07. ACUTE COR PULMONALE COMPLICATING ACUTE RESPIRATORY DISTRESS SYNDROME

In this setting, two associated factors combine to raise right ventricular outflow impedance :

» underlying pulmonary disease, which usually causes permanent diffuse arteriolar obstructions(15) ;

» assisted ventilation (16), which results in microvascular, intermittent or permanent obstructions, by elevation of transpulmonary pressure (17, 18).

Bedside echocardiography provided the first description of this complication of ARDS, at a time when high tidal volumes (13 ml/kg) were used (19).

Film 20 : First echocardiographic description of ACP in a female patient with ARDS. This example is taken from a publication (19). TTE in a four-chamber apical view shows marked dilatation of the right ventricle and right atrium.

Film 21 : In the same female patient as in *film 20,* the parasternal short-axis view of the LV reveals paradoxical septal motion.

The frequency of this complication was 61% then, a value close to mortality of the syndrome. It is now known that these tidal volumes, and the high plateau pressure they induce, are excessive. Reduction in plateau pressure to below 30 cm H 2O significantly reduces the frequency of ACP to about 25% (9). he frequency of this complication was 61% then, a value close to mortality of the syndrome. It is now known that these tidal volumes, and the high plateau pressure they induce, are excessive. Reduction in plateau pressure to below 30 cm H 2O significantly reduces the frequency of ACP to about 25% (9).

Film 22 : In this patient on controlled ventilation because of ARDS due to extensive lung disease, TEE revealed ACP with the RV dilated along the long axis and paradoxical septal motion on the transgastric short-axis view. The plateau pressure generated by the ventilation in this patient was 33 cmH2O, which is excessive. The systolic blood pressure was 92 mmHg.

Film 23 : In the same patient as in *film 22,* a few hours after reduction of tidal volume lowered the plateau pressure to 26 cmH2O, TEE shows the virtual disappearance of ACP. The systolic blood pressure is now 123 mmHg.

The onset of ACP during ARDS is generally more gradual than during PE and is observed after a certain time on assisted ventilation (9). In certain patients ACP :

- » may occur on introduction of assisted ventilation,

Film 24 : In this spontaneously breathing patient hospitalized because of sepsis of pulmonary origin, TTE visualized an undilated RV, indicating normal right ventricular function.

Film 25 : In the same patient as in film 24, mechanical ventilation is responsible for the onset of ACP, revealed by TEE.

» or can be triggered by untimely adjustment of respirator settings

Film 26 : TEE in a patient on controlled ventilation for ARDS caused by extensive lung disease. The examination was done at a PEEP of 5 cmH2O with a plateau pressure of 27 cmH2O. There was mild systolic overload of the RV. Systolic blood pressure was 135 mmHg, heart rate 100 bpm and systolic index of the RV 23 ml/m2 (Doppler measurement in the pulmonary artery).

Film 27 : Because of persistent and marked hypoxemia, a high PEEP (14 cmH2O) was tested. At the same time, the tidal volume was lowered to maintain the same plateau pressure (27 cmH2O). The TEE probe was left in place, frozen. Five minutes after these adjustments, the heart rate was 121 bpm, systolic blood pressure 115 mmHg, and systolic index of the RV 12 ml/m2. TEE now clearly shows ACP.

Film 28 : Given poor hemodynamic and right ventricular tolerance, the initial settings were restored (PEEP 5 cmH2O). In a few minutes, systolic blood pressure increased to 130 mmHg, systolic index of the RV to 23 ml/m2, and heart rate dropped to 110 bpm. TEE demonstrated a clear decrease in right ventricular systolic overload.

In some patients, the later onset of ACP indicates a fibroproliferative phase, which can be arrested by corticosteroid therapy.

Film 29 : Severe ACP occurred on D9 of treatment in this female patient who was mechanically ventilated because of ARDS. TEE revealed (i) right ventricular dilatation, (ii) paradoxical septal motion with right ventricular dilatation more marked at each insufflation, (iii) severe restriction of the LV as indicated by its “glove finger” appearance

on the long axis, flattening on insufflation, and an inverted mitral E/A ratio, (iv) biphasic pulmonary artery flow, indicative of marked obstruction of ejection. This is ascribed to progressive deterioration in ventilatory mechanics caused by incipient pulmonary fibrosis. The patient was then given corticosteroids.

Film 30 : Chez la même patiente que sur le film 29, la mise sous corticoïdes est responsable en quelques jours d'une amélioration très nette de la mécanique respiratoire. A J14, l'ETO objective la disparition du CPA, et un flux artériel pulmonaire normal.

If ACP occurs during ARDS, the following measures should be implemented immediately :

- » reduce plateau pressure to below 25 cm H 2O
- » lower PEEP to below 8 cm H 2O
- » reduce PaCO 2 to below 60-65 mmHg by use of a heater/humidifier in place of the filter (20), possibly by increasing respiratory frequency in certain patients. However, this maneuver is rarely effective and by generating an intrinsic PEEP often raises the plateau pressure, at the expense of right ventricular ejection (21). Remember that hypercapnia, which leads to systemic vasodilatation, has the reverse effect on the pulmonary circulation, resulting in arteriolar vasoconstriction (22).
- » prone positioning if the ratio PaO 2/FIO 2 remains below 100 mmHg
- » use TEE to check the absence of proximal PE

If ACP is accompanied by insufficiency circulatory, the most suitable vasoactive drug is norepinephrine, which restores systemic blood pressure and so improves right coronary flow and right ventricular systolic function.

Film 25 : In the same patient as in film 24, mechanical ventilation is responsible for the onset of ACP, revealed by TEE.

Film 31 : In the patient illustrated in film 25 who developed circulatory insufficiency and ACP while mechanically ventilated, norepinephrine infusion restored systemic blood pressure and resolved ACP. Note, however, a very clear decrease in left ventricular contractility.

When ACP appears after more than one week of assisted ventilation in a patient whose lung compliance is deteriorating, and in whom hypercapnia is increasing, this combination is strongly suggestive of a fibroproliferative phase. We then always use corticosteroid therapy.

Lastly, inhaled NO can also afford rapid relief and reduce or eliminate signs of ACP.
Film 32 : Female patient on controlled mechanical ventilation for ARDS caused by extensive bacterial lung infection. TEE indicated severe ACP which was, in part, responsible for circulatory insufficiency.

Film 33 : In the same female patient as in film 32, inhalation of 4 ppm NO normalized right ventricular function within a few hours.

In our experience, immediate implementation of these measures, which presupposes rapid echocardiographic diagnosis, has meant that ACP no longer results in excess mortality in ARDS. ACP can greatly reduce the likelihood of cure if specific and timely measures are not taken (19).