Appendices

Appendix A

Table 1. Effective Treatments for PE Based on Benchmark Case Reports. Case severity: (High/Low/Medium)

Number	Patient Age + Sex	Past Medical History	Presentation on Admission	Mortality (Yes/ No)	Treatment Administered	Details of the Treatment	Success Rate/ Remarks	Reference
1	63 F	2-year dementia with behavioral and psychological disturbances; chronic hypertension under treatment.	Delirium superimposed on dementia (past 2 years). Developed fever and tachycardia, tachypnea, and shortness of breath	Yes	Mortality before treatment could be administered	Mortality before treatment could be administered	Transthoracic echocardiography revealed a hypokinetic and enlarged right ventricle, suggestive of massive PE. Patient had a sudden cardiac arrest. Symptoms were mistaken for COVID-19 associated pneumonia.	[1]
2	26 M	No significant past medical history	Sudden collapse followed by spontaneous recovery. Extreme weakness, dyspnea, diaphoresis and tachypnea; but no chest pain or palpitations.	No	No thrombolytics started, administered anticoagulant therapy	Unfractionated heparin and oral anticoagulant administered. Oral warfarin administered post-discharge.	No recurring PE observed during long-term 4-month follow up.	[2]
3	50 M	3-year gout; recent treatment causing leg weakness; 20-year varicose veins; 10-year fatty liver; atrial fibrillation treated 3 years ago; family history of hypertension, diabetes, pulmonary embolism, and coronary heart disease.	Collapse due to breathlessness and amaurosis. Patient experienced dyspnea, shortness of breath, chest pain and fatigue after waking up. Patient experienced no palpitations.	No	No thrombolytics started, administered anticoagulant therapy via subcutaneous enoxaparin sodium injection. Oral warfarin and heparin were also prescribed and overlapped.	Patient reported no dyspnea, unconsciousness, breathlessness and chest pain after treatment	Pulmonary embolism resolved and no recurrence observed in 6-month follow up.	[3]

4	27 M	DVT	Severe chest pain, blood in sputum and swollen lower left extremity	No	Patient condition assessed as low risk PE; therefore, anticoagulant therapy administered.	Anticoagulant therapy continued post-recover.	Chest pain resolved after 5 days of initial treatment; anticoagulant continued after recovery.	[4]
5	~70 M	Stroke 3 years ago, moderate aphasia, gout, dyslipidemia, hypertension.	Sudden dyspnoea, tachypnoea and hypoxaemia experienced after being admitted to the hospital for a fall.	No	LMWH was initially administered, but the patient remained hemodynamically unstable. Thrombolytics advised after the patient 's condition was assessed to be severe.	Half-dose thrombolysis administered as the patient was at high risk for adverse bleeding due to their age and previous administration of enoxaparin that same day.	Normal right ventricular function and no pulmonary hypertension following thrombolysis treatment.	[5]
6	21 M	No significant past medical history	Swelling of the left leg with pain for a 1-month duration. Non-productive cough & exertional shortness of breath. Tachycardia	No	Started with intravenous heparin & discharged with rivaroxaban	Endovascular stenting may be contemplated to prevent recurrence of VTE or its long-term sequelae	The patient is stable and currently following up with the department of internal medicine	[6]
7	68 M	Exertional dyspnoea and dyspepsia for 4 days; diabetic, hypertensive, non-alcoholic.	Respiratory distress along with sweating for 2 hours, exertional dyspnoea for the last 4 days. Pansystolic murmur over the tricuspid area	No	Treated with streptokinase. Further echocardiography performed. Anticoagulant therapy was given, enoxaparin sodium, in conjugation with rivaroxaban.	Streptokinase was given as a loading dose, followed by a dose over 72 hours. Blood test showed low protein C level. Further echocardiography was performed and it revealed normal right heart function	Patient recovered from the acute stage.	[7]
8	59 F	Psoriatic arthropathy, 6 years post-menopause and family history of blood clots and stroke.	Breathlessness and chest pain for 2 weeks	No	Anticoagulation therapy: Treatment with unfractionated intravenous heparin	For hemodynamically stable patients, the optimal management strategy is poorly defined. Administration of only anticoagulation therapy was strongly debated, but in this case, it was fortunately successful.	After treatment, on day 13 the patient was discharged with no evidence of intracardiac thrombus and normal PA pressure.	[8]
9	36 M	Post-COVID-19 vaccine chest pain; admitted to pneumology. 10-year smoker, no hypertension, diabetes, or atrial fibrillation.	Repeated shortness of breath and chest tightness	No	Anticoagulant therapy and interventional thrombolysis were performed.	Patient was discharged on rivaroxaban	Suspected thrombosis linked with taking Olanzapine (second gen antipsychotic drug)	[9]
10	37 F	History of overweight	Syncope and shortness of breath. Fatigue and pain in	No	Thrombolytic therapy with Tenecteplase	Discharged on the fourth day.	Patient was 29 weeks pregnant, and diagnosis can be delayed by	[10]

			the side and back of the left thigh. Hypotensive, tachycardic, hypoxemic				reluctance to expose the fetus to ionizing radiation.	
11	16 F	Two orthopedic surgeries in infancy, two pregnancies, one miscarriage, recent cesarian at 20 weeks for placental abruption with deceased fetus.	Dyspnea, chest pain and loss of consciousness.	No	Anticoagulant therapy: unfractionated heparin, subcutaneously LMWH prescribed for three months. Oral therapy with acenocoumarol.	Patient discharged in good general status with the recommendation of life-long anticoagulation therapy.	Patient had thrombophilia.	[11]
12	40 F	No significant past medical history	Stair fall at a cranio-thoracic impact point, that leads to transient amnesia immediately after.	No	Anticoagulation based on LMWH.	Monitored for symptoms of intracranial hemorrhage with repeat CT scans. Discharged on day 13.	Post-traumatic thromboembolism remains a challenging condition. Decision to anticoagulate post traumatic PE is controversial, because it can lead to intracranial hemorrhage expansion.	[12]
13	51 F	1-year well-controlled hypotension; MRI revealed left supraspinatus tear; arthroscopic repair on day 3 after hospitalization.	No discomfort or swelling after the shoulder arthroscopy, but 12-hour post-op, found a decrease in arterial oxygen saturation	No	3-month treatment of rivaroxaban	--	Suspected that cases occurred due to abnormal decrease in partial pressures of oxygen and arterial oxygen saturation. Talks about early detection of shoulder arthroscopy associates PE.	[13]
14	75 F	History: 2-year atrial fibrillation, 6-year COPD. Exam: Hawkins sign (right shoulder), limited shoulder motion. MRI: partial supraspinatus tear.	PE happened 24h after shoulder arthroscopy. Asymptomatic PE	No	Anticoagulant treatment with LMWH, calcium and then oral rivaroxaban	After 10h, the patient presented with shortness of breath, which resolved after LMWH calcium treatment.		
15	38 M	History of obesity	Sudden-onset chest pain with breathlessness for four hours, associated with palpitations.	No	Thrombolysed with alteplase. Followed with heparin and NOAC	Follow-up for thrombophilic state, revealed: intermediate hyperhomocysteinemia.	---	[14]
16	45 F	No known comorbidities	Left lower limb pain and swelling for one week.	No	Parenteral anticoagulation with LMWH and warfarin. IVC filter was also placed.	Although treated for leg clots, the patient developed PTE after 6 days on anticoagulants, thus an IVC filter was placed.	Found presence of heterozygous factor V Leiden mutation.	

17	28 F	History of polycystic ovarian disease, receiving treatment.	Three episodes of loss of consciousness after fall in a day. Chest pain, palpitations and breathlessness. History with leg swelling and pain in the calf for about a week.	No	Thrombolytic therapy with intravenous streptokinase. Anticoagulation with LMWH for 3 months, and follow-up with NOAC (apixaban).	--	This is a case of high-risk PTE.	
18	24 M	The patient was a bodybuilder following severe diet restrictions.	Breathlessness for 3 days, palpitations, and pain in lower extremities for 5 days.	No	Anticoagulation with heparin	Follow-up procoagulant workup showed a decrease in protein C and S levels.	Blood tests found severe anemia. The patient was a bodybuilder and was following severe diet restrictions.	
19	42 M	No known comorbidities	Breathlessness for 5 days and palpitations for 1 day.	Yes	Anticoagulation was started immediately with LMWH. Then, thrombolysis was started with streptokinase	PE extended to the right ventricle. thrombolysis was started and was planned for emergency mechanical thrombectomy.	Patient was a walk-in in the outpatient department. Before the emergency procedure, the patient collapsed with hypotension and desaturation and was declared dead.	
20	55 F	10-month history: recurrent shortness of breath, dizziness; diagnosed with bronchitis, history of varicose veins for 10 years.	Chest distress and dyspnea for 2 days and then abrupt loss of consciousness for 1 h. History of bronchitis, and varicose veins for 10 years.	No	Anticoagulant drug (rivaroxaban, an Xa inhibitor) and an antiplatelet agent (clopidogrel, an ADP receptor inhibitor) was prescribed with a β-blocker and atorvastatin. 16 months later, angiotensin-converting enzyme inhibitor was started.	Over the first 10 months, the embolus gradually shrunk, but then started expanding. Combined anticoagulation and antiplatelet therapy were started.	Found acute myocardial infarction (AMI) and a cerebral infarction. Case of arteriovenous embolism. (rare case)	[15]
21	69 F	History of hypertension (peak BP 160/90 mmHg); cerebral infarction one year ago with no lasting effects.	Syncope accompanied by gait disturbance + history of hypertension. No resolving symptoms from cerebral infarction 91 yr ago), and myocardial markers increased.	No	Intravenous thrombolysis. Rivaroxaban po was continued for anticoagulation. Administration of mannitol to lower intracranial pressure.	Patient discharged on day 11. One week later, the patient had a cerebral hemorrhage (suspected due to intravenous thrombolysis). rivaroxaban tablets were discontinued.	Patient was initially misdiagnosed to have a cerebral infarction.	[16]

22	48 M	No relevant medical history	Left back pain, nausea and diaphoresis.	No	Anticoagulation therapy with heparin. Discharged with apixaban on the 4th day.	Initially given intravenous infusion of acetaminophen	Testing also revealed a Foramen Ovale, and a Renal Infarction.	[17]
23	64 F	Atrial fibrillation and prior varicose vein surgery, underwent total right hip replacement, anxiety and tachycardia.	1h later, when the surgery was finished, the oxygen level decreased from 100 to 66%.	No	LMWH, calcium was given and an IVC filter was placed.	Anticoagulation, alleviation of pulmonary hypertension and atrial fibrillation therapy was continued. The IVC filter was removed after 20 days.	APE occurred during orthopedic surgery under general anesthesia.	[18]
24	46 M	Mild Covid-19, psychosis. No family history of thromboembolisms	Acute dyspnea and left-sided thoracic pain for several hours. Pain and swelling of the right calf was reported 2 days prior.	No	Anticoagulant therapy with weight-adjusted LMWH. Switched to edoxaban after 5 days	Received ceftriaxone, but the patient experienced hallucinations, so switched to risperidone after 3 days. Discharged with edoxaban for 3 months. Within a few hours, the patient experienced seizures and was readmitted. Patient was treated with lorazepam and valproate.	SARS-CoV-2 infection can increase the prevalence of thromboembolisms. Diagnosed with dissociative disorder and adaptive disorder.	[19]
25	70 M	Hypertension, diabetes, dyslipidemia. Ischemic heart disease- had multiple percutaneous coronary interventions previously.	Fever and feeling unwell for the past 2 days.	No	Anticoagulant therapy; Subcutaneous Fondaparinux, then oral warfarin for 3 months.	Presented with atypical pneumonia symptoms and suspicion of PE. PE was confirmed with computed tomography pulmonary angiography scan.	Most DNPE cases happen in people with a chest injury, with no evidence of a DVT.	[20]
26	81 M	Pneumonia associated with severe SARS-CoV-2 and acute pulmonary embolism Hypertension, possible chronic lung disease	Cough, shortness of breath at rest and dyspnea when active, fever, substernal chest pain on the left side which increased when laying down, patient in respiratory distress on arrival to hospital	Yes	Initial treatment of PE with enoxaparin.	Patient admitted to the ICU for 3 days then transferred to the general medical floor. Patient experienced pleuritic chest pain episodes and hypoxemia-associated aspiration events along with significant bleeding from the nose and mouth. Patient transitioned to palliative and end-of-life care.	Patient passed away in palliative care on the 16th day of their hospital stay.	[21]
27	27 F	Pregnant (17w gestational age)	Dyspnea, fatigue, tachycardia, PM edema, DVT	No	Anticoagulant therapy during pregnancy with LMWH. Fragmentation plus catheter-guided thrombolysis (CDT) conducted. Inferior vena cava filter placed.	Initially, LMWH administered at 1 mg/kg every 12 hours. Patient admitted to ICU.	Patient alive and continually monitored at outpatient centre post-discharge. Pregnancy terminated via abortion (unrelated to PE or CDT).	[22]

28	37 F	Pregnant (11w gestational age)	Dyspnea, fatigue, pleuritic pain, tachycardia, PM edema	No	Anticoagulant therapy during pregnancy with LMWH. Fragmentation plus catheter-guided thrombolysis conducted. Inferior vena cava filter placed.	Initially, LMWH administered at 1 mg/kg every 12 hours. Patient admitted to ICU.	Patient alive and continually monitored at outpatient centre post-discharge. Pregnancy terminated via c-section. Patient reported dyspnea after 1 year and underwent echocardiography which displayed intermediate risk of pulmonary hypertension (PH), however the concern was alleviated through a heart catheterization which ruled out chronic thromboembolic PH.	
29	33 F	Pregnant (22w gestational age)	Dyspnea, pleuritic pain, tachycardia	No	Anticoagulant therapy during pregnancy with LMWH.	Initially, LMWH administered at 1 mg/kg every 12 hours. Patient admitted to ICU.	Patient alive and continually monitored at outpatient centre post-discharge. Pregnancy terminated via transvaginal delivery.	
30	18 F	Pregnant (32w gestational age)	Dyspnea, tachycardia	No	Anticoagulant therapy during pregnancy with LMWH.	Initially, LMWH administered at 1 mg/kg every 12 hours. Patient admitted to ICU.	Patient alive and continually monitored at outpatient centre post-discharge. Pregnancy terminated via transvaginal delivery.	

Appendix B.1: Drug Delivery Systems in Nanomedicine

Currently, nanomedicine applications encompass several scopes of healthcare, below we will discuss the general mechanisms of injectable nanomedicines, biosensors, nanocarriers and wearable technologies.

Targeted drug delivery

The main concern presented within heading 3.1 is the widespread effect of therapeutic drugs on the body. This insists on a better mechanism for targeting therapeutics to improve efficiency and subsequently reduce adverse effects. Thankfully nanomedicine presents a promising scope for targeting diseased cells in the body, while minimising the effect on healthy ones. NPs can be uniquely engineered with the specific design, materials and targeting strategy in their construction to carry out this function [1].

Controlled Release Systems

Controlled release systems (CRS) are a distinct branch of drug delivery systems that aim to deliver a drug at a predetermined rate and time in the body. Their purpose is to maintain a constant drug concentration in the body, and reduce the frequency of administration [2]. Nanoparticles can be useful for CRS purposes, as they have been shown useful in cloaking abilities of drug conjugates from lysis, as well as enhancing drug solubility and stability for a long-term effect [3].

Nanocarriers

Nanocarriers entail the delivery of drugs or other therapeutics to a target site via nanotechnology [4]. Conventional drug delivery methods require severe attention to detail and precision in order to maintain the therapeutic range of a given drug and ensure that only the site of interest is being affected [5]. By employing nanocarriers, treatments can be transported or "carried" directly to the site of interest and be released in a controlled manner [4]. Nanocarriers also alleviate bioavailability concerns due to the availability of various different types, such as hydrophilic and hydrophobic nanocarriers, which can effectively deliver the drug to the target site [4]. The release of a therapeutic from nanocarriers can either be a continuous process or triggered by external stimuli in the environment of the carrier [4]. These methods can also be leveraged to ensure the correct therapeutic index is maintained when administering the drug [4].

Magnetic NPs

Magnetic NPs are a class of NPs that can be manipulated with magnetic fields. This magnetic ability is usually achieved by manipulating the coating of NPs, furthermore the outer layers can be designed to bind to a specific target molecule. These particles have a promising scope within the biomedical field in aiding to diagnosis and treatment of diseases. For our purpose, magnetic NPs have been shown useful as drug carriers and as contrast agents in MRI imaging [6].

Appendix B.2: Nanomedicine-Based Devices

Biosensor Technology

Biosensors are components that read biological signals and translate them into electrical signals in order to convey crucial information [7]. The components involved in this process are analytes, bioreceptors, transducers and displays, which all function together to measure and convert biological signals to electrical ones [8]. Various types of biosensors exist such as magnetic biosensors, thermal biosensors, tissue and enzyme-based biosensors and more [7]. These sensors can be utilised for various applications such as food safety monitoring and medicine [8]. Presently, biosensors are composed of many different types of entities such as microbes or nanomaterial polymers [8]. These devices provide a non-invasive way to detect biological signals by ensuring specific recognition between an analyte of interest and the receptor on the biosensor itself [8].

Wearables

Wearables are devices that can be worn by the designated audience in order to fulfil a certain purpose, which in a medical context might be the monitoring of vital components in the body [9]. In a nanomaterial context, wearables are often paired with biosensors so that biological responses can be detected and aptly measured in real-time [9]. As with biosensors, wearable technologies provide a non-invasive way to obtain real-time results [9]. For example, the device may be used to monitor a patient's vitals at any given moment, or even as a way to administer drugs directly to the site of interest to limit side effects and adverse interactions [9].

List of Abbreviations Used for Appendix A and B

PE: pulmonary embolism

VTE: venous thromboembolism

MRI: magnetic resonance imaging

CT: computed tomography

COPD: chronic obstructive pulmonary disease

NOAC: novel oral anticoagulant

IVC: inferior vena cava

PTE: pulmonary thromboembolism

AMI: acute myocardial infarction

BP: blood pressure

DVT: deep vein thrombosis

VTE: venous thromboembolisms

LMWH: low molecular weight heparin

CDT: catheter-directed thrombectomy

nanoSTAT: Nanoscale self-titrating activatable therapeutic

GC: glycol-chitosan

AuNPs: gold nanoparticles

tPA: tissue plasminogen activator

MNP: magnetic nanoparticle

Chitosan-MNP: chitosan-magnetic nanoparticle

tPA: tissue plasminogen activator

USPIO: ultrasmall superparamagnetic iron oxide

Fols: fullerenols

PEG: polyethylene glycol

MMB: magnetic microbubbles

DPN: discoidal polymeric nanoconstruct

TDN: Thrombosis detecting nanosensor

uPA: urokinase-type plasminogen activator

nUK: nanogels that react to ultrasound waves

NP: nanoparticle

CRS: controlled release system

Appendix A References

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