ORIGINAL ARTICLE

CONTENTS

In vivo induction of different venous thromboembolism by use of DC electrical shock on jugular vein endothelium

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ABSTRACT

Aim: This study aimed to establish a novel rabbit model using direct electrical stimulation to induce internal jugular vein injury and subsequent organ-specific thrombosis.

Materials and Methods: Twenty-one male New Zealand White rabbits underwent jugular vein thrombosis induction *via* direct current (DC) electrical stimulation. D-Dimer assays confirmed thrombus formation. Histopathological analyses of the lungs, heart, and brain assessed thromboembolic changes. Thrombosis induction was tested at varying DC voltages and exposure durations: 6 V for 60 s, 12 V for 120 s, and 24 V for 180 s.

Results: In the lungs (6 V/60 s), thrombi exhibited characteristic lines of Zahn with dense platelet and fibrin deposition, alveolar wall destruction, emphysematous changes, and wedge-shaped hemorrhagic pulmonary infarctions. In the heart (12 V/120 s), thrombus formation was observed in coronary arteries, with interstitial hemorrhage and altered cardiac muscle fibers showing rounded and oval nuclei. In the brain (24 V/180 s), acute neuronal injury was evident, including shrunken eosinophilic "red neurons," cerebral edema, liquefactive necrosis, small-vessel hemorrhage, and astrocyte proliferation. These findings indicate voltage- and duration-dependent induction of thromboembolism with organ-specific distribution.

Conclusions: Direct DC electrical stimulation induces deep vein thrombosis in rabbits in a voltage- and duration-dependent manner. Lower voltage and shorter exposure primarily affect the lungs, moderate settings target the heart, and higher voltage with longer duration affect the brain. This model provides a controlled platform for studying organ-specific thromboembolism and evaluating therapeutic interventions, especially if laboratory tests related to coagulation parameters are applied, they may help in identifying the components of the formed clot. Similarly, using lower or higher electrical currents at different intervals may reveal more precise pathological changes, thus contributing to the application of more specialized pharmaceutical treatments..

KEYWORDS: venous thrombosis, pulmonary infarctions, thrombus formation

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INTRODUCTION

Thrombosis means the formation of blood clots within blood arteries and veins, which can block blood flow and lead to serious conditions such as a heart attack, myocardial infarction (MI), stroke, pulmonary embolism (PE), or tissue ischemia, depending on the thrombus site [1]. A common clinical condition is venous thrombosis (VT), with a significant financial burden on the US healthcare system. Despite its spread, VT diagnosis and management remain primitive [2]. Dysfunction in the endothelium has been associated with VT that activates the clotting system, with both thrombus types. White thrombus is rich in platelets and fibrin with few erythrocytes, and red thrombus consists of erythrocytes and less fibrin that is trapped in [3]. Deep vein thrombosis (DVT) and PE are called venous thromboembolism (VTE), which is a very common health problem [4]. Trauma is a powerful trigger factor for VTE, the leading cause of death worldwide [5]. Internal Jugular Vein thrombosis (IJV) is a potentially life-threatening disease due to the development of a localized ischemic stroke thrombus [6]. Animal models of thrombosis are crucial in pathophysiological research to understand the complex mechanisms of hemostasis and thrombogenesis, and to screen anticoagulant drugs. Numerous in vitro and in vivo models are routinely used in thrombosis research [7]. The experimental rabbit was employed as a model because the Coagulation factor (FX) profile in rabbit and human plasma has equivalent binding affinity to enzyme substrate and small–molecule with FXa inhibitors [8]. Electrical currents that are implemented in animals, e.g., electrical stunning during slaughter or electro-cauterization as a surgical tool, or accidentally when animals contact with electrical currents, can cause DVT in organs. The physiological impact is modulated by current, voltage, heat production, and the duration

of the electrical flow. Once introduced into the body, electrical currents travel the most direct path from the contact source to the grounding or exit site [9].

MATERIALS AND METHODS

EXPERIMENTAL ANIMALS

Twenty-one healthy male New Zealand White rabbits, weighing between 3.0 and 3.5 kg, were utilized in this experimental study, kept in good health under standard laboratory conditions. During quarantine, animals undergo an acclimation period to adapt to the housing environment and routine management practices. Rabbits were maintained on standard feed with unrestricted access to water. All housing, handling, and experimental procedures adhered to the regulations of the Local Ethics Committee for Animal Experimentation (University of Basra, College of Pharmacy), under the affiliation of the National Ethics Committee for Animal Experimentation, Polish Ministry of Science and Higher Education. "In order to ensure adequate adaptation to the laboratory environment, rabbits were transported from the farm to the animal housing facility 14 days prior to the initiation of experiments [10].

LATEX AGGLUTINATION ASSAY

As the latex agglutination assay, D-Dimer latex (ATLAS Medical GmbH, Germany) was used pre- and post-experiment to be sure that platelet aggregation and the clot formation process began. Plasma is prepared by centrifugation of whole blood collected using a sodium citrate anticoagulant tube. Place 20 µL of the reagent within a field on the reaction slide. A 20 µL aliquot of undiluted plasma or control solution was accurately pipetted next to the Latex Reagent. The mixture was homogenized using a stirrer until uniform. The slide was mounted on a mechanical rotator operating at 80–100 rpm, and agglutination was examined under intense illumination exactly at 3 minutes.

SURGICAL PROCEDURE

All experimental rabbits were pretested with D-Dimer test (a protein that breaks down blood clots) before performing any surgical procedures, to be sure that all animals didn't have any coagulation problems. Anesthesia was induced and maintained using ketamine (50 mg/kg initial dose, followed by 50 mg/kg/h, intramuscularly) in combination with xylazine (10 mg/kg initial dose, followed by 10 mg/kg/h, intramuscularly).

After achieving adequate anesthesia, the fur in the cervical region was carefully shaved. The rabbit was subsequently transitioned to isoflurane anesthesia, and vital parameters—including respiratory rate, blood oxygen saturation, and heart rate—were continuously monitored.

An incision was made in the shaved cervical region to expose and carefully isolate the jugular vein from surrounding tissues. Thrombosis was induced by electrical stimulation of the left jugular vein at different times as shown in Table 1, and Fig. 1 with a DC electrical supply (3-24V, 3A,72W) using a stainless-steel probe [11-12].

As shown in Figures 2, and 3 the jugular veins were carefully exposed by dissecting the fascia and exposing and preparing the sternocleidomastoid muscle. To accelerate the thrombus formation, wet the leg that is tied with a negative charge probe. The vein is exposed simply by placing the positive electrode on the external surface of the jugular vein. As a result of the electric current passing through the body, which leads to the induction of trauma in the venous endothelium, it is noticeable as a result of the color change of the vein surface to white. Its intensity depends on the voltage of the current and the period of exposure. Suturing of the incision and treatment with antibiotics were applied to prevent contamination.

HISTOPATHOLOGICAL EXAMINATION

Primary fixation by perfusion of an entire animal with normal saline solution is performed by pumping it via the left ventricle (IV). The fixative arrives at and fills the capillary net, where most of the perfusion occurs by allowing the fixative solution to leave the body, following preservation in 10% formaldehyde, kidney and lung specimens were embedded in paraffin wax. Sections of 3 µm were obtained using a rotary microtome. Histological evaluation was performed after staining the sections with hematoxylin and eosin (H&E).

RESULTS

LUNG

Characteristic alternating layers of erythrocytes and platelets/fibrin, termed the lines of Zahn, are observed during thrombus formation. A thromboembolus (TE) introduced into the pulmonary artery may, over time and with survival of the rabbit, undergo both structural organization and dissolution. The clot formation can occur in a blood vessel near the shock, and if the clot travels through the bloodstream, it may end up getting trapped in the pulmonary artery, leading to a pulmonary embolism. Additionally, the lung is a receptive location for clot formation due to slower blood flow.

Table 1. Mean ± SD values according to agglutination test results for Lung, Heart, and Brain under different voltages and exposure times (P<0.05 considered significant)

Organ	6V 60s	6V 120s	6V 180s	12V 60s	12V 120s	12V 180s	24V 60s	24V 120s	24V 180s
Lung	2.29±0.90*	2.86 ± 0.38	3.00±0.00	3.00±0.00	2.86±0.38	2.86±0.38	2.86±0.38	2.86±0.38	3.00±0.00
Heart	4.00±2.16*	0.43±0.53*	0.57±0.53*	1.14±1.05*	2.57±0.53*	2.29±1.13*	0.29±0.49*	3.00±0.00	3.00±0.00
Brain	2.57±0.79	2.86±0.38	3.00±0.00	3.00±0.00	3.00±0.00	3.00±0.00	3.00±0.00	3.00±0.00	3.00±0.00*

Source: Compiled by the authors of this study

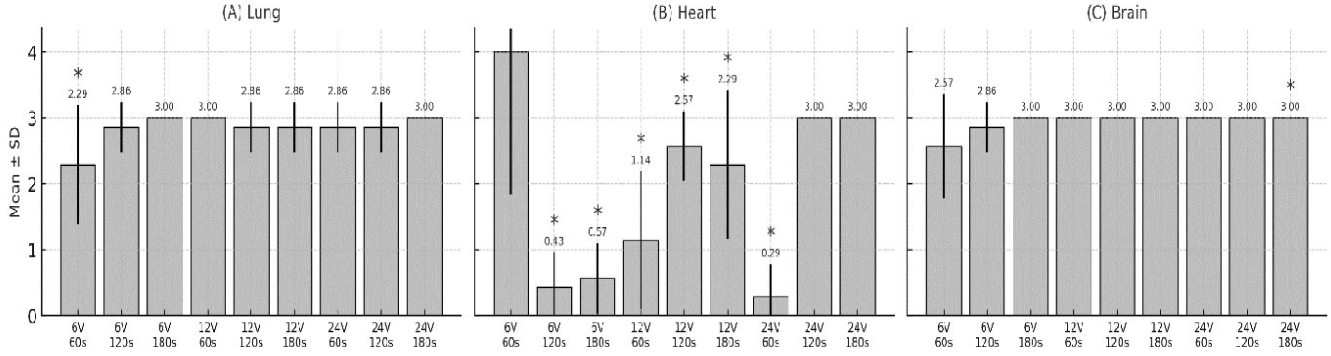

Fig. 1. Effect of voltage and exposure time on organ responses measured by agglutination test. Mean ± SD are shown for lung **(A),** heart **(B)**, and brain **(C).** Significant differences (P < 0.05) are indicated by an asterisk (*). Each bar represents the mean of three independent experiments
Source: Own materials

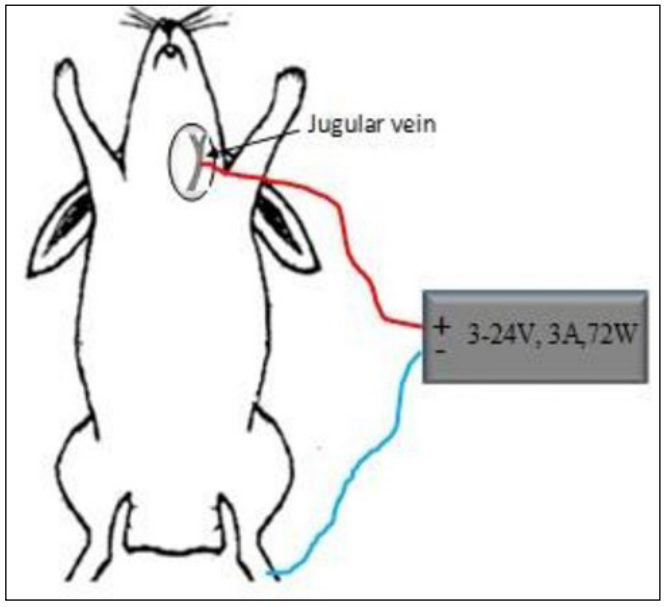

Fig. 2. Rabbit neck incision exposing the jugular vein. An electrode placed on the vein is connected to a DC source to deliver an electrical shock and induce thrombosis
Source: Own materials

As illustrated in Figure 4, the thrombus is adherent to the arterial wall, with characteristic lines of Zahn indicated by the arrow. At high magnification, microscopic examination reveals destruction of alveolar walls accompanied by emphysematous changes, with remaining airspaces markedly dilated. Hemorrhagic pulmonary infarctions are wedge-shaped and sub pleurally based, reflecting the survival of the tissue. These infarcts are hemorrhagic because, despite obstruction of the pulmonary artery - which supplies the majority of blood and oxygen to the lungs - the bronchial arteries from the systemic circulation, providing approximately 1% of pulmonary blood flow, remain patent.

HEART

Cardiac muscle fibers running in different directions with an acidophilic cytoplasm and central pale oval nuclei, with areas of interstitial hemorrhage and rounded or square nuclei, and some rounded nuclei (Fig. 5).

BRAIN

Shrunken, eosinophilic neurons (also known as "red neurons") badge of acute neuronal injury. Cerebral edema and liquefactive necrosis with hemorrhage in small vessels and Astrocyte proliferation were noticed (Fig. 6).

DISCUSSION

The present study demonstrated that electrical current can induce thrombus formation in multiple organs, including the lungs, heart, and brain, with the severity and location of thrombosis being dependent on both voltage intensity and duration of exposure. These findings support previous hypotheses suggesting that electrical injury can directly damage vascular endothelium, thereby promoting thrombogenesis.

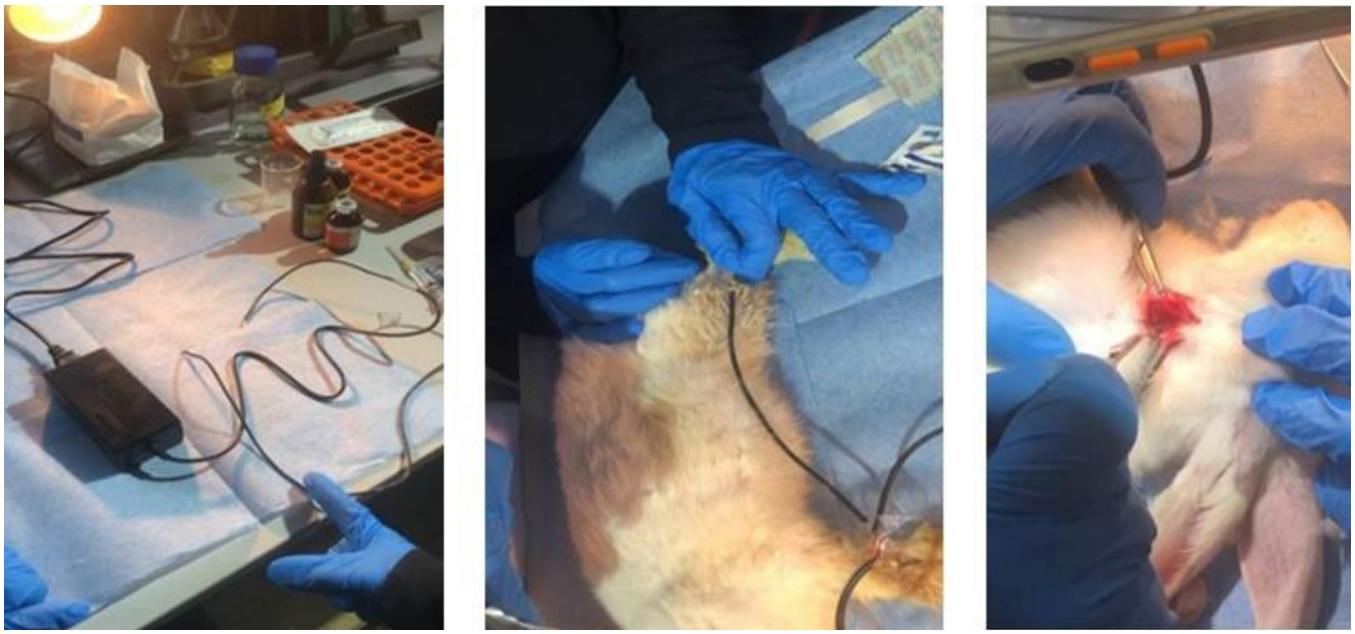

Fig. 3. Intraoperative view demonstrating surgical exposure of the rabbit's jugular vein. A stainless-steel electrode is applied directly to the jugular vein and connected to a DC power source to induce endothelial injury and thrombus formation via controlled electrical stimulation
Source: Own materials

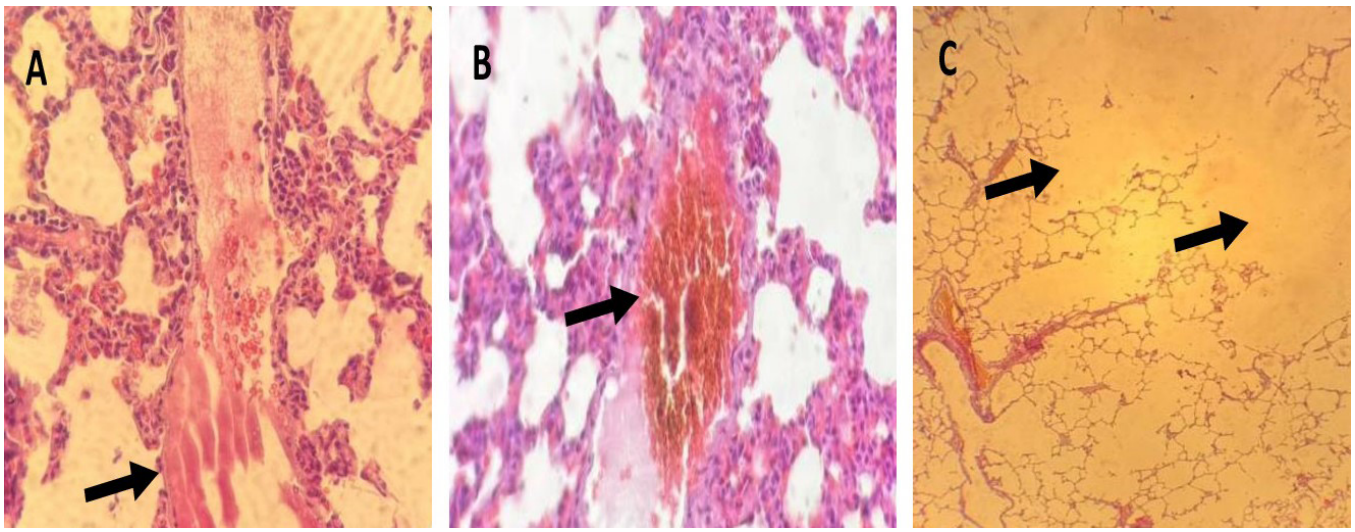

Fig. 4. Lung histology from 6 voltages for 60 sec. **(A):** Show thick white and red line of Zahn with condense of platelets deposition all over thrombus (H&E stain ×40). **(B):** Loss of alveolar walls with emphysema (H&E stain ×20). **(C):** Clear hemorrhagic pulmonary infarction in bronchial artery (H&E stain x 40)
Source: Own materials

In the lungs, significant thrombus formation was observed following relatively low-voltage exposure, accompanied by pulmonary infarction and vascular congestion. Similar mechanisms have been proposed in clinical studies where direct electrical injury to the vascular endothelium increased the risk of deep venous thrombosis (DVT), particularly during the early period following electrical exposure [13]. Furthermore, evidence-based reviews of electrical injuries have suggested that both thermal and electrical damage to endothelial cells contribute to venous and arterial thrombotic events [14]. The observed pulmonary thrombosis in the present study may therefore result from endothelial dysfunction combined with alterations in blood flow and local coagulation activity.

The findings of this study are also consistent with reports indicating that the severity of tissue and vascular injury increases with higher electrical current and voltage, regardless of whether alternating current (AC) or direct current (DC) is involved [15, 16]. Previous investigations have shown that electrical injuries cause heat-induced tissue destruction, vascular endothelial

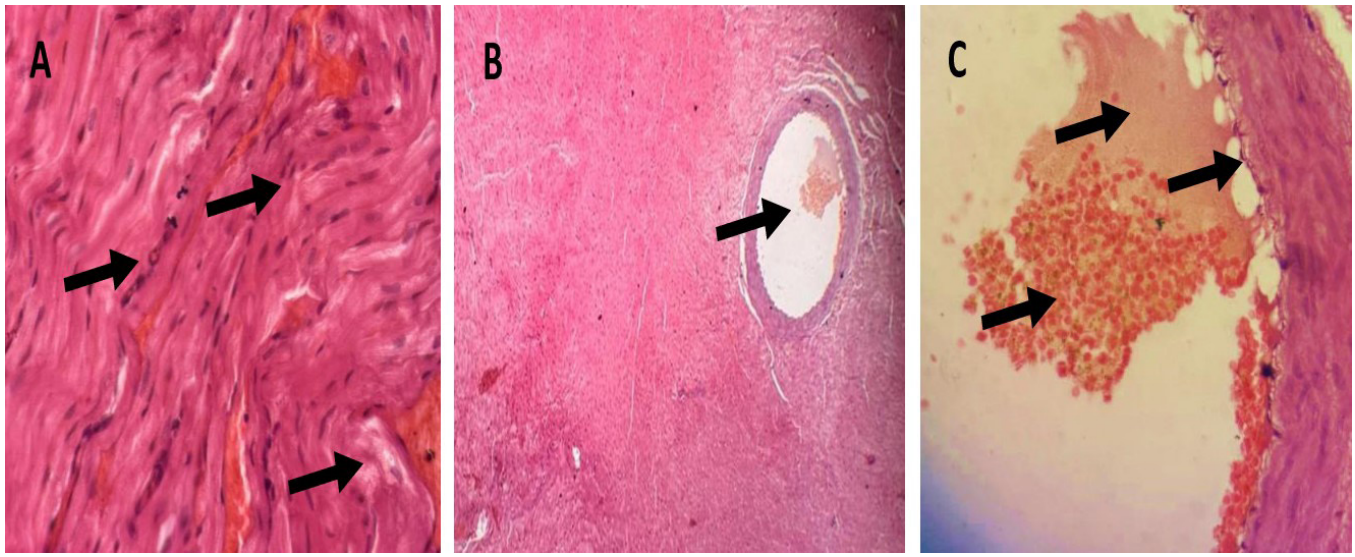

Fig. 5. Heart histology from 12V for 120 sec. **(A)**: Show different direction fibers with rounded and oval nuclei (H&E stain x 40). **(B&C)**: Show thrombus formation in the coronary artery with clear fibrin (H&E stain ×10 A; ×40 B)
Source: Own materials

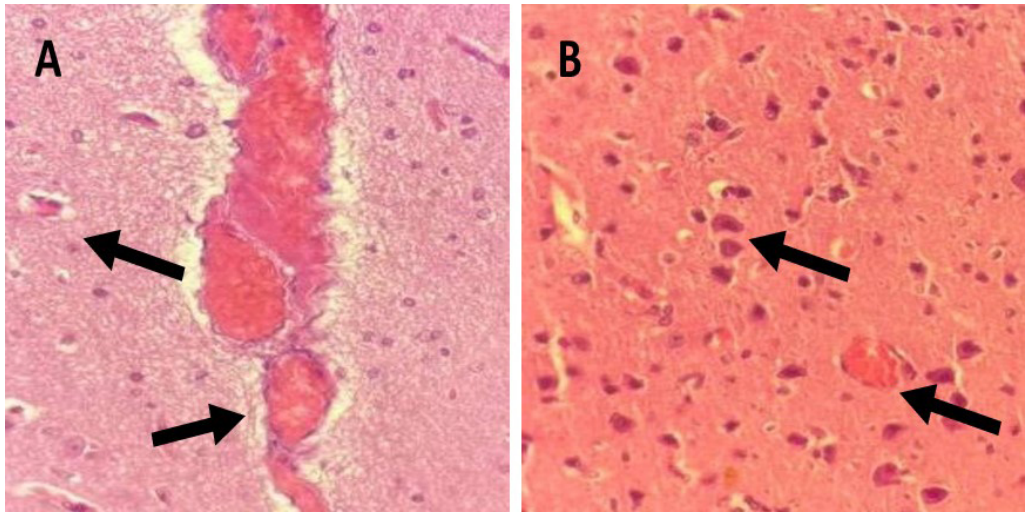

Fig. 6. Brain histology from 24V for 180 sec. (A): Shrunken, eosinophilic neurons with cerebral edema and liquefactive necrosis and Hemorrhage in small vessels with clear Astrocyte proliferation (B) Cerebral cortex demonstrating acute ischemic neuronal injury ("red neurons"), characterized by shrunken eosinophilic neurons with pyknotic nuclei, consistent with early hypoxic–ischemic brain damage (H&E stain ×10 A; ×40)
Source: Own materials

damage, and vessel thrombosis, supporting the pathological findings observed in the current experimental model [17].

In the heart, coronary artery thrombosis and fibrin deposition were observed at intermediate voltage and exposure duration. These findings correlate with several clinical reports describing coronary vascular injury after electrical exposure. Jacob et al. documented a case of coronary artery thrombosis and dissection following a 6,000-volt electrical shock, suggesting that electrical current can directly damage coronary vessels and trigger thrombotic events [18]. Similarly, comprehensive reviews of electrical cardiac injuries have highlighted arrhythmias, myocardial infarction, and coronary thrombosis as important consequences of electrical exposure [19]. Additional case reports described acute myocardial infarction with refractory cardiogenic shock after high-voltage electrocution [20], while delayed ventricular dysfunction and myocardial fibrosis have also been reported in survivors of severe electrical injuries [21]. Moreover, multiple cases of coronary thrombosis requiring surgical intervention following electric shock have been documented, further supporting the relationship between electrical injury and coronary vascular thrombosis [22].

In the brain, prolonged exposure to higher voltages resulted in marked neuronal injury, cerebral edema, hemorrhage, and vascular thrombosis. These findings are in agreement with several reports describing cerebrovascular complications after electrical injury. Huan-Jui et al. reported an acute ischemic stroke following low-voltage electrical exposure and suggested that vasospasm may play a major role in the pathogenesis of cerebral ischemia [23]. Patel et al. described cerebral venous thrombosis after accidental electrocution, emphasizing that thrombotic events may occur even when

the brain is not directly within the electrical pathway [24]. Likewise, cerebral infarctions involving the frontotemporal and parietal regions have been reported following low-voltage electrical injury [25], while acute ischemic stroke after high-voltage electrocution has also been documented [26]. These observations support the current findings and indicate that electrical injury may produce both direct vascular damage and secondary thrombotic complications within the central nervous system.

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CONCLUSIONS

Overall, the present study demonstrates a clear relationship between electrical exposure and thrombus formation across multiple organ systems. The experimental findings suggest organ-specific susceptibility, with pulmonary thrombosis occurring at lower electrical settings, coronary thrombosis developing at intermediate exposure levels, and cerebral vascular injury appearing predominantly after higher voltage and prolonged exposure. These results support existing clinical and experimental evidence indicating that electrical injury can induce thrombosis through endothelial damage, thermal injury, vascular dysfunction, and coagulation activation. Consequently, this rabbit model provides a reproducible platform for investigating the mechanisms of electrical injury-induced thrombosis and may facilitate the development of preventive and therapeutic strategies for thromboembolic complications associated with electrical shock.

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CONFLICT OF INTEREST

The Authors declare no conflict of interest

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