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EMERGING TRENDS IN ANTICOAGULATION THERAPY

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ABSTRACT

Anticoagulation therapy is the first line and drug of choice for both the treatment and prophylaxis of venous thromboembolism (deep vein thrombosis and/or pulmonary embolism). Anticoagulation drugs, ranging from different preparations of heparin, warfarin, and newer direct oral drugs such as rivaroxaban and dabigatran, work mainly by inhibiting important factors and enzymes in the coagulation cascade by preventing fibrin formation, platelet aggregation, and clot assembly With recurrent thrombosis and embolisms being a feared complication for many physicians treating such cases, anticoagulation is often extended beyond the initial three- to six-month acute phase after an incident of venous thromboembolism. For some groups of patients, anticoagulation needs to be offered indefinitely to decrease the risk of a recurrent thrombosis. However, this concomitantly increases obvious and dangerous adverse effects such as increased risk of hemorrhage, as the ability to clot is hindered. This tradeoff between recurrent venous thromboembolism and bleeding is what underscores the controversy of the clinical question.

KEYWORDS: General introduction of Direct oral anticoagulants, Venous thromboembolism, Deep venous thrombosis, VTE, chemoprophylaxis, VTE extended therapy, Anticoagulation, Perioperative bridging G Therapy.

INTRODUCTION

In the last several years, anticoagulation pharmacology has been dramatically altered in the United States with the FDA approval of five new direct oral anticoagulant (DOAC) agents In 2012, the American College of Chest Physicians recommended treatment of acute VTE with vitamin K antagonists, while recognizing a major shift on the horizon "Given the paucity of currently available data and that new data are rapidly emerging, we give a weak recommendation in favor of vitamin K antagonists and LMWH therapy over dabigatran and rivaroxaban. By the time the updated ACCP guidelines were released in 2016 DOACs were a routine part of the prevention and treatment of venous thromboembolism (VTE).[1-3]

Available Anticoagulants

- **Unfractionated Heparin (UFH):** These include heparin, make complexes with antithrombin 3, and inactivates various clotting factors. Its onset of action is rapid, has a short half-life, and can be monitored using activated partial thromboplastin (aPTT), activated clotting time, and anti-factor 10a activity. The recommended target ratio of aPTT is 1.5 to 2.2 times the patients' aPTT.
- **Low Molecular Weight Heparin (LMWH):** These are enoxaparin, dalteparin, tinzaparin, nadroparin, have a longer length of action, long half-life, and can be monitored using anti- factor 10a activity. However, monitoring is not indicated except in certain conditions like pregnancy and renal failure.
- **Vitamin K Dependent Antagonists (VKA):** Warfarin, one of the most common anticoagulants available. It acts by inhibiting vitamin K epoxide reductase (VKOR), which is needed for the gamma-carboxylation of vitamin K-dependent factors (factors 2, 7, 9, 10, protein C and S). It has a narrow therapeutic window of dosing, and its effect is profoundly altered by certain factors including diet (leafy green vegetables, fruits like avocado, kiwi), medications, and genetic mutations in the VKOR complex which leads to resistance. It requires frequent monitoring with an international normalized ratio (INR).[4]
- **Direct Thrombin Inhibitors:** Bivalirudin, argatroban, and dabigatran are direct thrombin inhibitors; these inhibit the cleavage of fibrinogen to fibrin by thrombin. All products are renally metabolized.
- **Direct Factor 10a Inhibitors:** These include rivaroxaban, apixaban, edoxaban, and betrixaban. Mechanism of action involves inhibition of the cleavage of prothrombin to thrombin by binding directly to factor 10a. These products are only orally.

- The terms direct oral anticoagulants (DOACs), new oral anticoagulants (NOACs), or target-specific oral anticoagulants (TSOACs) refer to those oral anticoagulants which specifically inhibit factors 2a (thrombin) or 10a. According to the International Society of Thrombosis and Haemostasis, DOACs is the preferred term. DOACs have been found to have similar effects when compared to other anticoagulants. Some studies have also shown possible decreased bleeding incidence with DOAC.[5-6]

HISTORY

Over the last several years, anticoagulation pharmacy has been dramatically altered with U.S. FDA approval of 5 direct oral anticoagulants, one novel reversal agent and a second designated for fast track approval. Trial data surrounding current trends in anticoagulant choice for VTE, reversal and bridging are constantly redefining modern day practice.

Extended therapy for unprovoked VTE has expanded to include low dose DOACs, ASA and the use of a new HERDOO2 scoring system to identify women who can stop anticoagulant therapy without increased risk of recurrent VTE.

Trends in thromboprophylaxis include extended duration low dose DOACs to prevent VTE in high risk orthopedic and medical patients. Development of successful DOACs Scientists in the 2000s perfected the molecular design and conducted extensive clinical trials to introduce the four major DOACs currently in use.[7]

1. **Dabigatran (Pradaxa®)**

- **Developer**: Boehringer Ingelheim.
- **Timeline**: Approved by the FDA in November 2010.
- **Mechanism**: This was the first widely successful oral direct thrombin inhibitor. Its development was influenced by earlier research into the thrombin-inhibiting properties of compounds derived from the leech.

2. **Rivaroxaban (Xarelto®)**

- **Developer**: Bayer.
- **Timeline**: Approved in 2011, it was the first Factor Xa inhibitor available in the U.S.
- **Mechanism**: Rivaroxaban inhibits factor Xa, a different target than dabigatran, and was developed through a targeted drug design approach. In 2010, the discovery and development history of rivaroxaban was formally published by researchers associated

with its creation.

3. Apixaban (Eliquis®)

- **Developer**: Bristol-Myers Squibb.
- **Timeline**: Approved in 2014.
- **Mechanism**: Another oral factor Xa inhibitor, apixaban's metabolic and pharmacokinetic profile was published by C.E. Frost and colleagues in 2009. The ARISTOTLE trial, involving scientists like Christopher Granger and John Alexander, was a major clinical trial that proved apixaban's safety and efficacy compared to warfarin.

4. Edoxaban (Savaysa®)

- **Developer**: Daiichi-Sankyo.
- **Timeline**: Approved in 2014.
- **Mechanism**: Also a factor Xa inhibitor, its development was supported by large, global clinical trials involving hundreds of researcher.

Table no. 1: Advantages of DOAC & Warfarin.[8]

FEATURE	WARFARIN	NEW DOAC
Onset	Slow	Rapid
Dosing	Variable	Fixed
Food Effect (Vitamin K)	Yes	No
Drug Interaction	Many	Few
Monitoring	Yes	No
Offset	Long	Shorter

Table no 2: Effect of DOAC on 'Coagulation Studies'.[9-11]

	Dabigatran	Rivaroxaban	Apixaban	Edoxaban
aPTT	Provides estimate of effect but limited response; dose response curve gets flat at higher concentrations; response varies with thromboplastin reagent used	Shows dose-dependent effect but is nonlinear; response varies with thromboplastin reagent used	Very low sensitivity	Small response with large variability among thromboplastin reagents in use
PT	Linear response but poorly reflects intensity of anticoagulation; INR is usually below 2 even at peak therapeutic drug concentration	Poorly reflects intensity of anticoagulation; INR is usually below 2 even at peak therapeutic drug concentration; response varies with reagent used	Not sensitive; small and variable response	Small and variable response
ECT	Quantifiable dose-response; reliable but needs standardization because of variability in sensitivity to dabigatran among different ecarin lots[a]	No effect	No effect	No effect
TT	Too sensitive, can be used only to exclude the presence of dabigatran	No effect	No effect	No effect
Diluted TT	Quantifiable dose-response; very reliable (not widely available)	No effect	No effect	No effect
Anti-factor Xa activity	No significant effect	Accurately estimates plasma drug concentrations; standardization of results across reagents is required	Accurately estimates plasma drug concentrations	No studies available
Preferred test	ECT and diluted TT	Anti-factor Xa activity	Anti-factor Xa activity	NA

Abbreviations: aPTT, activated partial thromboplastin time; DOAC, direct oral anticoagulant; ECT, ecarin clotting time; INR, international normalized ratio; NA, not applicable; PT, prothrombin time; TT, thrombin time.
[a] Ecarin is a commercially available snake venom that converts prothrombin into meizothrombin, which can be measured to evaluate the activity of dabigatran.

Table no. 3: Comparison of Newly invented DOAC.[12]

	Dabigatran	Apixaban	Rivaroxaban	Edoxaban
Brand Name	Pradaxa	Eliquis	Xarelto	Savaysa
Mechanism of Action	Direct thrombin inhibitor	Direct factor Xa inhibitor	Direct factor Xa inhibitor	Direct factor Xa inhibitor
FDA Indication(s)	• Prevention of stroke in NVAF • Treatment of DVT and PE • Secondary prevention of DVT and PE	• Prevention of stroke in NVAF • Postoperative DVT prophylaxis • Treatment of DVT and PE • Secondary prevention of DVT and PE	• Prevention of stroke in NVAF • Postoperative DVT prophylaxis • Treatment of DVT and PE • Secondary prevention of DVT and PE	• Prevention of stroke in NVAF • Treatment of DVT and PE
Half-Life	12-17 hours	12 hours	5-9 hours	10-14 hours
Renal Elimination	80%	27%	66%	50%
Protein Binding	35%	87%	92%-95%	55%
Dosing in NVAF	150 mg twice daily	5 mg twice daily	20 mg daily	60 mg daily
Dosing in Treatment of VTE	150 mg twice daily after 5-10 days of parenteral anticoagulation	10 mg twice daily for 7 days, then 5 mg twice daily	15 mg twice daily for 21 days, then 20 mg daily	60 mg daily after 5-10 days of a parenteral anticoagulant
Dosing in Secondary Prevention	150 mg twice daily	2.5 mg twice daily	20 mg daily	n/a
Dosing in Post-operative Prophylaxis	n/a	2.5 mg twice daily	10 mg daily	n/a
Dosing Adjustments	Clcr 15-30 mL/min: 75 mg twice daily (NVAF) Clcr < 15 mL/min (AF): no dosing recommendation Clcr < 30 mL/min (VTE): no dosing recommendation	In NVAF, if 2 of the following 3 present reduce dose to 2.5 mg twice daily: Age ≥ 80 years, weight ≤ 60 kg, SCr ≥ 1.5 mg/dL	Clcr 15-50 mL/min (NVAF): 15 mg daily Clcr < 15 mL/min (NVAF): avoid use Clcr < 30 mL/min (VTE): avoid use	Clcr > 95 mL/min (NVAF): avoid use Clcr 15-50 mL/min: 30 mg daily Weight ≤ 60 kg: 30 mg daily
Administration	With or without food	With or without food	With food[a]	With or without food
Pertinent Drug Interactions (incomplete listing)	Avoid use in Clcr 15-30 mL/min PLUS one of following: Dronedarone Ketoconazole Decrease dose to 75 mg twice daily in those with Clcr 30-50 mL/min PLUS one of following: Dronedarone Ketoconazole Avoid use with: Rifampin	Decrease dose by 50% if current dose > 2.5 mg twice daily with: Ketoconazole Itraconazole Ritonavir Clarithromycin Avoid use with: Rifampin Carbamazepine Phenytoin St. John's wort	Avoid use with: Ketoconazole Itraconazole Lopinavir/ritonavir Ritonavir Indinavir Conivaptan Avoid use with: Rifampin Carbamazepine Phenytoin St. John's wort	Avoid use with: Rifampin

VENOUS THROMBOEMBOLISM (VTE)

Collectively It refers to deep vein thrombosis (DVT) and pulmonary embolism (PE), and their respective chronic complications including post-thrombotic syndrome (PTS) and chronic thromboembolic pulmonary hypertension (CTEPH).

Anticoagulant choice For VTE

Whether these new trends become widely adopted remains uncertain. Certainly, the prescription of DOACs for the treatment of VTE has become widespread, but whether daily use of the SAMe-TT2R2 score is as readily adopted, as say CHADS2 score for atrial

fibrillation, remains uncertain.

In fact, in most cases, there will be overriding co-morbidities or patient preference that will drive the selection of anticoagulant. In our own practice, the SAMe-TT2R2 score serves to identify patients at high risk of low time in therapeutic range, and thereby allow for shorter term and more frequent follow up.

As a result of the multitude of evidence showing both the effectiveness and safety of DOACs, in 2016 the American College of Chest Physicians recommended DOACs as first line treatment of acute VTE over VKAs in patients whom do not have an associated cancer (Grade 2B). In the patients with cancer who develop acute VTE, LMWH remains the recommended first line treatment.

Although guidelines recommend DOACs over VKA in non-malignancy related VTE there are several scenarios which would preclude the use of DOACs; for example, patients with mechanical heart valves, patients who cannot afford the cost of the medication, and patients with impaired renal function.[13]

Table no 4: Pharmacokinetics profile of DOAC for treatment and prevention of VTE.[14]

Key Points	Rivaroxaban	Apixaban	Edoxaban	Dabigatran
Mechanism of action	Factor Xa inhibitor	Factor Xa inhibitor	Factor Xa inhibitor	Direct thrombin inhibitor
Time to peak	2–4 h	3–4 h	1–2 h	1.5 h
Half-life	9–13 h	12 h	10–14 h	12–17 h
Oral bioavailability	66%	> 50%	62%	3%–7%
Excretion	Kidney, 36%; feces, 7%	Kidney, 28.8%; feces, 56%; minimal biliary	Kidney, 50%; rest is biliary/ intestinal and metabolism	Kidney, 80%
Plasma protein binding	92%–95%	~ 90%	55%	35%
Absorption	Primarily proximal small intestine; some gastric absorption	Primarily proximal small intestine; some gastric absorption	Proximal small intestine	Lower stomach and duodenum
Dosing: for initial VTE treatment	15 mg twice daily for 21 d followed by 20 mg daily (with largest meal)	10 mg twice daily for 7 d followed by 5 mg twice daily	Parenteral agent for 5–10 d followed by 60 mg daily or 30 mg daily if any of following: CrCL 15–50 mL/min, weight ≤ 60 kg, or concomitant P-glycoprotein inhibitor	Parenteral agent for 5–10 d followed by 150 mg twice daily
Dosing: for VTE prophylaxis or extended treatment	10 mg daily after at least 6 mo of therapeutic anticoagulation	2.5 mg twice daily after at least 6 mo of therapeutic anticoagulation	Not studied	No dose adjustment
Special considerations	Avoid if CrCL ≤ 30 mL/min or Child-Pugh class B and C; must be taken with food	Avoid if CrCL ≤ 15 mL/ min or Child-Pugh class B and C	Avoid if CrCL ≤ 15 mL/min or Child-Pugh class B and C	Avoid if CrCL ≤ 30 mL/min or Child-Pugh class B and C, if dyspepsia, upper GI symptoms
Dose adjustments*	None (no adjustments for age, weight, or sex)	None (no adjustments for age, weight or sex)	Decrease to 30 mg daily if any of following: CrCl 15–50 mL/min, weight < 60 kg, or concomitant P-glycoprotein inhibitor	None (no adjustments for age, weight, or sex)
Drug interactions	P-glycoprotein, CYP 3A4/5	P-glycoprotein, CYP 3A4/5	P-glycoprotein	P-glycoprotein, PPIs
Laboratory measurement (to determine if present/ not present only)	Anti-Xa	Anti-Xa	Anti-Xa	Dilute thrombin time
Reversal agent	Andexanet (specific) or 4F-PCC (nonspecific)	Andexanet (specific) or 4F-PCC (nonspecific)	4F-PCC (nonspecific)	Idarucizumab (specific)

Abbreviations: 4F-PCC, four-factor prothrombin complex concentrate; CAD, coronary artery disease; CrCl, creatinine clearance; DOAC, direct oral anticoagulant; GI, gastrointestinal; PAD, peripheral artery disease; PPI, proton pump inhibitor; VTE, venous thromboembolism.
*No dose adjustments are necessary for the treatment and secondary prevention of VTE. There are different doses and dose adjustments for the use of DOACs in other indications such as prevention of stroke in atrial fibrillation, prevention of VTE in elective hip/knee surgery, and prevention of cardiovascular events with PAD or CAD.

Table no 5: Study Supporting DOAC for the treatment of VTE.[15-18]

TRIAL WITH SIGNIFICANT DIFFERENCE	RE-COVER[36] (N=1274)	EINSTEIN[36] (N=3449)	EINSTEIN-PE[37] (N=4832)	HOKUSAL-VTE[37] (N=4921)	AMPLIFY[38] (N=26009)
Trial design	Randomized double blind non inferiority	Randomized open label non inferiority	Randomized open label non-inferiority	Randomized double blind non-inferiority	Randomized double blind non-inferiority
Drug regimen	Dabigatran 150mg Twice a day VS Warfarin	Rivaroxaban 15mg Twice daily for 3 weeks followed by 20mg daily VS warfarin	Rivaroxaban 15mg Twice daily for 3 weeks followed by 20mg daily VS warfarin	Edoxaban 60mg daily VS warfarin	Apixban 10mg twice daily for 7 days followed by 5mg twice daily VS warfarin
Treatment duration	160 days	3,6,12 month	3,6,12 month	3-12 month	6 month
VTE & all cause mortality	2.4% (dabigatran) VS 2.1% (warfarin)	2.1% (rivaroxaban) VS 3% (warfarin)	2.1% (rivaroxaban) VS 1.8% (warfarin)	3.2% (edoxaban) VS 3.5%(warfarin)	2.3% (apixaban) VS 2.7%(warfarin)
Relative risk	1.10	0.68^0	1.12^0	0.89^0	$o.84^0$
Death	1.6 VS 1.7%	2.2% VS 2.9%	2.4% VS 2.1%	3.2% VS 3.1%	1.5% VS 1.9%
Major-bleed	1.6% VS 1.9%	0.8% VS 1.2%	1.1% VS 2.2%	1.4% VS 1.6%	0.6% VS 1.8%
Clinically relevant non major bleeding	5.6 VS 8.8%	7.3% VS 7%	10.3%Vs11.4%	7.2% VS 8.9%	3.8% VS 8%

Table showing the common direct oral anticoagulation medications with their statistically significant difference in VTE.

DEEP VEIN THROMBOSIS

Deep vein thrombosis (DVT) is defined as development of thrombosis within the deep veins of the pelvis or lower limbs Vessel endothelium injury causes sluggish blood flow, which promotes blood clot formation and reduces venous blood flow, or in severe cases can induce pulmonary embolism (PE) as the thrombi move from the deep veins to the lungs via the vasculature.

DVT is a potentially dangerous condition with a myriad of risk factors. Prophylaxis is very important and can be mechanical and pharmacological. The mainstay of treatment is anticoagulant therapy. Low-molecular-weight heparin, unfractionated heparin, and vitamin K antagonists have been the treatment of choice.

Currently anticoagulants specifically targeting components of the common pathway have been recommended for prophylaxis. These include fondaparinux, a selective indirect factor Xa inhibitor and the new oral selective direct thrombin inhibitors (dabigatran) and selective factor Xa inhibitors (rivaroxaban and apixaban). Others are currently undergoing trials. Thrombolytics and vena caval filters are very rarely indicated in special circumstances.[19]

Include

- Swelling, pain, or tenderness in the affected limb.
- A feeling of warmth in the swollen area.
- A change in the color of the skin, such as redness or purple discoloration.
- Enlarged veins near the skin's.

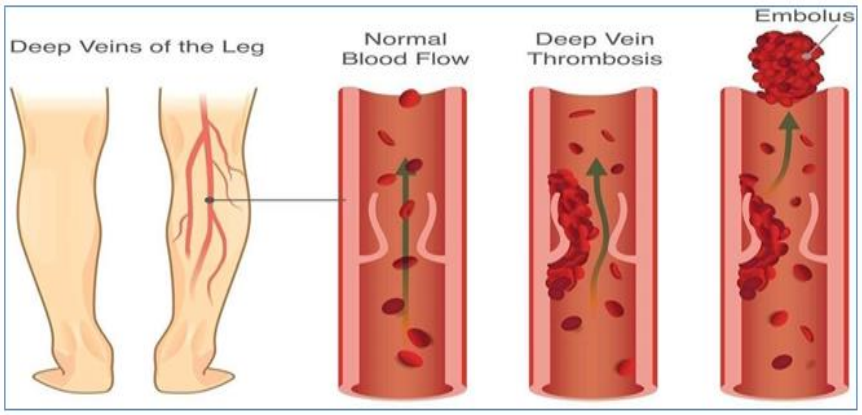

Fig. no. 1: Deep Vein Thrombosis.[20]

Table no. 6: Bridging And Reversible Strategy of DOAC.[21]

Agent	Mechanism of Action	# days to hold for minor procedures	# days to hold for major procedures	Reversal Agent	Alternative Treatment Option	Bridging therapy required
Apixaban	Factor Xa inhibitor	1 (2 doses)	2 (4 doses)	Unavailable	• 4 Factor PCC (*Kcentra*) • Factor VIIa • Tranexamic acid • Hemodialysis	No
Rivaroxaban	Factor Xa inhibitor	1 (1 dose)	2 (2 doses)	Unavailable	• 4 Factor PCC (*Kcentra*) • Factor VIIa • Tranexamic acid	No
Dabigatran	Direct thrombin inhibitor	1 (2 doses) **hold for 2 days if renal insufficiency*	2 (4 doses) **hold 4 days if renal insufficiency*	Idarucizumab	• 4 Factor PCC (*Kcentra*) • Factor VIIa • Tranexamic acid	No
Edoxaban	Factor Xa inhibitor	1 (1 dose)	2 (2 doses)	Unavailable	• 4 Factor PCC (*Kcentra*) • Factor VIIa • Tranexamic acid	No

Table no. 7: Dosing Considerations for Various Anticoagulant choice.[22-23]

	Dosing	Half Life	Considerations
Apixaban	10mg BID x7 days Then 5mg BID (2.5mg BID for long-term therapy)	7–11hrs	• Superior to standard therapy with no increase in bleeding • Twice daily dosing • No renal adjustment
Rivaroxaban	15mg BID x3 wk Then 20mg daily	12 hrs	• Once daily regimen • May need to be renal adjusted • Increase in GI bleeding compared to warfarin
Dabigatran	LMWH for 5–10d 150mg BID	8–15hr	• Poor choice in renal dysfunction • Requires heparin bridge • Increase in GI bleeding compared to warfarin • Up to 10% have dyspepsia • Avoid in patients with significant CAD
Edoxaban	LMWH for 5–10 days 60mg Daily 30mg Daily ≤60kg or CrCl 15–50	10–14 hrs	• Once daily dosing • Requires heparin bridge • Needs to be renal adjusted • Increase in GI bleeding compared to warfarin
Warfarin	Variable dosing titrate to goal INR	~ 40 hrs	• Reliable and predictable reversal • Can use SAMe-TT_2R_2 to predict poor candidates • Requires bridging • Requires frequent monitoring • Has many interactions with food and other medications

The following table Showing the "Dosing and considerations for various anticoagulant choices." The table compares five different anticoagulants, providing details on their dosing, half-life, and key considerations for their use.

EXAMPLE OF DOAC

- **Anticoagulants**: Apixaban, Rivaroxaban, Dabigatran, Edoxaban, and Warfarin.
- **Dosing**: Specifies the dosage and regimen for each drug, including initial and long-term therapy where applicable.
- Half-life: Lists the approximate half-life for each medication.
- **Considerations**: Provides important notes for each drug, such as whether it's a once-daily or twice- daily regimen, the need for renal adjustment, potential for increased GI bleeding, and other Specific cautions like drug interactions or bridging requirement.
- **Dabigatran Etexilate:** It s a medication used to prevent blood clots.
- **Uses**: It is used for stroke prevention, deep vein thrombosis, and pulmonary embolism. It helps prevent the formation of blood clots in the legs, lungs, brain, or heart.

- **Mechanism**: It is a novel oral anticoagulant (NOAC) that works by preventing the formation of blood clots in the body.

Dabigatran Etexilate

- Written By Dr. Swati Mishra
- Reviewed By Dr. Lalit Kanodia
- Last updated 20 Nov 2024|04:21 AM (UTC)

Dabigatran Etexilate Uses

- Dabigatran Etexilate is used for stroke
- Prevention, deep vein thrombosis, pulmonary. embolism, heart attack and stroke. It prevents the formation of blood clots in the legs, lungs, brain or heart**.**

How Dabigatran Etexilate works

- Dabigatran Etexilate is a novel oral Common side effects of Dabigatran anticoagulant (NOAC). It works by preventing the formation of blood clots in the body**.**
  Dabigatran Etexilate
- Nausea, Anemia (low number of red blood cells), Gastrointestinal bleeding, Dyspepsia, Diarrhea, Abdominal pain, Nosebleed.

Description

- The image lists various medicine brands for the drug Dabigatran Etexilate, along with their manufacturers, price ranges, and number of variants:
- **Pradoxa**: Manufactured by Boehringer Ingelheim, priced at ₹333 to ₹673, with 3 variants.
- **Afogatran**: Manufactured by Torrent Pharmaceuticals Ltd, priced at ₹300 to ₹379, with 3 variants.
- **DABIstar**: Manufactured by Lupin Ltd, priced at ₹300 to ₹362, with 3 variants.
- **Dablexa**: Manufactured by Abbott, priced at ₹339 to ₹380, with 3 variants.
- **Dabigat**: Manufactured by Natco Pharma Ltd, priced at ₹260 to ₹328, with 3 variants.
- **Dabiclot**: Manufactured by Alkem Laboratories Ltd, priced at ₹274 to ₹559, with 5 variants.
- **Dabigza**: Manufactured by Glenmark Pharmaceuticals Ltd, priced at ₹235 to ₹433, with 4 variants.
- **Dabitra**: Manufactured by Emcure Pharmaceuticals Ltd, priced at ₹178 to ₹271, with 3

variants.

- **Dabilong**: Manufactured by Micro Labs Ltd, priced at ₹262 to ₹334, with 2 variants.
- **Dabipla**: Manufactured by Cipla Ltd, priced at ₹187 to ₹324, with 3 variants.[24]

CONCLUSION

Direct oral anticoagulants (DOACs) have changed how we manage and prevent blood clots.

They are easier to use than older blood thinners because they do not require regular blood tests. Recent trends show that DOACs are being used in more patient groups and health conditions, and new reversal agents have made them even safer in emergencies.

While challenges remain—such as choosing the right dose for people with kidney problems, very high or low body weight, or multiple medications—DOACs continue to improve the overall safety and convenience of anticoagulation therapy. As research grows, DOACs are expected to play an even bigger role in providing effective and patient-friendly treatment for preventing and treating blood clots.

Direct Oral Anticoagulants (apixaban, rivaroxaban, dabigatran, and edoxaban) have largely replaced VKAs as the first-line treatment for many conditions, including stroke prevention in atrial fibrillation (AF) and treatment/prevention of venous thromboembolism (VTE).

The main advantages driving this shift are

- Predictable pharmacokinetics and a wider therapeutic window.
- No requirement for routine laboratory monitoring (like INR testing).
- Fewer drug and food interactions compared to VKAs.
- Improved safety profiles, particularly a lower incidence of major and intracranial bleeding.

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