and DBP>105 mmHg(3). These patients have resistant HT and we lost time while decreasing SBP and DBP.

Our study showed that increased SBP and glucose levels were associated with ICH after iv-tPA treatment. In previous studies, high levels of SBP were found as a predictor of ICH(16). In a recent trial, Nissar et al found increased SBP levels in patients with ICH(17). But there aren’t many studies about association between SBP and ICH after iv-tPA treatment. In same study, serum glucose levels>185 md/dl were found as a predictor of ICH. It isn’t known well how elevated serum glucose levels increase the risk of ICH. Mishiro et al reported that chronic hyperglycemia aggravated hemorrhagic transformation after ischemia-reperfusion injury by middle cerebral artery occlusion resulting with endotetial injury in diabetic mice(18). This can explain why high glucose levels increase the risk of ICH in AIS.

Our study had some limitations. Our study was designed as retrospectively. Our patient group was small and we didn’t have control group. We didn’t evaluated blood pressures during and after iv-tPA treatment.
Table 4: Demographic, Clinical, Radiological and Laboratory Findings of Patients With Good and Poor Clinical Outcome

|                        | Good Outcome(%) | Poor Outcome(%) | p     |
|------------------------|-----------------|-----------------|-------|
| n(%)                   | 38(77,6)        | 11(23,4)        | 0,311 |
| Male                   | 20(52,6)        | 8(72,7)         | 0,062 |
| Age                    | 64,7±12,2       | 76,8±6,9        | 0,415 |
| Hypertension           | 28(73,7)        | 10(90,9)        | 0,673 |
| Diabetes Mellitus      | 7(18,4)         | 3(27,3)         | 0,673 |
| Smoking                | 16(42,1)        | 5(45,5)         | 0,001 |
| Coronary Artery Disease| 5(13,2)         | 3(27,3)         | 0,355 |
| Hyperlipidemia         | 3(7,9)          | 3(27,3)         | 0,117 |
| Stroke History         | 1(2,6)          | 4(36,4)         | 0,007 |
| Atrial Fibrillation    | 12(31,6)        | 2(18,2)         | 0,475 |
| Hearth Failure         | 2(5,3)          | 1(9,1)          | 0,542 |
| Symptom-needle time (min) | 173±49         | 193±47          | 0,808 |
| Symptom-door time (min) | 133±54          | 145±42          | 0,354 |
| Door-needle time(min)  | 36±9            | 48±23           | 0,095 |
| ASPECT                 | 9,76±0,54       | 9,18±0,98       | 0,037 |
| NIHSS at onset         | 7,6±3,7         | 10,6±4,5        | 0,121 |
| NIHSS at 24th hour     | 3,2±2,2         | 10,5±7,8        | 0,001 |
| Cardioembolism         | 12(31,6)        | 2(18,2)         | 0,475 |
| Atherosclerosis        | 6(15,8)         | 1(9,1)          | 0,500 |
| Lacunary infarction    | 9(23,7)         | 1(9,1)          | 0,419 |
| Undefined cause        | 11(28,9)        | 7(63,6)         | 0,072 |
| WBC(/mm³)              | 8686±4569       | 7854±1790       | 0,222 |
| Neutrophile %          | 62,9±15,7       | 60,9±11,3       | 0,275 |
| Hemoglobin(gr/dl)      | 12,6±2,3        | 12,7±2,5        | 0,853 |
| MCV(fl)                | 84,3±14,8       | 86,2±10,3       | 0,755 |
| Platelet(/mm³)         | 240078±79435    | 250454±57547    | 0,776 |
| MPV(fl)                | 9,06±0,89       | 8,79±0,96       | 0,958 |
| Glucose (mg/dl)        | 124±47          | 135±64          | 0,657 |
| Urea (mg/dl)           | 37,7±20,4       | 40±10,9         | 0,134 |
| Creatine(mg/dl)        | 0,91±0,42       | 0,97±0,29       | 0,250 |
| AST(U/L)               | 43±120,8        | 25±12,9         | 0,442 |
| ALT(U/L)               | 30,2±82,6       | 23±20,5         | 0,981 |
| GGTT(U/L)              | 30,5±41,7       | 39,9±34,7       | 0,449 |
| INR                    | 1,08±0,13       | 1,05±0,12       | 0,832 |
| CRP(mg/l)              | 14,9±16,3       | 9,81±8,7        | 0,346 |
| Triglyceride(mg/dl)    | 130,8±64,3      | 145,3±51,5      | 0,496 |
| Total Kolesterol(mg/dl)| 189,9±42,6      | 185,8±46,7      | 0,892 |
| HDL(mg/dl)             | 42,5±14,5       | 41,2±6,2        | 0,245 |
| LDL(mg/dl)             | 122,6±36,1      | 115,5±36,9      | 0,862 |
| TSH(μU/ml)             | 2,78±6,86       | 2,79±2,56       | 0,188 |
| T3(pg/ml)              | 2,61±0,46       | 2,41±0,48       | 0,748 |
| T4(ng/ml)              | 1,25±0,24       | 1,13±0,11       | 0,029 |
| HbA1c(%)               | 6,26±1,45       | 6,24±1,10       | 0,340 |
| Systolic BP(mmHg)      | 136±23          | 170±34          | 0,257 |
| Diastolic BP(mmHg)     | 78±14           | 90±18           | 0,125 |
| Antiaggregant          | 17(44,7)        | 5(45,5)         | 1,000 |
| Warfarin               | 3(7,9)          | 0(0)            | 1,000 |
| ICH                    | 1(2,6)          | 2(18,2)         | 0,122 |
CONCLUSION

Ivt-tPA is an effective and confident method in treatment of AIS patients without LVO. This treatment reduces the mortality and disability due to AIS. Administration by experienced centers may reduce side effects of iv-tPA. Increasing centers which can treat patients with iv-tPA will reduce negative effects of AIS on public. More studies are needed about this topic.

Ethical approval: Kocaeli Derince Training and Research Hospital Clinical Research Ethics Committee (11.03.2021 / 2021/26)
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Conflict of Interest: None.

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