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Pulmonary Thromboembolism Perioperative Therapeutic Management

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SUMMARY

Pulmonary thromboembolism (PTE) in the perioperative field is –besides being devastating– a real therapeutic challenge, since the circumstances surrounding this pathology are very serious. In several occasions, we face the impossibility of performing a thrombolisis, due to an immediate postoperative state, or we have to take the patient to urgency surgery since he/she has developed pulmonary thromboemboli. This last fact complicates and reduces the therapeutic spectrum and it is associated to high perioperative mortality. This paper reviews the both the diagnostic approach of patients suffering from thromboembolism and the management strategies based upon evidence, with regard to effectiveness. It is described the most commonly employed medicines, such as heparins of low molecular weight, thrombolitics (streptokinase, alteplase), the hemodynamic management of the patients with (secondary) cardiogenic shock and the interventions, both surgical and percutaneous (embolectomy or catheter embolectomy). Finally, it is proposed a management and diagnosis algorithm for the surgical patient.

Key words: Pulmonary thromboemboli, embolectomy, cardiogenic shock, perioperative mortality.

RESUMEN

El tromboembolismo pulmonar (TEP) en el ámbito perioperatorio es – además de devastador – un verdadero reto terapéutico, dadas las circunstancias que rodean a esta patología. En muchas ocasiones confrontamos la imposibilidad de efectuar trombólisis debido a un estado postoperatorio inmediato, o bien debemos llevar a cirugía de emergencia a un enfermo que ya ha desarrollado tromboembolia pulmonar, lo cual complica y reduce el espectro terapéutico y se asocia con una alta mortalidad perioperatoria. Este artículo revisa tanto el abordaje diagnóstico del paciente con tromboembolismo, como las estrategias de manejo basadas en evidencia, en lo que a efectividad se refiere. Se describen los medicamentos más empleados como las heparinas de bajo peso molecular, los trombolíticos (estreptoquinasa, alteplasa, etc.), el manejo hemodinámico de los pacientes con choque cardiogénico secundario y las intervenciones tanto quirúrgicas como percutáneas (embolectomía o embolectomía con catéter). Finalmente se propone un algoritmo de manejo y diagnóstico en el paciente quirúrgico.

Palabras clave: *Tromboembolia pulmonar, embolectomía, choque cardiogénico, mortalidad perioperatoria.*

INTRODUCTION

Pulmonary thromboembolism (PTE) constitutes a true therapeutic challenge in the perioperative setting, given the circumstances that surround this pathology. The situation of being faced with impossibility to perform thrombolysis due to an immediate postoperative status -in the majority of cases- or when a surgical procedure is imminent in a patient who have already developed PTE illustrates how demanding this situation is.

It is generally known that PTE is responsible for 2 to 20% of postoperative deaths. However, fatal PTE has been reported in 0.8% of patients who undergo general surgery, 0.3 to 2.4% of patients who were submitted to hip arthroplasty, and 4 to 7% of patients with hip fracture [1].

To determine the dimension the problem, differences between the clinical picture of the *hemodynamically stable (HS) PTE and the picture that causes hemodynamically unstable (HU) PTE* must be established, as their mortality vary. In this regard, the mortality attributable to PTE for the first group of patients is between 1.8 and 4.1%[2].

As for the patients that develop hemodynamically unstable (HU) PTE, mortality ranges between 17.4% three months later as reported ICOPER study [3] and 31% of intrahospitalary mortality reported by the MAPPET study[4].

When hemodynamic instability (HU) occurs, two-thirds of the patients die within the first hour, but it is important to stress that only half of those deaths are attributable to massive emboli (emboli that occlude 50% of the pulmonary artery trunk or two lobar arteries, a phenomena called *anatomically massive embolism*) and the rest is caused by recurrent emboli or minor (submassive) thrombi.

In a study of perioperative mortality conducted 20 years ago, it was reported that mortality associated with PTE was 8.8% of total deaths and there was a decrease to 2.3% by 1980[5]. This decrease was undoubtedly due to an early suspected PTE and to a better knowledge of its physiopathogeny, along with the prophylactic use of low-molecular-weight heparins.

As is known, there are populations at special risk for perioperative thrombotic or embolic phenomena, for example, the population of obese patients. The occurrence of deep venous thrombosis (DVT) or PTE in this group has been reported to be 0.2 a 2.4% in spite of heparin prophylaxis [6].

Other adult patients at risk for DVT or PTE are: polytraumatized young individuals, immobilized patients, oncological or pelvic surgery patients, and pregnant women. In children, this risk is increased by lower limb traumatism, hydrocephalus with shunt valve, congenital cardiopathy, infections, or cancer [7].

Next, we will cover some physiopathogenic aspects related to the perioperative period that should be stressed in order to better understand this disease.

PHYSIOPATHOGENY

It is important to highlight some of the characteristics of the patients with DVT that can lead in the end to the development of massive PTE during the perioperative period, as it is known that in some risk populations it occurs even in spite of prophylactic measures.

Table I shows the correlation between the location of the DVT and the risk for developing PTE according to the location of the clot [8]. It highlights the fact that in patients with risk factors who undergo major surgery, DVT at the level of the calf can be observed in up to 80% of the cases and, among them, generally fatal massive PTE can occur in up to 5% (Table I).

It should be remembered that PTE displays a variety of clinical presentations that depend on the responsiveness of the right ventricle. This is how classical clinical manifestations such as cough, pleuritic thoracic pain, dyspnea and even cardiogenic shock or sudden death (fatal PTE) can occur.

Massive pulmonary thromboembolism is the name given to the presence of a clot in the pulmonary vascular tree that causes *right ventricular failure*. It is estimated to constitute 10% of all the variety of presentations of PTE. Unfortunately, this is the most frequently observed clinical manifestation in the perioperative period and not so with those which correspond with the classical and progressive clinical manifestations described throughout this article. This is the reason for us to address patients with clinical evidence of right ventricular failure and to discuss massive PTE and its therapeutic considerations in the following sections.

Figure 1 explains the clinical evolution in the presence of pulmonary embolism[2].

The severity of the clinical picture is plotted on the abscissa axis, highlighting the size of the embolus and the cardiovascular state. The mortality rate in function of the severity of clinical picture is shown on the ordinate axis.

First, it stands out that the size of the clot is a direct function of mortality, this is, the larger the embolus, the higher the mortality. However, when right ventricular failure is associated (gray box, Figure 1) a clot of large dimensions is not necessary for the event to be fatal and under these conditions the mortality is as high as 30%.

On the other hand, when *cardiac arrest* is the initial clinical manifestation of PTE, the associated mortality is 70%, and that of *sudden death* reaches 100%; this is probably because the cardiac arrest is generally intrahospitalary and witnessed, as opposed to the case of sudden death.

Regarding once again the patients with right ventricular failure, in the Figure 1 it is remarkable that the most frequent initial presentation in this group of patients is the cardiogenic shock status and, as previously mentioned, a

Table I. Risk for developing PTE according to the location of DVT and surgery type.

Risk level	DVT in calf (%)	Proximal DVT (%)	Clinic PTE (No massive)	Massive PTE
Low risk: Minor surgery in patients under 40 years without additional risk factors.	2	0.4	0.2	0.002
Moderate risk:				
• Minor surgery in patients with additional risk factors. • Surgery no major in patients between 40 and 60 years without additional risk factors. • Major surgery in patients under 40 years without additional risk factors.	10 – 20	2 – 4	1 – 2	0.1 – 0.4
High risk 1 (Properly speaking):				
• Surgery no major in patients over 60 years with additional risk factors (Prior DVT, cancer, hypercoagulability).	20 – 40	4 – 8	2 – 4	0.4 – 1.0
High risk 2: (Very high risk)				
• Major surgery in patients over 40 years with a history of DVT, cancer or a hypercoagulable state, hip surgery, knee ortoplasty, hip fracture, major trauma or spinal surgery.	40 – 80	10 – 20	4 – 10	0.2 – 5

Abbreviations: DVT= Deep venous thrombosis; PTE= Pulmonary thromboembolism
Source: Modified and Translated from: Geerts WH, Pinco GF, Heit JA et al. Prevention of Venous Thromboembolism. The Seventh ACCP Conference on Antithrombotic and Thrombolytic Therapy. Chest 2004; 126: 338S – 400S).

large size clot of is not always necessary for a state of shock to be triggered. This fact is of great importance when considering the type of treatment to be implemented given that, as will be reviewed later, not always have to resort to surgical embolectomy, such as when large clots are lodged in the main branch of the pulmonary artery or its primary branches; instead, catheter thrombolysis in medium caliber arteries may be performed.

It is important to highlight that in the preoperative period, specially in patients with risk factors (e.g., patients with obesity or cancer to undergo surgery), detailed anamnesis is indispensable to detect subclinical pictures or clinical pictures clinically compatible with PTE. In these cases, the diagnostic approach algorithms described in other communications must be followed [9-11]; however, as previously stated, most of the times the initial presentation is a state of shock or even cardiorespiratory arrest secondary to PTE, and this may be observed specially in the transoperative or immediate postoperative period.

In many cases, only clinical background that indicates an increased risk for PTE is available and this should be enough for the clinician to suspect the pathology, specially if it occurs in a patient with otherwise inexplicable cardiogenic shock, *e.g*, ischemic cardiopathy. This urges the clinician to install an hemodynamic monitoring system, which should minimally consist of a central venous pressure (CVP) catheter.

There are studies that have correlated the size of the pulmonary arterial obstruction with modifications in the value

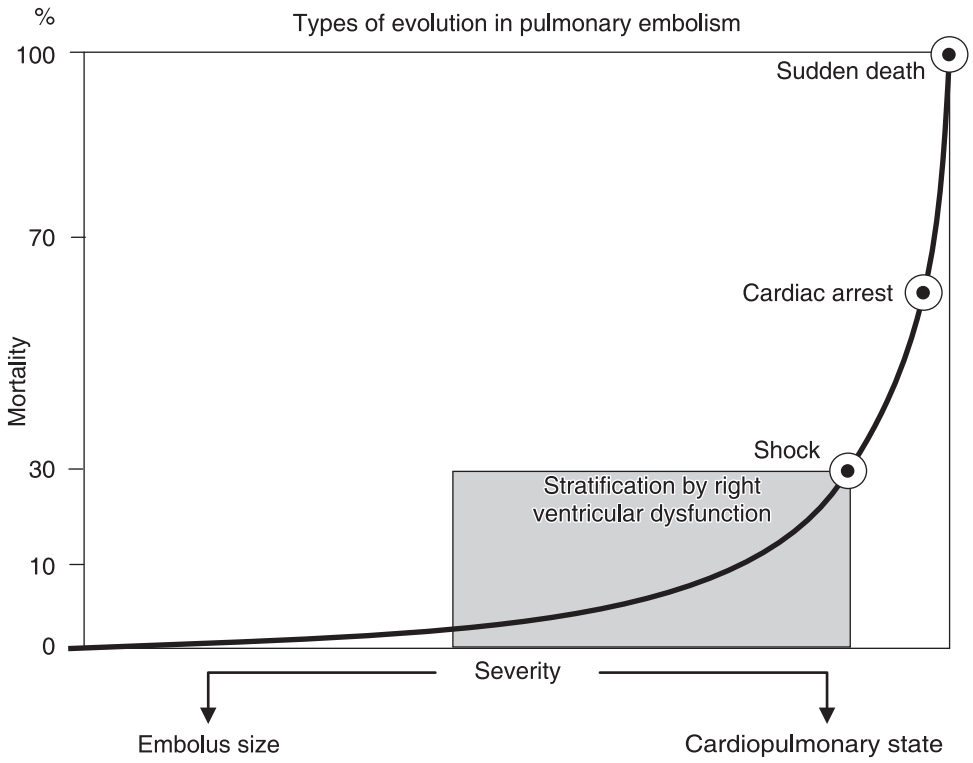

Figure 1. For analysis of the scheme see text *(Source: Modified and Translated from: Wood KE. Massive Pulmonary Embolism. Review of a Pathophysiologic Approach to the Golden Hour of Hemodynamically Significant Pulmonary Embolism. Chest 2002; 121: 877 – 905)*

of the CVP or even in the value of the mean pulmonary artery pressure (MPAP); however, it should be noted that these studies have been conducted in patients with normal cardiopulmonary function [9].

In this sense, it has been described that a MPAP of 22 mmHg is correlated with an obstruction of 30% of the pulmonary artery and a mean pulmonary pressure of 36 mmHg is correlated with an obstruction of 50% of the artery. When this degree of obstruction occurs, the coexistence of chronic cardiopulmonary disease should be suspected, specially if the MPAP exceeds 40 mmHg.

Regarding the CVP, it is over 5 mmHg in most cases of pulmonary artery obstruction larger than 35%. This implies that, in the absence of preexistent cardiopulmonary disease, the CVP does not increase substantially even in the case of a significant obstruction.

Since the administration of fluids should be closely monitored, the placement of a catheter to measure the patient's CVP it is considered to be indispensable. The pulmonary capillary pressure (PCP) obtained by means of a pulmonary flotation catheter may be normal and independent from the extent of the obstruction, as the observed failure is in the right ventricle.

In the presence of PTE, as the clot causes obstruction, a reactive increment in the MPAP is observed. If a pulmonary flotation catheter had been placed in the patient and a decrease in the MPAP is observed after the embolic event, this may mean either that the obstruction is in resolution or that the cardiac output is deteriorating.

Right ventricular failure (RVF) is clinically presented as state of shock and this may be an element that helps to discern the type of approach to limit the mortality associated with hemodynamically unstable (HU) PTE.

The physiopathology of RVF has been described elegantly in previous publications and it has been named the «vicious cycle» of the RVF[10].

In synthesis, when a decompensation of the right ventricle (RV) occurs due to an increment in the pressure (embolus) after its outflow tract, a concurrent increase of its residual volume is produced. This results in a decrease of the diastolic *distensibility* of the left ventricle (an effect denominated ventricular interdependence), which in turn leads to a decrease in preload. Finally, all of the above described changes originate systemic hypotension as a result of the reduction of the cardiac output and the coronary perfusion pressure, particularly on the RV. Eventually, this leads to ischemia which, along with the increment of the myocardial oxygen demand originated by the overdistension of the VD, perpetuates its decompensation.

It is evident that if the administered fluids are not calculated in a rational manner, a further deterioration of the RVF may be brought about, and there lays the importance of the CVP surveillance.

Nowadays, it is known that the patients who present HU (shock) and massive anatomical PTE have the highest associated mortality. This knowledge has contributed to develop a differentiated management algorithm, as will be discussed later.

Treatment varies according to the type of patient and the inherent complications of PTE; in the preoperative period, the patient with PTE may display two modalities of presentation: *the hemodynamically stable and the hemodynamically unstable (shock)*. The diagnostic-therapeutic approach may be performed taking into consideration the algorithm presented in Figure 2.

The most frequent presentation feature in this period is that of the hemodynamically stable patient that develops the «most characteristic» clinical picture, although it can sometimes be very subtle.

It is necessary to initiate heparinization at therapeutic doses in the surgical patient with hemodynamic stability (SH), if there are no contraindications for it, until PTE is ruled out or the extension of the event is known (Table II). This is because the risk for recurrent PTE is higher in early stages and because the recurrent PTE is the most frequent cause of death in the hemodynamically stable patient; therefore, *an early impregnation with heparin must* be started [11].

If the embolic event is not massive, effective heparinization is advisable (monitored with aPTT [activated partial thromboplastin time]) for 4 to 5 days, with non-fractioned heparin (NFH) and impregnation with warfarin since the first day; however, it has been suggested to use NFH for 9 to 10 days and, afterwards, initiate the oral intake of warfarin without this causing significant differences in outcome.

Regarding massive embolism, it is recommended to use NFH for 7 to 10 days and to initiate warfarin after 3 continuous days of presenting effective aPTT levels. The special considerations about the differences between NFH and low molecular weight heparins (LMWH) are presented in their corresponding subsection of this chapter.

On the subject of heparin, recent studies have yielded some remarks included in the recommendations issued by the ACCP (American Society of Chest Physicians) Consensus and are summarized below [23]:

- Short treatment with subcutaneous LMWH or IV NFH is recommended for patients with confirmed non-massive PTE.
- LMWH is preferred over NFH in patients with non-massive, acute and stable PTE.
- As initial treatment for patients with non-massive, acute and stable PTE, LMWH or NFH is recommended for at least 5 days.
- For patients with severe renal failure, the use of IV NFH is preferred over LMWH.
- If treatment with IV NFH is chosen, continuous infusion is recommended with the necessary dose adjustments to maintain proper aPTT values.
- If the patient receives high doses of NFH without attaining therapeutic values of aPTT, the use of anti-Xa levels is recommended as a guide for dosing. It is advisable to start using vitamin K antagonists (warfarin) since the first day of treatment with LMWH or NFH and to stop them when the NRI is stable and> 2.0.

Once established the heparinization and continuing with the diagnostic approach, two physiopathogenic primordial elements should be kept in mind: first, a massive embolism will not always cause HU, but a small embolism in the presence of pre-existent ventricular damage may cause it.

Second, DVT will not always be present when symptomatology related to PTE is displayed and PTE will not invariably develop if DVT exists. For this reason, and if the clinical picture and the risk factors support the diagnosis, it is advisable to perform a study that shows the anatomy of the pulmonary tree (V/Q scanning, spiral CT, angiography o angio-MRI); moreover, a lower extremity US Doppler may also be performed later.

If the imaging studies of the pulmonary vasculature do not evidence massive PTE and the patient continues to be hemodynamically stable, the treatment with heparin must be maintained and eventually initiate oral anticoagulation.

If the aforementioned studies suggest massive PTE (> 30% of perfusion defect), heparinization must be continued and it is necessary to perform an echocardiogram to determine the state of the right ventricle and to investigate whether there is dysfunction

The manifestations of right ventricle failure include: ventricular dilatation with hypokinesia and an increase in the

Table II. Contraindications to use heparin.

Absolute:
Previous bleending tendency
Bacterial endocarditis
Cerebral or subarachnoid hemorrhage
Malignant hypertension
Hipersensitivity to heparin
Relative:
History of major vascular procedures with placement of grafts
Recent surgery (< 10 days)
Pericarditis

(Source: The author).

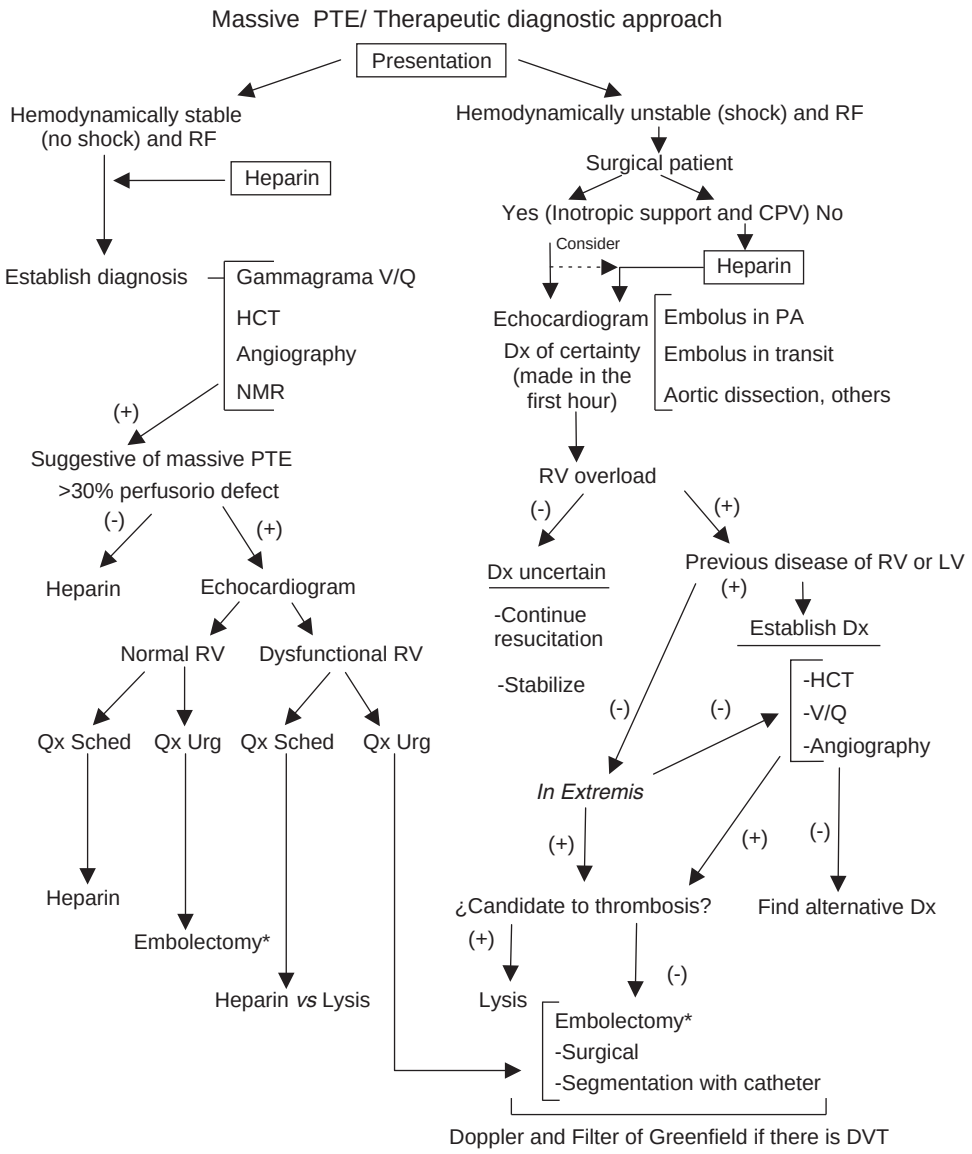

Figure 2. Therapeutic diagnostic approach to massive PTE *(Source: Modified from: Wood KE. Massive Pulmonary Embolism. Review of a Pathophysiologic Approach to the Golden Hour of Hemodynamically Significant Pulmonary Embolism. Chest 2002; 121: 877 – 905)* Abbreviations: RF = Risk factors; CVP = Central venous pressure; V/Q = Ventilation/perfusion; RV = Right ventricle; LV = Left ventricle; Qx = Surgery; Sched = Scheduled; Urg. = Urgente; PA = Pulmonary artery; Disecc. = Disection; HCT = Helical computed tomography; NMR = Nuclear magnetic resonance; DVT = Deep vein thrombosis; * see table *IV*

diameter RV/LV, paradoxical septal movement, dilatation of the pulmonary artery or tricuspid failure.

If massive embolism is detected (> 30% of perfusion defect) with normal right ventricle or if the embolism is submassive without ventricular failure and the patient is candidate for elective surgery, the heparin treatment must be continued and oral coagulation must be started later.

If it is not possible to postpone the surgery for a long period, the use of thrombolysis must be evaluated, provided that the waiting period can be longer than ten days. If data compatible with right ventricle failure are found, which in most cases suggests a massive embolic event, thrombolysis should be considered as well.

It is known that thrombolytic therapy is contraindicated in patients with recent surgery (< 10 days), patients who underwent neurosurgery, pregnant patients, or patients with cardiocirculatory arrest who required cardiopulmonary resuscitation maneuvers (Table III). It has been suggested that the use of

alteplase (tPA) may be a substitute for other thrombolytics (see the section on thrombolytics) in these clinical situations (12).

On the other hand, it must be considered that in several cases evaluated in the preoperative period, risk factors for or even clinical evidence of DVT can be detected. These factors are important to assess the effect of the anesthesiological technique over this pathology when defining a general surgical plan.

It is important to consider that a population suitable to perform a prophylactic maneuver is one in which a proximal thrombus has been found in the lower extremities and those, additionally, having high risk factors for the development of PTE. This clinical picture may be fatal in up to 5% of cases. This group of patients benefits from the placement of a vena cava filter and may benefit from techniques of regional anesthesia.

The incidence of DVT and PTE in hip surgery is known to have decreased in the last two decades as a consequence of the use of epidural anesthesia and catheter analgesia in the postoperative period [13]. The mechanism that explains this fact includes an increase in the blood flow to the lower extremities, a diminishment of the stasis an the activation of the coagulation factors.

On the other hand, a decrease in the incidence of DVT from 51 to 12% has been described in patients undergoing open prostatectomy [14]. Regarding other types of surgical intervention such as abdominal surgery, however, it is of notice that epidural anesthesia at a thoracic level, without vasodilatation of the lower extremities, does not diminish the occurrence of DVT in populations of moderate risk [15].

Even though they are outside the scope of this article, it is important to stress that there are certain conditions for the use of regional anesthesia in patients under prophylaxis with low-molecular-weight heparins. However, we refer the reader to other known publications on the subject [8,23].

In the case of an urgent surgery, with or without right ventricular failure, some of the therapeutic options that imply embolectomy, surgical or by means of catheters, must be contemplated. These resources will be discussed later in this work.

Patient with hemodynamically unstable PTE

A patient with hemodynamically unstable PTE presents in a state of shock and the surgical approach follows the right branch of the algorithm presented in Figure 2.

When the patient will undergo surgery, heparinization cannot be immediately started unless there are high chances that the surgery can be postponed. In this case, the use of heparin should be the initial step (Figure 2).

Being or not being an urgent surgery, in the case of a patient in state of shock and with suspected PTE, support with inotropics and control of fluids by means of a central venous pressure catheter must be initiated.

Table III. Contraindications to thrombosis.

Absolute
Active internal bleending
Stroke recent (< 2 months) or other active intracranial process
Relative majors
Recent procedures (< 10 days): Major surgery (includes neurosurgery), previous punsiones of blood vessels noncompressible
Acute gastrointestinal bleeding recent
Severe hypertension (systolic > 200 mmHg, diastolic > 110 mmHg)
Relative minor
Recent minor trauma, including CPR
Thrombus in the left cardiac cavities posibble (AF or mitral valve disease)
Bacterial endocarditis
Hemostatic defects including those associated with hepatic or renal disease severe
Pregnancy
Age over 75 years
Diabetic retinopathy

Abbreviations: CPR = Cardiopulmonary resuscitation; AF = Atrial fibrillation

Use of fluids

The management of fluids should be specially careful, given that a fluid overload is not well tolerated by these patients and their state may worsen. If the CVP is below 12 to 15 mmHg, it is advisable to compensate with cristalloids and avoid the use of colloid solutions as much as possible.

It is useful to reiterate that fluid repletion should not be started if a CVP catheter is not available to monitor the state of the right ventricle.

Use of Inotropic, Vasodilator and Vasopressor Agents

The support of the right ventricle almost simultaneously must be initiated in order to improve the myocardial perfusion and the state of shock.

It is essential to keep in mind that the dilatation and claudication of the RV cause ventricular interdependence, which in turn diminishes the cardiac output.

The decrease in the preload of the LV conditioned by the embolic obstruction causes a drop in the coronary perfusion, which along with the increase in the residual volume in the RV, compromise the myocardial oxygen consumption (VO_2M).

In the classical therapeutics, it has been recommended to use dobutamine as an inodilatator and norepinephrine as a vasopressor agent [11,16]; however, the current therapeutics has focused on drugs that do not compromise the VO_2M as adrenergic drugs do.

It must be taken into consideration that, in the unstable patient, the priority is to increase the perfusion pressure and, for this reason, the drug to be used primarily is norepinephrine according to the pressor response (the dose ranges from 0.08 to 1.1 micrograms/kg/min) and until reaching a minimal mean arterial pressure of 65 mmHg.

If there is a substantial increase of the CVP (> 15 mmHg), an inodilatator must be added to the therapeutic scheme. Conventionally, dobutamine[16] has been recommended as the drug of choice starting at doses of 5 micrograms/kg/min, performing down-titration until attaining a decrease on CVP with stability of the blood pressure. However, recent studies have questioned the use of this drug and other adrenergic agents for the management of the acute ventricular failure [17].

Our group prefers the use of norepinephrine in a dose-response scheme as a primary vasopressor, but using milrinone as inodilatator. As far as the dose that we used, it is of a loading dose at a reason of 50 micrograms/kg/minute slowly administered (for up to 30 minutes), starting the infusion at a reason of 0.3 micrograms/kg/minute.

The use of this phosphodiesterase inhibitor is founded on the fact that, on the patients who develop acute heart failure, the expression of beta receptors decreases and beta 1 receptors are specifically down-regulated[18]; for this reason, the inotropic response to the adrenergic stimulation is lower. Moreover, the use of dobutamine has been associated with an increased mortality at 6 months after its use for the management of acute episodes of heart failure [17].

Milrinone has been proposed as an alternative to dobutamine, given that it does not increase the VO_2M, and the studies regarding short duration infusion (48 to 72 h) have demonstrated an increase in survival rate at 60 days when compared to dobutamine or placebo. It must be considered, however, that a study with a large number of patients (OPTIME-CHF) reported a higher rate of mortality at 60 days when ischemia associated with heart failure presents [19]. This report includes patients with ischemic cardiopathy as a cause of cardiogenic shock, but it doesnot include the ischemia observed in PTE cases. Therefore, further studies are required to assess this issue; however, regarding the pathology that motivates this article, the results appear encouraging.

On the subject of vasopressor agents, it is desirable to count on alternatives that do not increase VO_2M. In this sense, our group has started the use of vasopressin in cases related to ventricular failure.

We have previously mentioned that the primary objective is to increase the perfusion pressure, which may be compromised by starting an inodilatator. Relatively recent reports give it a role in the therapeutic arsenal to vasopressin to correct the milrinone-induced vasodilatation [20].

In our clinical practice we use a dose of vasopressin (VP) between 0.0003 and 0.002 Units/kg/minute, performing dose titrationing until obtaining an adequate pressor response.

Particularly in the patient with catecholamine-resistant shock, we prefer the simultaneous use of NE and VP as the requirements of the first drug may be reduced, attaining a better response than that observed using norepinephrine as a single medication[21].

Drugs that reduce right ventricular preload and which have been classically used are hydralazine, nitroprusside, and/or prostaglandins. These drugs have several effects on the systemic hemodynamics, said effects are deleterious in most cases.

Up until two decades ago, there were no drugs without significant systemic side effects; this situation improved with the development of inodilatators such as dobutamine and even more with milrinone. However, the use of nitric oxide has been proposed, either by a non-invasive technique or by means of an endotracheal tube, as an strategy to improve the right ventricular failure on its acute phase, given its selectivity for the pulmonary vasculature and its null systemic effect [22].

As method of use, we recommend to start with 40 ppm monitoring the pulmonary pressure and continuing administration until obtaining an improvement on the echocardiographic findings of right ventricular failure. The admin-

istration of the gas can be interrupted when 5 ppm have been reached.

In our group, we used nitric oxide as a right ventricular function enhancer after thromboendarterectomy. We will discuss the characteristics of use in the corresponding section.

Finally, and once established the diagnosis of certainty by means of the element chosen from the Figure 2, it should be decided if the patient is a candidate for thrombolysis, since actually it is indicated only for hemodynamically unstable PET.

If the surgery can be postponed and the patient continues to be unstable or to have high requirements for inodilatators and vasopressors, thrombolysis should be resorted to; if this is not possible, embolectomy in any of its modalities is to be performed.

Thrombolytic therapy (TT) has been used since the decade of 1970 and it is currently accepted that the first hand indication for its use is the hemodynamically unstable PTE [23].

Both streptokinase and urokinase have similar thrombolyitc effects. The recommended dose of streptokinase is: 250,000 IU IV as a loading dose, followed by an infusion of 100,000 IU/h IV for 24 hours.

For urokinase, the recommended dose is: 4,400 IU/kg IV as a loading dose, followed by an infusion of 2,200 IU/kg IV for 12 hours.

Finally, for tPA, the recommended dose is 100 mg IV to be administered in 2 two hours (the simultaneous use of heparin is optional) [23]. Up until current date, there is no evidence that direct instillation in the thrombus through a catheter is superior to systemic administration [12,24].

It is important to underscore that invasive procedures must not be performed after the thrombolytic therapy has been started, since retroperitoneal hemorrhages have been reported. This applies even to patients who have undergone only femoral puncture. Moreover, 1% of intracranial hemorrhage is reported with the use of thrombolytics.

Another treatment modality is catheter embolectomy. This type of embolectomy was described in the decade of 1980 and, ever since, three types of catheter were developed. There is little world-wide experience on this procedure.

That is why it is considered that the use of this technique is not recommended, except for the extremely compromised patients that are not candidates to receive thrombolytic therapy, either due to their risk of bleeding or to their critical state [24-26]. In general, surgical embolectomy is still used in emergency situations and when other alternatives cannot be used such as in the patients who will undergo emergency surgery. The conditions to be met by the patient for the performance of this procedure are displayed in Table IV.

A variant of this procedure is the thromboendarterectomy for recurrent PTE and the most complications occur in this procedure. in both both primary embolectomy and endarterectomy, associated mortality is very high. When an extracorporeal circulation system is readily available, the mortality associated with embolectomy ranges between 10 and 75%. In patients whose presentation variant was cardiocirculatory arrest, the mortality ranges between 50 and 94%.

Our group has reported the successful performance of pulmonary thromboendarterectomy in a patient with recurrent PTE using nitric oxide and milrinone for transoperative and postoperative support but the casuistics in this subject is limited. However, as shown in Figure 3, the nitric oxide effect on the pulmonary hemodynamics is very relevant and it may constitute an alternative of management to improve the survival rate in this group of patients [27].

Nitric oxide was used on the outflow of the extracorporeal circulation pump as the ventilation reinitiated, starting at 40 ppm. This dose is continued for the first 12 hours, beginning to decrease by 5 ppm every 4 hours, performing blood gas analysis to supervise oxemia and monitoring pulmonary pressure. The mean time of use is 72 hours before withdrawal of the nitric oxide.

As with the catheter embolectomy, the evidence-based recommendation for surgical embolectomy restricts its use to extremely compromised patients that are not candidates to receive thrombolytic therapy, either due to their risk of bleeding or to their critical state [24-26].

As mentioned in the section of Physiopathology, when a preoperative patient has DVT, specially in the proximal region of the lower extremities, the probability that these thrombi are released is very high. This makes the placement of a vena cava filter is highly recommendable [28]. Transoperative PTE is object of case reports with variations on the outcome; however, it can be stated that every reported case presented with HU.

The typical presentation of PTE in the anesthesized patient is characterized by cyanosis, hypoxemia, hypocapnia, hypotension, neck venous distension, pulmonary hypertension, and increase in the CVP. In the non-intubated patient, wheezing and decrease in the thoracic distensibility may be evidenced. If the patient has a pulmonary flotation catheter, then PCP is normal and it is decreased only when the patient suffers from previous cardiovascular disease. In this type of patient, the pulmonary flotation catheter is generally used during the transoperative period.

Depending on the characteristics of the clot and the RV function, hypoxemia, hypocapnia and hypotension may not develop. The therapeutic recommendation of choice for the transoperative PTE is the embolectomy in extracorporeal circulation pump [27].

As we mentioned previously, the PTE presents in up to 20% of the general surgical procedures in the *postoperative period*.

Table IV. Conditions required for the patient to be subject to surgical embolectomy.

1. Massive pulmonary thromboembolism (if possible, documented by angiography).
2. IH (shock) despite of using heparine and haemodynamic support.
3. Thromboembolic therapy failure or contraindication to be used.

(Source: Büller HR, Ahnelli G, Hull RD, Hyers TM et al. Antithrombotic Therapy for Venous Thromboembolic Disease. The seventh ACCP Conference on Antithrombotic and Thrombolytic Therapy. Chest 2004; 126: 401S – 428S).
Abbreviation: IH Induced hypothermia.

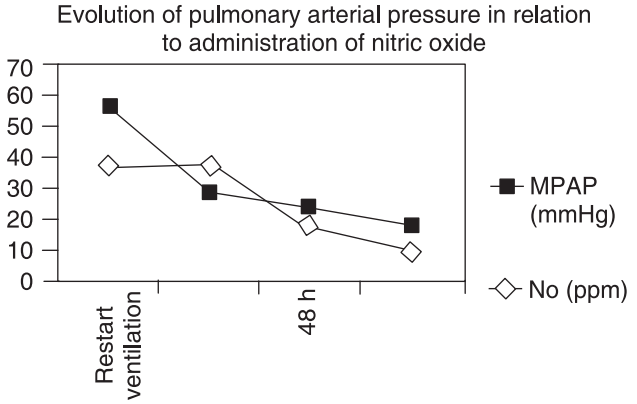

Figure 3. Evolution of the mean pulmonary artery pressure in the postoperative of pulmonary thromboendarterectomy *(Source: Mateos M, Tamariz-Cruz O, Palacios-Macedo A. Management of patient undergoing pulmonary thromboendarterectomy: Role of nitric oxide. A case report. Rev Mex Anest 2003; 26: 21 – 23).*

In general, the approach is similar to that recommended for the patient in the preoperative period. The limitation is that when thrombolysis is reached in the decision tree and the patient is unstable, the only resolution options are those corresponding to embolectomies, given that the administration of thrombolytic drugs is not advisable.

Once the patient has developed PTE in the postoperative period and at the prospect of a recurrent embolic phenomenon, lower extremity Doppler imaging should be performed.

If thrombi are found or if the patient has a preoperative diagnosis of a prothrombotic condition, it is advisable to place a vena cava filter since the recurrence of the clinical picture may be fatal. This must be taken into consideration before the potential recurrent heparinization or thrombolysis in the immediate postoperative period (28).

CONCLUSION

In this article we have stated most of the situations related to the presence of PTE in the perioperative period; the possible settings, proposals for their solution, and their diagnostic strategies were defined.

It should be kept in mind that the use of an adequate clinical judgment is indispensable, as surgical patients, due to the characteristics of their condition, are more likely to present complications secondary to poorly supported decisions. Unfortunately, in this period, most of the events involve massive thrombosis clinically associated with HU (shock) and the decision making process may cause that the patient to undergo surgical embolectomy.

On this basis, it is important to underscore the preventive strategies for both DVT and PTE, specially when the patient has the diagnosis of DVT in the preoperative period. *In this regard, the reader is referred to the Consensus sponsored by the* Colegio Mexicano de Anestesiología *in relation to the management guidelines for the diagnosis and treatment of the DVT, in order to broaden the view not only with aspects related to the surgical field, but also with aspects related to the medical scope* [29].

It is very important to acknowledge the supportive therapeutic considerations for the patient with HU (shock), because on that depends the possibility of clot lysis and thus salvage of the surgical patient.

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