

What on earth is going on with this patient?

Liver Failure? DIC? Massive Blood Loss? Contaminated sample?

Haematology	20-11-2019 04:49	19-11-2019 23:29	19-11-2019 21:52	19-11-2019 16:30	19-11-2019 12:17	19-11-2019 04:17	19-11-2019 01:43
FBE							
<input type="checkbox"/> Hb	76					101	130
<input type="checkbox"/> WBC	23.69					29.29	29.13
<input type="checkbox"/> Platelets	102					208	241
<input type="checkbox"/> Hct	0.22					0.32	0.40
<input type="checkbox"/> MCV	87					91	90
<input type="checkbox"/> MCH	29.2					28.8	29.2
<input type="checkbox"/> MCHC	338					317	323
<input type="checkbox"/> RBC	2.60					3.50	4.45
<input type="checkbox"/> RDW	13.8					14.4	14.2
<input type="checkbox"/> Neutrophils	20.24					27.22	25.93
<input type="checkbox"/> Lymphocytes	1.90					0.80	1.89
<input type="checkbox"/> Monocytes	1.54					1.23	1.19
<input type="checkbox"/> Eosinophils	0.01					0.01	0.06
<input type="checkbox"/> Basophils	0.01					0.03	0.06
Morphology						* Comment	
Coagulation							
Anticoagulant?	Heparin	Heparin		Heparin	Heparin	None	Heparin, Not Sp
<input type="checkbox"/> INR	1.3	1.4		1.9	1.8	12.2	17.7
<input type="checkbox"/> PT	16.8	18.1		24.8	23.1	140.4	200.0
<input type="checkbox"/> APTT	* 69.6	* 51.9		* > 300	* 122.1	* 79.0	* 116.5
<input type="checkbox"/> Fibrinogen Level	2.4	2.0		1.1	1.1	< 0.1	< 0.1
<input type="checkbox"/> TCT	146.6	46.4		>300	>300	126.6	184.4
Coag Phone Comment	* Phoned Comm		* Phoned Comm		* Phoned Comm		* (c) Phoned Co
<input type="checkbox"/> D-Dimer	* 57.32						
<input type="checkbox"/> Anti Xa Heparin		* 0.02		* 0.75			

Presenting Complaint

Presumed PE

Cardiogenic shock

History of Presenting Complaint

Transferred from Sunshine Hospital

HPC

3/52 ago sustained torn ACL and relatively immobile since

OT 14/11 for repair at Warringal Private Hospital (Surgeon: Matthew Barnes)

- episode of hypoxia post operatively -> CXR with no changes -> discharged following day

Yesterday (18/11) - lightheaded and dizzy

- associated dyspnoea, called AV

- on AV arrival: moderate to severe resp distress, HR 170 sinus, hypoxic to 78% on 15L HM

- given atrovent and salbutamol nebs to no effect

On arrival to Sunshine ED:

- Bedside TTE: dilated RV, flattening of septum and D shaped RV

- placed on BiPAP to improve oxygenation

- On preparing for intubation and thrombolysis -> bradycardia and PEA arrest

- ROSC after 2nd cycle

- Recurrent PEA arrest

- Intubated during arrest

- Commenced on adrenaline and noradrenaline - reached 100mcg/min for both

- Thrombolysed with 50mg alteplase x2 (second dose 15 min after first dose) - 2053

- Referred for ?ECMO after 6th PEA arrest

VBG 2118: pH 6.5, pCO2 132, lactate 16, HCO3 11

Also received 10u actrapid + dex for hyperkalaemia

Lines - L femoral arterial line and L femoral CVC inserted at Sunshine

Family - Mother Linda, Dad Michael and Fiance Josh updated at Sunshine prior to departure

On arrival to Alfred ICU:

Initially on norad 8 adr 8

Became haemodynamically unstable after transfer to bed

Given metaraminol + increased adr/norad doses

Paralysed with cisatracurium

Bedside TTE by Dr Tan

Given total 1.5L IVF in addition to above

PMHx

?ASD, VSD as infant

- thoroughly investigated, no intervention

ICU LOS

Day 1

Inpatient Progress

Transferred from Sunshine hospital overnight following PEA arrest (45 mins downtime) secondary to massive PE

Issues:

1. Severe haemodynamic instability requiring high dose vasopressors

- Noradrenaline at 55mcg/min

- Adrenaline at 4mcg/min

- Milrinone, vasopressin

2. Large PE

- Multifocal pulmonary emboli involving the left main pulmonary artery distally, left lower lobar artery and its segmental branches (occlusive), and right lower lobe segmental arteries (non-occlusive) as described. CT features of right heart strain as described

- Right upper lobe combination of collapse and consolidation and left upper lobe patchy opacities may represent aspiration, contusion (CPR related) or less likely concomitant infection

3. Coagulopathy

- thrombolytised x 2 in sunshine hospital

- INR 12.2

- now on heparin infusion to treat massive PE

4. Metabolic Acidosis

- on CRRT

- given bicarbonate overnight

<https://www.wisegeek.com/what-is-fibrinolytic-therapy.htm>

Fibrinolytic therapy, also sometimes called “thrombolytic therapy,” is the use of special drugs to break up [blood clots](#) that are blocking a person or animal’s major artery. They are most commonly given to people after a heart attack or when a major [blood clot](#) has been discovered in a medical scan. In some places this sort of therapy is also being used, often on an “experimental” basis, for stroke victims. Health care workers sometimes refer to these types of drugs as “clot busters” because of their ability to break down and neutralize otherwise life threatening buildups. They do have the ability to save patients, but often work best either on minor clots or in the very early stages of an attack or episode. In many cases they are just one of many different medical interventions used to divert a health crisis.

How it Works

Blood clots happen when blood proteins coagulate to form a mass. Clots are really important on the surface of the body, as they can help wounds heal and prevent people from bleeding to death. Within veins and arteries, though, they can block blood flow, impede heart function, and sometimes even lead to death if they actually get into the [brain](#), heart, or other organs. Most healthy people perform some level of clot break down as a natural biological process. This isn’t always enough, though. When larger blockages form, pharmaceutical therapy is often required.

Drug-based “clot busting” is typically known as “secondary fibrinolytics,” since it is designed to augment what the body already does or should be doing. It works primarily by using the enzyme [plasmin](#) to engage in a process called “thrombolysis,” which basically means breaking clots into smaller, more manageable fragments that can be dissolved or otherwise neutralized. Aminocaporic acid and tranexamic acid are two of the most frequently used inhibitors in this process.

This sort of therapy is almost always administered intravenously, which is to say with either an injection or a direct line into a vein. Sometimes pills or capsules can also be used as supplementation. It is usually only done by a professional health care provider in a hospital or clinic; rarely if ever are these sorts of drugs prescribed for home use or self-care.

Use in Heart Attacks

The therapy is very commonly used in heart attack victims to slow the rate of the attack and to help stabilize blood pressure and flow. A heart attack, also called a myocardial [infarction](#), can happen when blood clots or plaque build up in and subsequently block a major artery that feeds blood to the heart. When blood flow to a specific area of the heart is stopped, that area usually starts to die. This is called ischemia. An ischemic heart can sometimes cause abnormal heart rhythms, which could lead to fainting or sudden death.

The best outcome for a heart attack victim occurs if fibrinolytic therapy is administered within 12 hours or less after the onset of symptoms. The efficiency of the drugs often depends on the age of the clot, since the longer a clot is present, the more [fibrin](#) it produces. “Fibrin” is a protein found in blood that helps it to clot. A clot that contains a lot of fibrin is harder to dissolve.

Pulmonary Embolisms

Blood clots can also be problematic even outside of the heart. A variety of deadly clots known as “pulmonary embolisms” are most commonly found in the lungs; on their own they can make it difficult to breathe and can cause chest pain, but the bigger risk is that they could break free and travel through the pulmonary artery directly into the heart, which often causes almost immediate death. Therapy in these cases can break down the clot before it has the chance to become deadly.

Potential Help for Stroke Victims

A number of medical experts are also experimenting with fibrinolytic therapy as a potential treatment for stroke victims. Strokes happen when the brain loses [oxygen](#) and cells die as a result. They are sometimes caused by blood and arterial obstructions, but not always; in any event, once they’ve happened it’s often too late to break up a clot or otherwise clear an obstruction. Still, some studies have shown that administering these sorts of drugs to stroke victims, particularly in the moments immediately after the event, *may* help restore function and improve recovery time.

Risks and Side Effects

Drugs used in fibrinolytic therapy often have a high risk for causing severe bleeding. As a result they shouldn't be given to patients who may have brain cancers, active internal bleeding, or recent [trauma](#). Patients who have had major surgery within three weeks before treatment and pregnant women are generally not be given fibrinolytic drugs either.

Common side effects include low blood pressure, general feelings of [weakness](#), and low energy. Allergic reactions to the therapy’s proteins or other ingredients are rare, but can happen. In isolated cases patients can also develop [antibodies](#) to the therapy that prevent it from being effective if used repeatedly.