To Give or Not to Give? Rethinking Fluid Resuscitation for Massive Pulmonary Embolisms in the Prehospital Setting: A Review of the Literature

E. Kalkidis, J. Martin, J. Veronese
Griffith University, School of Medicine and Dentistry

Introduction

Pulmonary embolism (PE) is a potentially life-threatening condition caused by obstruction of the pulmonary arteries (Figure 1) (1), with an overall mortality of 14.7% even in those receiving treatment (2). Clinical presentation varies widely depending on clot burden and location, from mild symptoms to severe respiratory and hemodynamic compromise (3). Massive PE (Figure 2) (MPE), defined by sustained hypotension with a systolic blood pressure (SBP < 90 mmHg) (4, 5), carries particularly high mortality, with thirty-day mortality at 57.1%. In contrast, sub-massive PE has a mortality of around 6.7%, and low-risk cases about 3.5% (6). In severe cases, MPE can precipitate cardiac arrest within 1–2 hours of onset (3).

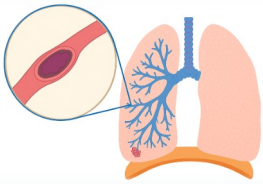

Figure 1: PE in pulmonary artery

In MPE, hypotension results from obstructive shock and right ventricular (RV) failure rather than fluid loss (7). Fluid resuscitation remains an option in MPE management; however, the evidence for its efficacy is unclear. In fact, excessive fluid administration may worsen RV strain and further compromise hemodynamics, with some evidence suggesting that volume removal may be an effective treatment option in certain cases (7). This review examines current evidence on fluid resuscitation strategies for adult patients with MPE in the prehospital setting, with the aim of informing safer and more effective care.

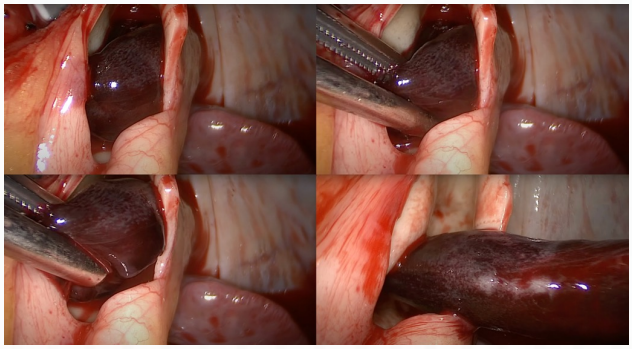

Figure 2: Intraoperative view of a surgical pulmonary embolectomy, showing extraction of a thrombus from the pulmonary artery.

Methods

A literature search was conducted in July 2024 using the electronic databases Ovid MEDLINE and EMBASE, covering publications from 1950 to July 2024. Search terms included combinations of keywords and MeSH headings: massive pulmonary embolism, submassive pulmonary embolism, prehospital care, fluid therapy, volume expansion, intravenous fluids, resuscitation, and paramedics.

Peer-reviewed articles were included if they examined fluid resuscitation (FR) using any fluid type in the management of pulmonary embolism in human or animal models, in either prehospital or in-hospital settings. Animal studies were considered due to the limited availability of prehospital human data. Articles were excluded if they were non–peer-reviewed, involved conditions unrelated to pulmonary embolism, were conducted in non-clinical settings, or were systematic reviews or meta-analyses.

Results

The database search (Figure 3) identified 542 articles, which were reduced to 41 following title and abstract screening. Of these, 32 were excluded after full-text review based on the predefined criteria, leaving nine peer-reviewed articles for inclusion (Figure 1).

Analysis of the included studies indicated that fluid administration for acute MPE was associated with RVD and reduced cardiac output, increasing the risk of haemodynamic instability and cardiogenic shock (8-12). In contrast, alternative interventions – such as positive inotropes and diuretic therapy (13-16) – were linked to improved RV function and stroke volume, with a lower risk of exacerbating myocardial dysfunction and a greater likelihood of promoting haemodynamic stability.

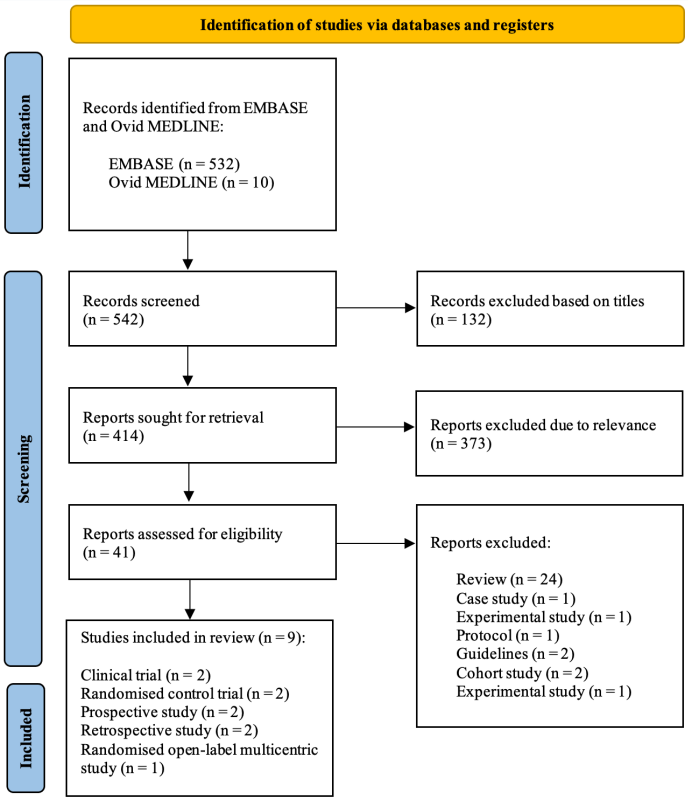

Figure 3: PRISMA Flow Diagram of retrieved included and excluded studies.

Discussion

This review examined FR in the pre-hospital management of MPE. Evidence shows FR often worsens RVD and haemodynamic instability. Although intended to restore preload, its benefits appear to be outweighed by adverse effects. Alternative strategies – such as positive inotropes and diuretics – appear safer for MPE patients.

FR in PE reduces left ventricular stroke work and area index, increases RV afterload, and can precipitate RV failure, compromising stroke volume (SV) and cardiac output (CO) (8, 9). Studies caution that FR raises myocardial oxygen demand, causes interventricular septal shift, reduces LV filling, and further decreases CO (17-19).

Positive inotropes improve RV contractility, enhance SV, and counteract high afterload (9, 14). Diuretic therapy (DT) offers faster normalisation of B-type natriuretic peptide (BNP) compared with VE (12, 13, 16) and improves SBP and creatinine without worsening RV echocardiographic findings. By reducing RV preload, DT lowers RVD risk and supports haemodynamics via increased intrinsic RV contractility (15).

Overall, routine fluid administration in hypotensive MPE patients risks worsening RV function and CO. DT and vasopressors such as noradrenaline appeared to better maintain haemodynamic stability, prevent fluid overload, and support RV performance in this high-risk group.

References

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