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Intelligentvascularmedicine:Artificialintelligence,Precisiontherapeutics,And Regenerative Innovations In Coronary Artery Disease,Deep Vein Thrombosis,And Stroke

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ABSTRACT

Coronary artery disease (CAD), deep vein thrombosis (DVT), and stroke represent interconnected vascular disorders that remain leading causes of global morbidity and mortality. Despite affecting distinct vascular territories, these conditions share common pathophysiological mechanisms, including endothelial dysfunction, systemic inflammation, and hypercoagulability. Conventional management strategies—comprising antiplatelets, anticoagulants, statins, antihypertensives, revascularization procedures, and lifestyle modification—have significantly improved survival; however, substantial residual risk persists. Recent advances in artificial intelligence (AI) are reshaping vascular medicine through enhanced risk prediction, automated imaging interpretation, and real-time clinical decision support systems. AI-driven models facilitate early diagnosis, optimize anticoagulation strategies, improve stroke triage for reperfusion therapies, and refine plaque characterization in CAD. Emerging translational research, including regenerative therapies, nanotechnology-based drug delivery, and robotic-assisted interventions, further expands therapeutic possibilities. Integrating AI with precision medicine and interdisciplinary innovation offers a promising strategy to reduce disease burden and improve long-term cardiovascular and cerebrovascular outcomes.

KEYWORDS

Coronary artery disease; Deep vein thrombosis; Stroke; Artificial intelligence; Precision medicine; Thromboembolism; Risk stratification; Regenerative therapy; Nanotechnology; Clinical decision support systems.

I. INTRODUCTION

Coronary artery disease (CAD), the leading cause of death worldwide, is largely preventable. Clinical trials show that reducing major risk factors such as smoking, hypertension, and high cholesterol lowers the risk of myocardial infarction and death by 30–40%. Early genetic studies using microarrays with hundreds of thousands of single-nucleotide polymorphisms initially failed to identify strong associations, but later research linked more than 1,300 genetic variants to increased risk of over 160 diseases. [1] Deep vein thrombosis (DVT) affects about 67 per 100,000 people annually. Despite treatment, pulmonary embolism can be fatal in 1–8% of cases and may cause long-term complications such as post-phlebitic syndrome and chronic thromboembolic pulmonary hypertension. Although anticoagulants reduce recurrence, they increase bleeding risk. Before 1995, all suspected DVT cases underwent imaging, with repeat testing if results were negative. [2] Stroke is the third leading cause of death in Western countries and accounts for about 10% of global deaths. It results from blocked or ruptured brain blood vessels, leading to brain cell death and neurological disability. In 2001, the National Institutes of Neurological Disorders and Stroke formed the Stroke Program Review Group to address this major public health burden.[3]

EPIDEMOLOGY

Coronary artery disease (CAD) is mainly caused by atherosclerosis and remains a leading global cause of death. Although incidence has declined in developed countries due to better prevention and care, prevalence may rise with aging, immigration, and comorbidities. In

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developing nations, CAD is increasing because of Western diets and sedentary lifestyles, with outcomes influenced by social inequality and access to treatment. [4][5] Deep vein thrombosis (DVT) occurs in about 0.1% of the population annually, increasing with age. Venous thromboembolism has high morbidity, mortality, and recurrence, and distal clots may extend proximally, raising pulmonary embolism risk.[6] Stroke is a heterogeneous disorder caused by interrupted cerebral blood flow, leading to outcomes from recovery to death. Despite advances in imaging, its clinical definition remains unchanged to allow comparisons across regions, as limited access to imaging means many studies focus on overall stroke burden rather than subtypes. [7]

CAUSES

Coronary artery disease (CAD) develops due to both modifiable and non-modifiable risk factors. Modifiable factors includehypertension, hypercholesterolemia, diabetes mellitus, smoking, obesity, unhealthy diet, physical inactivity, and psychological stress, while non-modifiable factors include age, male sex, family history, and race. Hypertension is a major contributor to CAD and stroke. Earlier beliefs that high blood pressure was normal in the elderly were disproved by studies such as the Framingham Study, which showed a strong link between hypertension and atherothrombotic events.[8]Deep vein thrombosis (DVT) is characterized by thrombus formation in deep veins and is influenced by several risk factors, including malignancy, kidney and liver dysfunction, hematological abnormalities, insulin resistance, prior hemorrhage, inadequate antithrombotic therapy, recent surgery, prolonged immobility, alcohol dependence, and use of non-steroidal anti-inflammatory drugs. DVT may result in serious complications such as pulmonary embolism.[9]Stroke occurs due to impaired cerebral blood flow and may be ischemic or hemorrhagic. Hypertension is a key risk factor, with atherothrombotic brain infarction being the most common stroke type associated with elevated blood pressure. Additional causes, particularly in younger individuals, include large-artery disease, small-vessel disease, cardioembolic sources, thrombophilic states, vasculitis, pregnancy, and genetic disorders such as CADASIL, MELAS, and Fabry disease. [10]

PATHOPHYSIOLOGY

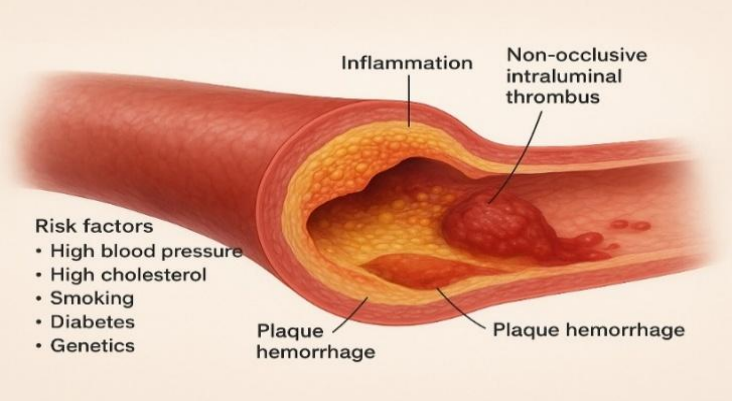

FIG:1 PATHOPHYSIOLOGY OF CORONARY ARTERY DISEASE [11]

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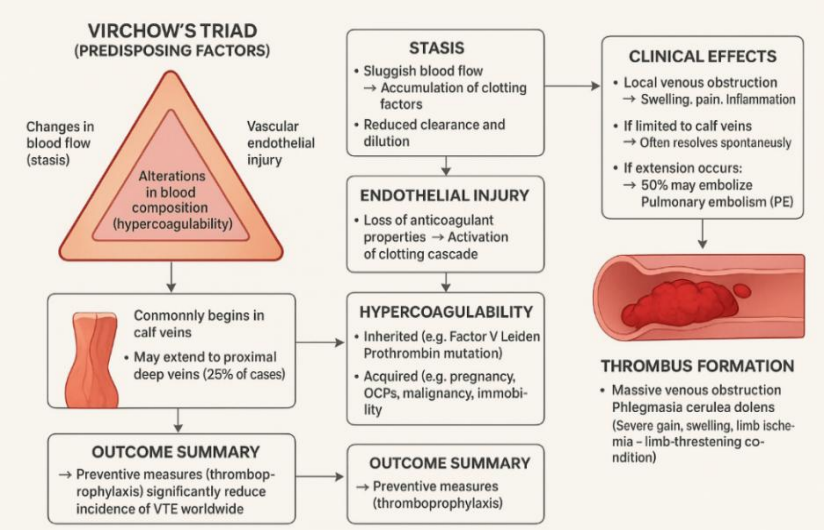

FIG :2 PATHOPHYSIOLOGY OF DEEP VEIN THROMBOSIS [6]

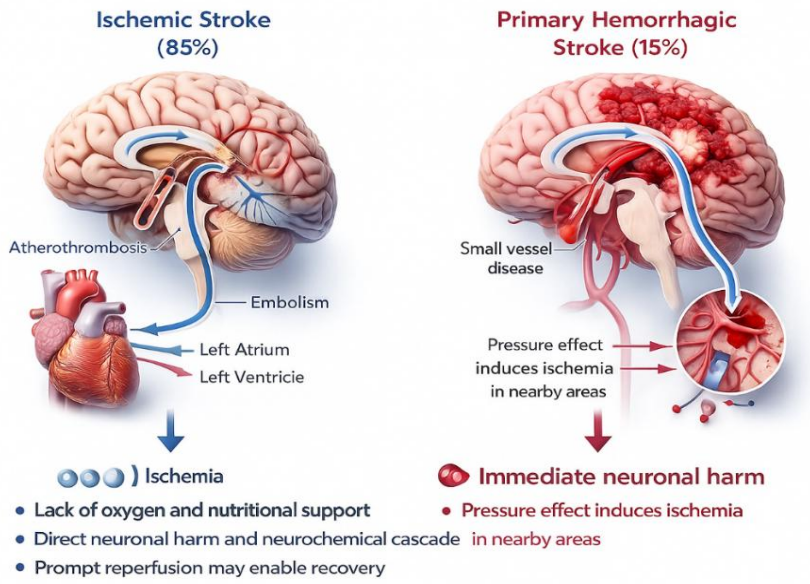

FIG:3 PATHOPHYSIOLOGY OF STROKE [12]

CLINICAL FEATURES

Coronary artery disease commonly presents with angina pectoris, characterized by chest pain or pressure that may radiate to the left arm, neck, jaw, or shoulder. Other symptoms include shortness of breath, fatigue, palpitations, dizziness, nausea, and sweating. Severe cases may lead to myocardial infarction, arrhythmias, heart failure, or sudden cardiac death.[13] Deep vein thrombosis (DVT) cannot be reliably diagnosed by history or physical examination alone, as classical signs may be absent. Proximal DVT commonly presents with calf pain and leg swelling, while severe forms such as phlegmasia alba dolens and phlegmasia cerulea dolens cause extensive ischemic changes. [14]Stroke results from impaired cerebral blood flow and presents with region-specific neurological deficits. Occipital and temporal lobe involvement may cause prosopagnosia and pure alexia, whereas brainstem strokes affect cranial nerves, motor, sensory, and cerebellar functions, leading to dysarthria, dysphagia, hemiparesis, and ataxia.[15]

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RISK FACTORS

Disease	Risk Factors / Conditions / Details
Coronary Artery Disease (CAD)	High blood pressure, high blood cholesterol, diabetes mellitus, smoking, obesity/overweight, physical inactivity, poor diet, stress
	Increasing age, male sex, family history, race
	Higher risk in mild–moderate hypertension with diabetes, dyslipidaemia, left ventricular hypertrophy.
	Complications: peripheral arterial disease, stroke, silent myocardial infarction, TIA, retinopathy, reduced renal function
	Framingham study: ≥60% of older men and ≥50% of older women with hypertension had ≥1 cardiovascular complication. [16]
Deep Vein Thrombosis (DVT)	Muscle strain or blunt force injury
	Ruptured muscle with blood collection under fascia
	Spontaneous bleeding or hematoma
	Ruptured Baker's cyst
	Arthritis, synovitis, muscle inflammation
	Cellulitis, lymphangitis, inflammatory lymph node swelling
	Superficial thrombophlebitis
	Reduced arterial circulation
	Pregnancy or hormonal contraceptive use
	Chronic venous insufficiency / venous reflux
	External venous compression (tumours, lymph nodes, abscess, right iliac artery). [17]
Stroke	Diabetes, high homocysteine levels
	Elevated arterial blood pressure (↓ <5 mmHg reduces stroke risk & mortality)
	Atherosclerosis
	Medications: ACE inhibitors, diuretics, antiplatelets, antihypertensives
	Statins normalize vessel function
	Statins reduce first and recurrent stroke risk by 25–30%
	Combined preventive strategies reduce risk up to 70%
	Increased demand for acute stroke care due to aging population
	Need for improved acute management and post-stroke recovery. [18]

DIAGNOSIS

1.CORONARY ARTERY DISEASE

Elderly Patients with Acute Coronary Syndrome (ACS)

Atypical / Unusual Initial Symptoms

↓

Delay in Medical Care and PCI (>120 minutes)

↓

Coronary Artery Disease with Atheromatous Plaque

↓

Imbalance in Myocardial Oxygen Supply and Demand,

→Myocardial Ischemia → Chronic Stable Angina

↓

Physician Risk Underestimation

→ Undertreatment ("Risk Treatment Paradox")[19]

2.DEEP VEIN THROMBOSIS

1. Clinical suspicion: unilateral limb swelling, warmth, pain; check DVT risks (surgery, immobility, pregnancy, OCPs, cancer, prior VTE).
2. Pretest probability: apply Wells score → categorize as likely or unlikely.
3. If unlikely: do D-dimer.
    - Negative → DVT excluded.

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- Positive → proceed to compression venous ultrasound (US).

4. If likely: skip D-dimer → perform compression venous US directly.
5. If D-dimer may be falsely elevated (infection, recent surgery, cancer) → favour immediate US.
6. Ultrasound interpretation:
   - Positive → DVT confirmed — start treatment.
   - Negative but high suspicion → repeat US in 48–72 hours or use alternative imaging.
7. Alternative imaging if US inconclusive/unavailable: CT venography, MR venography, or contrast venography.
8. Document findings and act promptly based on imaging and bleeding risk. [20]

3.STROKE

1. Record exact symptom onset time and verify with a witness.
2. Take rapid focused history and perform neuro exam.
3. Look for ischemic signs: focal weakness, speech disturbance, visual/sensory deficits.
4. Perform NIHSS for standardized scoring (5–8 minutes).
5. Note examiner confidence — flag low-confidence exams for repeat/second opinion.
6. If stroke suspected, act immediately (time-critical management).
7. Order urgent CT or MRI to distinguish ischemic vs hemorrhagic stroke.
8. Recognize hemorrhage clues: progressive worsening, decreased consciousness.
9. Suspect SAH if "worst headache," vomiting, seizures, or meningeal signs — still image even if focal signs absent.

Document: onset time, witness, NIHSS, exam findings; then manage per imaging/timing. [21]

TREATMENT

1.CORONARY ARTERY DISEASE

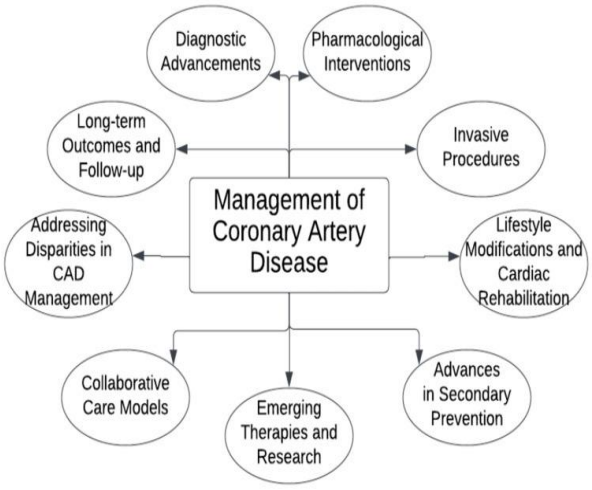

Fig4:Treatment of coronary artery disease[22]

2.DEEP VEIN THROMBOSIS

1. Confirm diagnosis & assess bleeding risk (duplex US ± D-dimer).
2. Prevent thrombus extension/PE and recurrence.
3. Start anticoagulation immediately if not contraindicated — preferred: DOAC (apixaban, rivaroxaban) or LMWH.
4. Choose agent: LMWH for pregnancy/cancer or transition to warfarin; UFH if severe renal failure or need rapid reversal.
5. Setting: outpatient if stable; admit for limb-threatening DVT, massive swelling, or high bleeding risk.
6. Duration: 3–6 months for provoked DVT; extend/indefinite for unprovoked or active cancer if bleeding risk acceptable.

If anticoagulation impossible, use temporary IVC filter; consider catheter-directed thrombolysis for severe cases.[2]

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3.STROKE

1. Patient assessment & admission – evaluate clinical stability, bleeding risk, and admission location.
2. Diagnosis confirmation – duplex ultrasound ± D-dimer for DVT; neuro exam + imaging (CT/MRI) for stroke.
3. Anticoagulation / Thrombolytic therapy – start DOACs or LMWH for DVT; thrombolysis for eligible stroke patients.
4. Agent selection / special considerations – LMWH for pregnancy/cancer; UFH for renal failure or rapid reversal; thrombectomy or catheter-directed therapy for stroke/DVT complications.
5. Supportive care – cardiac monitoring, fever management, seizure control, complication prevention, swallowing assessment.
6. Treatment setting & monitoring – outpatient if stable; admit for limb-threatening DVT, massive stroke, or high-risk patients.
7. Duration & follow-up – 3–6 months anticoagulation for provoked DVT; extended for unprovoked or cancer-associated; ongoing rehab for stroke.

Preventive measures – mobility, DVT prophylaxis, lifestyle modifications, secondary stroke prevention.[23]

PREVENTION

1.Coronary Artery Disease (CAD)

Preventing coronary artery disease mainly focuses on adopting a heart-healthy lifestyle. This includes consuming a balanced diet low in saturated fats and sodium, engaging in regular physical activity, avoiding smoking, maintaining a healthy body weight, and effectively controlling blood pressure, cholesterol, blood glucose levels, and stress. Adequate sleep and limiting alcohol intake further reduce CAD risk. Regular physical activity plays a key role in prevention, with recommendations of at least 150 minutes of moderate-intensity exercise per week or 75 minutes of vigorous-intensity activity. Moderate activities include brisk walking (2.4–4 mph), cycling (5–9 mph), active yoga, and recreational swimming, while vigorous activities include jogging, running, cycling,>10mph,tennis,swimming.
Body mass index (BMI) monitoring is essential, as overweight (BMI 25–29.8 kg/m²) and obesity (BMI ≥30 kg/m²) significantly increase the risk of atherosclerotic cardiovascular disease (ASCVD). Annual BMI assessment, calorie restriction, and lifestyle modification are recommended preventive strategies. [24]

2. Deep Vein Thrombosis (DVT)

Limited mobility is a major risk factor for deep vein thrombosis. Regular physical activity and avoiding prolonged immobility help reduce venous stasis and thrombus formation. During long-distance travel, maintaining mobility through periodic walking, stretching, and adequatehydrationisessential.
Maintaining a healthy body weight also contributes to DVT prevention. In hospitalized or post-surgical patients with increased risk, pharmacological prophylaxis such as heparin may be required. In rare cases, individuals may have a genetic predisposition to DVT, and informing healthcare providers about a family history of venous thromboembolism is important for risk assessment and preventive planning.[25]

3. Stroke

Stroke prevention involves a combination of community-level, individual-level, and healthcare system–based strategies. Community interventions include smoking restrictions, promotion of healthy urban environments, and salt reduction. Personal strategies such as lifestyle modification, use of polypills, and adherence to preventive medications are effective.
Innovative approaches including mobile health (mHealth) technologies, task shifting, and international collaborations supported by global health initiatives enhance stroke prevention efforts. Depending on the stage of epidemiological transition, preventive strategies may be classifiedas primordial or primary prevention. Targeting highly prevalent and modifiable risk factors—particularly hypertension, diabetes, unhealthy diet, physical inactivity, and smoking—provides the greatest reduction in stroke incidence and improves long-term outcomes. [26]

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ARTIFICIAL INTELLIGENCE

1.CORONARY ARTERY DISEASE

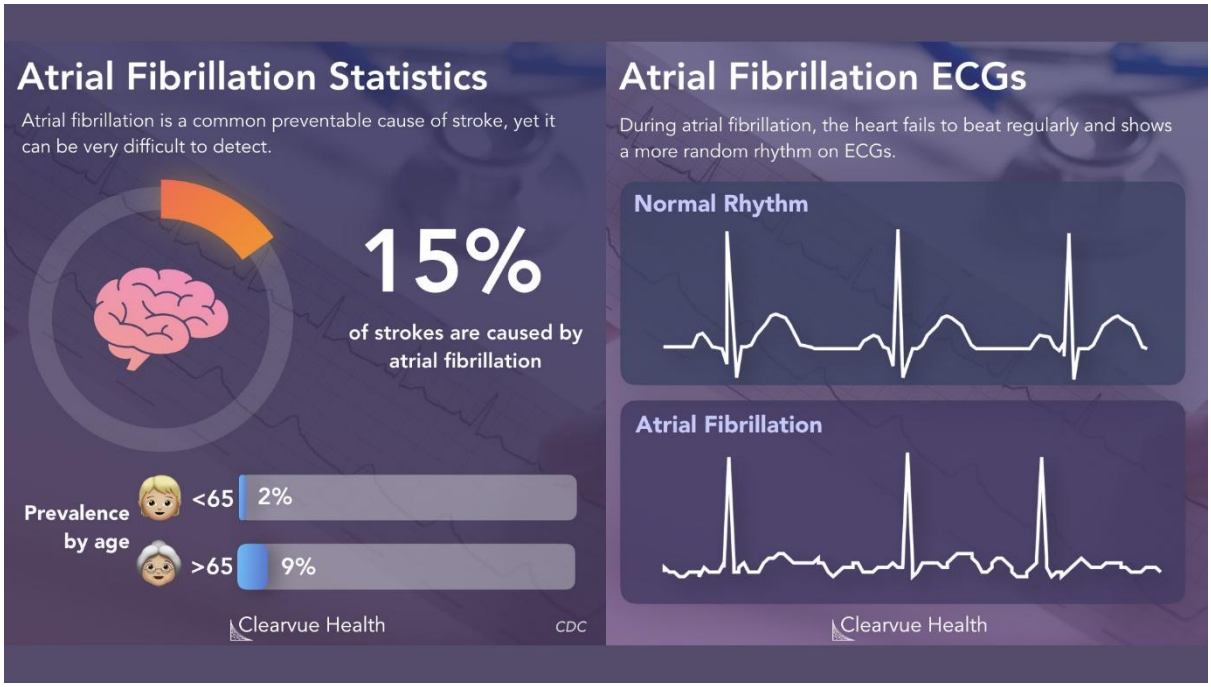

FIG:5ARTIFICIAL INTELLIGENCE IN CORONARY ARTERY DISEASE

AI has demonstrated substantial utility in coronary artery disease (CAD) management by analyzing multimodal datasets including coronary CT angiography, electrocardiograms, laboratory markers, and clinical history. Deep learning enhances plaque characterization and stenosis assessment, supporting non-invasive diagnosis. AI-assisted ECG interpretation improves ischemia and arrhythmia detection, while predictive analytics guide revascularization and medical therapy decisions. Integration with wearable devices further enables remote cardiac monitoring and personalized long-term cardiovascular care.[27]

2.DEEP VEIN THROMBOSIS

In deep vein thrombosis (DVT) and hospital-acquired venous thromboembolism (VTE), AI focuses primarily on dynamic risk assessment and prevention. Machine learning algorithms continuously analyze patient data—such as age, body mass index, mobility status, surgical history, malignancy, and laboratory findings—to estimate thrombotic and bleeding risks. AI-driven CDSS platforms automatically issue prophylaxis alerts and assist in anticoagulant dosing. Natural language processing further enhances detection by extracting relevant information from ultrasound and radiology reports, improving surveillance accuracy.[28]

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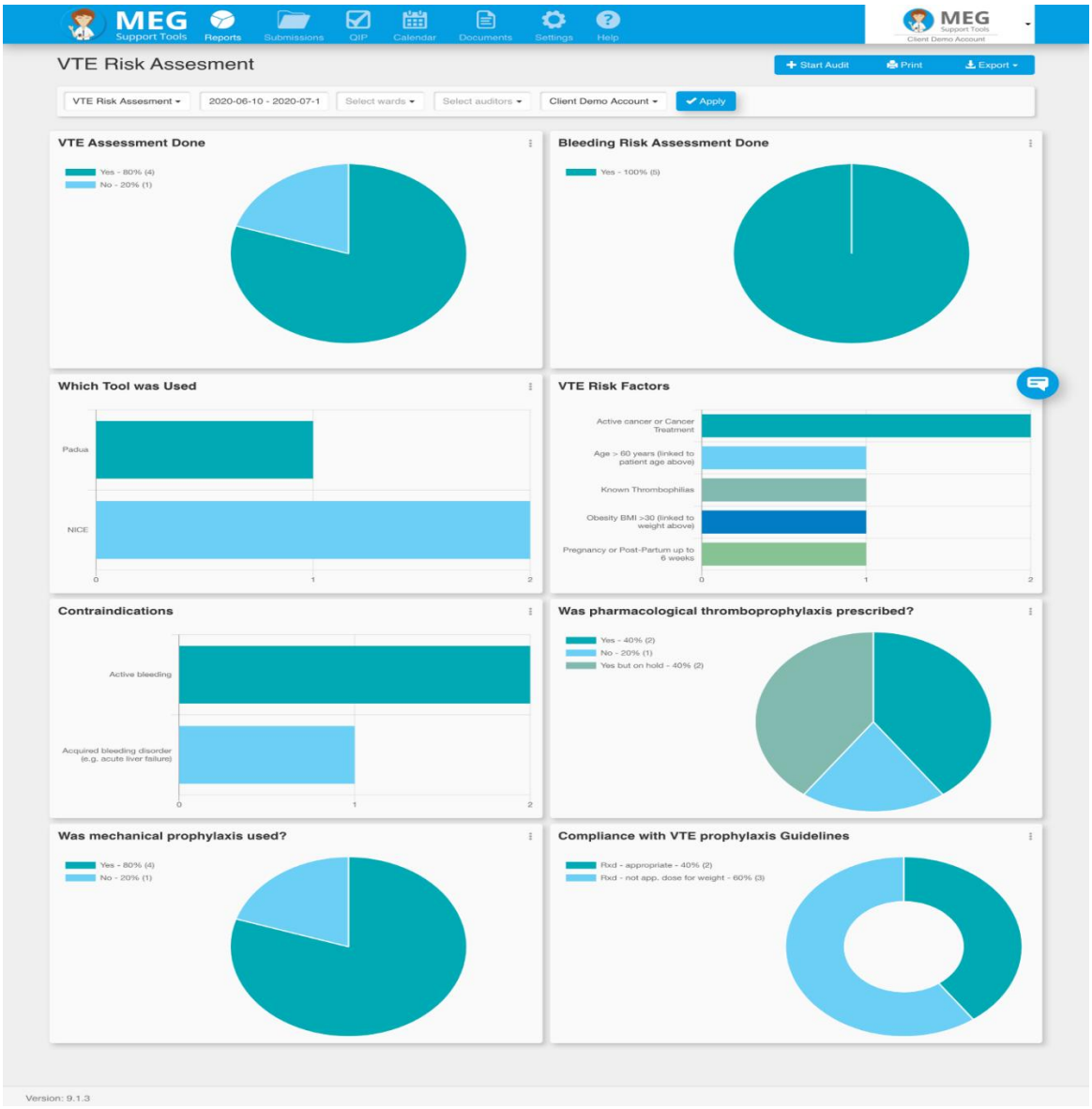

FIG:6 ARTIFICIAL INTELLIGENCE IN DEEP VEIN THROMBOSIS

3.STROKE

Artificial intelligence (AI) has significantly improved stroke diagnosis and management by enabling rapid interpretation of neuroimaging and personalized risk prediction. Deep learning algorithms integrated into CT and MRI systems can detect large vessel occlusion, estimate infarct core volume, and identify salvageable penumbra within minutes, facilitating timely thrombolysis and mechanical thrombectomy. Beyond imaging, machine learning models combine demographic data, vascular risk factors, laboratory values, and genetic information to predict primary and recurrent stroke risk. AI-based clinical decision support systems (CDSS) embedded in electronic medical records generate alerts for anticoagulation in atrial fibrillation–related stroke and optimize secondary prevention strategies.[29]

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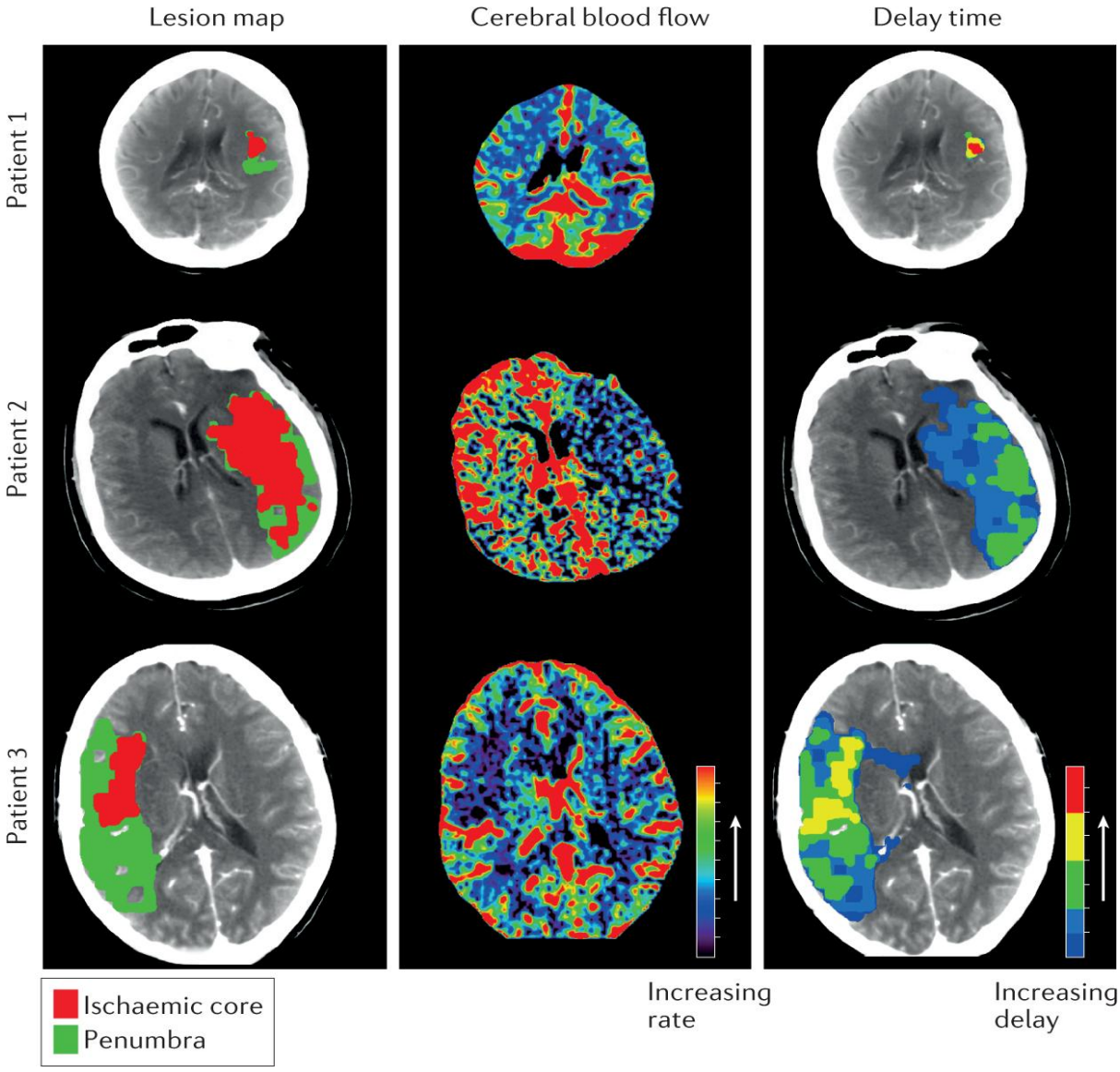

FIG:7 ARTIFICIAL INTELLIGENCE IN STROKE

FUTURE RESEARCH

1.CORONARY ARTERY DISEASE

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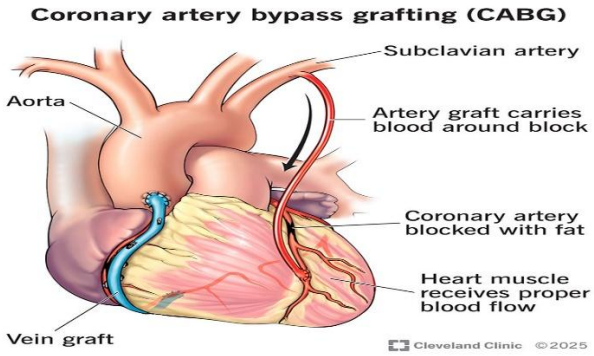

FIG:8 FUTURE RESEARCH IN CORONARY ARTERY DISEASE

Future research in coronary artery disease should emphasize regenerative therapies and technological innovation. Stem cell–based cardiac repair, nanotechnology-enhanced stents, robotic-assisted interventions, and advanced drug delivery systems require large-scale trials to confirm long-term efficacy and safety in reducing cardiovascular mortality.[30]

2.DEEP VEIN THROMBOSIS

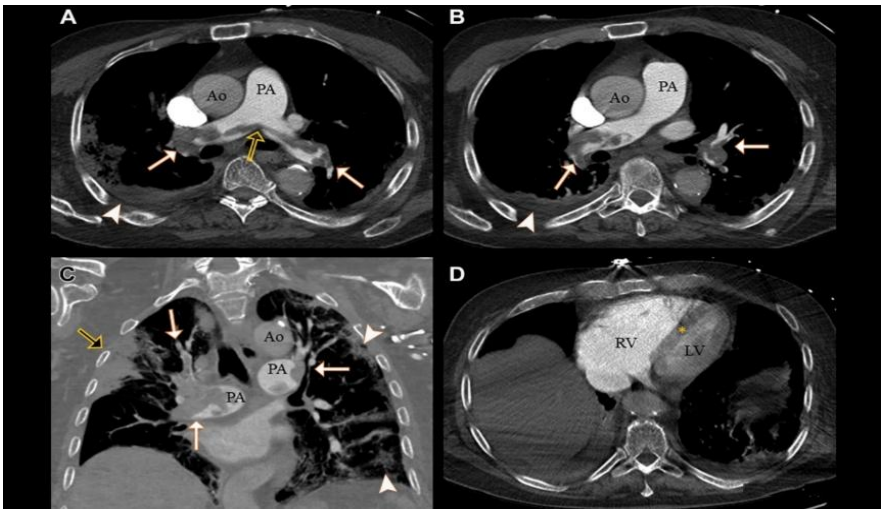

FIG:9 FUTURE RESEARCH IN DEEP VEIN THROMBOSIS

Future research in venous thromboembolism should address optimization of direct oral anticoagulant therapy, including individualized dosing, treatment duration, and safety in high-risk populations. Improved predictive models and biomarkers are required to assess recurrence and bleeding risks accurately. Enhanced strategies for early detection and hospital-based thromboprophylaxis will be essential to reduce disease burden.[31]

3.STROKE

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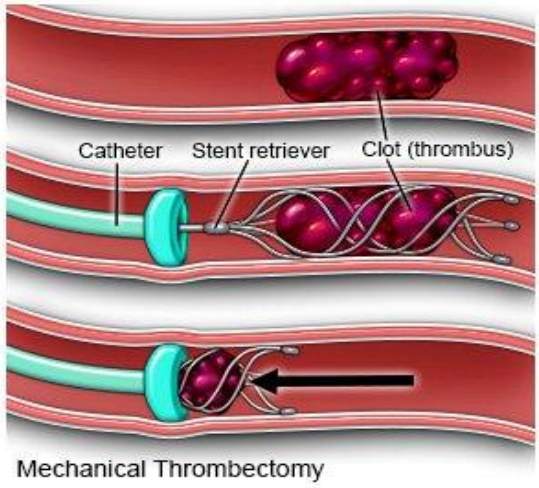

FIG:10FUTURE RESEARCH IN STROKE

Future research in stroke should focus on expanding eligibility for thrombolysis and mechanical thrombectomy, as only a small proportion of patients currently qualify due to strict criteria and limited therapeutic windows. Although these treatments reduce mortality and disability, many survivors continue to experience neurological deficits. Therefore, studies must prioritizeimproved patient selection through advanced imaging, biomarkers, and prehospital triage systems. Greater emphasis is also needed on understandingneuroplasticity and regenerative mechanisms to enhance long-term functional recovery and rehabilitation outcomes.[32]

CONCLUSION

Coronary artery disease, deep vein thrombosis, and stroke remain leading causes of global morbidity and mortality. Despite advances in pharmacological and interventional therapies, significant residual risk and long-term complications persist. These vascular disorders share common mechanisms, including endothelial dysfunction, inflammation, and thrombosis, emphasizing the need for integrated management strategies. The incorporation of artificial intelligence into clinical practice enhances risk prediction, imaging interpretation, and treatment optimization, supporting precision medicine approaches. Emerging innovations such as regenerative therapies and nanotechnology-based interventions further expand therapeutic possibilities. Continued research and ethical implementation of advanced technologies are essential to improve outcomes and reduce the global vascular disease burden.

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