Gynaecological transurethral resection of the prostate syndrome–induced acute pulmonary oedema treated with high-dose nitro-glycerine: a case report

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Case report

A previously healthy 48-year-old woman developed acute respiratory distress during operative hysteroscopy for submucosal myomectomy. The preoperative chest X-ray was normal (Fig a). During surgery, facial oedema and acute dyspnoea developed following intrauterine irrigation with 19 100 mL normal saline. Analysis of arterial blood showed mixed metabolic acidosis and respiratory acidosis with pH 6.989, PaCO2 57.8 mm Hg, and a bicarbonate concentration of 14.0 mmol/L (Table). Desaturation developed progressively and her SpO_2 was 85% with O_2 mask at a flow rate of 10 L/min. The PaO_2/FiO_2 ratio was 121.5 mm Hg.

On arrival in the intensive care unit, the patient had a blood pressure of 160/90 mm Hg, heart rate 72 bpm, respiratory rate 30 breaths per minute, and SpO_2 84%. Chest auscultation revealed bilateral crackles and point-of-care ultrasonography showed a bilateral diffuse B-line pattern consistent with acute pulmonary oedema. Cardiac ultrasound revealed preserved left ventricular systolic function (75.01%) [Fig b]. There was neither regional wall motion abnormality nor aortic stenosis. Non-invasive positive pressure ventilation with bi-level positive airway pressure was commenced to assist ventilation. Subsequent chest X-ray showed acute pulmonary oedema (Fig c) and intravenous diuretic (furosemide, 40 mg) was administered; nevertheless her PaO_2/FiO_2 ratio dropped to 80.5 mm Hg (Table). The overall presentation was compatible with gynaecological transurethral resection of the prostate (TURP) syndrome–induced acute pulmonary oedema.

Intravenous high-dose 1 mg nitro-glycerine (NTG) was administered followed by 1 mg NTG bolus 5 minutes later and continuous infusion at a rate of 1 mg/h. Transient hypotension was noted after the high-dose NTG bolus. The patient's respiratory distress and oxygenation improved gradually and arterial blood gas analysis revealed improvement with pH 7.304, $PaCO_2$ 46.9 mm Hg, PaO_2 64.4 mm Hg, and lactate 38 mg/dL (Table). The next day, chest X-ray revealed resolution of bilateral pulmonary congestion (Fig d). The patient was maintained in a negative fluid balance and fitted with a nasal cannula that was well tolerated. She was transferred to the general ward the next day and discharged from hospital on the third day after surgery.

TABLE. Time course of arterial blood gases for a 48-year-old woman who developed acute respiratory distress during operative hysteroscopy for submucosal myomectomy. At 1 hour after diuretic use, the arterial blood gas showed deterioration of the PaO_2/FiO_2 ratio. Following a high-dose nitro-glycerine bolus and continuous nitro-glycerine infusion with diuretic and bi-level positive airway pressure support, PaO_2/FiO_2 ratio increased

Variables	Intra-operative	1 hour later	13 hours later
pH	6.989	7.304	7.468
$PaCO_2$ (mm Hg)	57.8	46.9	38.4
HCO_3- (mmol/L)	14.0	23.5	28.1
Base excess (mmol/L)	-17.1	-2.5	4.8
PaO_2 (mm Hg)	115.4	64.4	143.9
PaO_2/FiO_2 (mm Hg)	121.5	80.5	287.8
Haemoglobin (g/dL)	13.5	11.2	8.4
Sodium (mmol/L)	146.5	146.2	138.4
Potassium (mmol/L)	3.37	3.93	3.11
Lactate (mg/dL)	65.8	38.0	17.8

Discussion

Hysteroscopic myomectomy is a surgical treatment for submucosal myoma. During hysteroscopy, continuous fluid irrigation is required to distend the uterine cavity for optimal visualisation of the operative field. However, when intrauterine pressure exceeds mean arterial pressure, rapid intravasation

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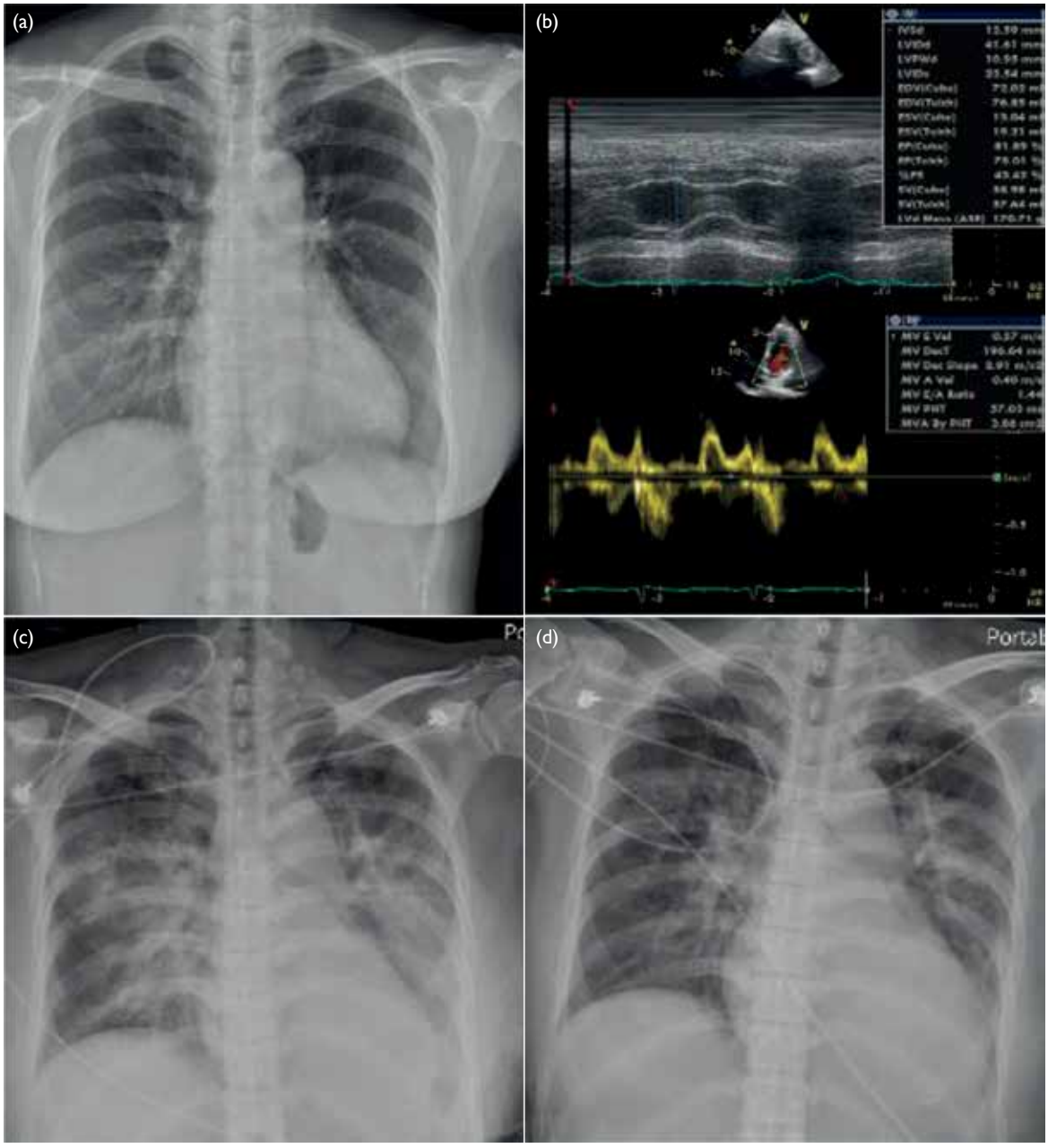

FIG. A 48-year-old woman who developed acute respiratory distress during operative hysteroscopy for submucosal myomectomy. (a) Normal preoperative chest radiograph; (b) cardiac ultrasound showing preserved left ventricular systolic function and trivial mitral regurgitation with no regional wall motion abnormality; (c) chest radiograph after hysteroscopy showing bilateral perihilar haziness, consistent with pulmonary oedema; (d) chest radiograph showing resolution of bilateral pulmonary oedema the day after surgery

via the venous sinuses may occur and absorption of excessive irrigation fluid may cause hypervolaemia and noncardiogenic pulmonary oedema. This phenomenon is termed operative hysteroscopy intravascular absorption syndrome with pathogenesis similar to that of TURP syndrome; as such, operative hysteroscopy intravascular absorption syndrome is also known as gynaecological TURP syndrome.[1]

Irrigation with electrolyte-free fluid in monopolar operative hysteroscopy may result in dilutional hyponatraemia and hypo-osmolality. Treatment of severe hyponatraemia-related neurological symptoms such as convulsion or cerebral oedema requires an infusion of 3% hypertonic saline solution. Use of a glycine-based solution can result in hyperammonaemia since

ammonia is a by-product of glycine once absorbed and metabolised in the liver. In bipolar operative hysteroscopy, 0.9% normal saline is the commonly used irrigant, so there is no significant procedure-related hyponatraemia. Nonetheless in our patient, severe metabolic acidosis developed due to rapid absorption of chloride causing smaller plasma strong ion difference, leading to an increase in hydrogen ion, and therefore acidosis. There are no definitive diagnostic criteria for gynaecological TURP syndrome; the signs are varied and non-specific. Clinical symptoms following a hysteroscopic procedure in patients should be evaluated carefully to exclude or diagnose gynaecological TURP syndrome.

An intravenous NTG infusion is one of the treatments for acute pulmonary oedema. Nitro-glycerine produces nitric oxide that increases the formation of cyclic guanosine 3',-5'-monophosphate.[2] The increased intracellular cyclic guanosine 3',-5'-monophosphate inhibits the influx of calcium into cells thereby decreasing intracellular calcium levels and causing vascular smooth muscle relaxation. This vasodilator effect on veins causes a decreased venous return, helps reduce cardiac preload and reduces pulmonary capillary hydrostatic pressure. The vasodilator effect on arteries in turn reduces systemic vascular resistance, helps reduce afterload and increases cardiac output. The main effect of NTG is on fluid redistribution of blood volume away from the lungs rather than on fluid removal per se.

To treat acute heart failure, the initial dose of NTG is 10 to 20 μg/min titrated to a total dose up to 200 μg/min until the desired haemodynamic effect is obtained.[3] The main side-effects of NTG are hypotension and headache. The major concern of NTG therapy is resistance and tolerance under continuous infusion. In a previous study, intravenous bolus of high-dose NTG (2 mg) with repeated administration every 3 minutes, up to a total of 10 doses in treatment of severe decompensated heart failure, was associated with lower frequency of endotracheal intubation and intensive care unit admissions as well as few adverse events.[4] The contra-indications to high-dose NTG bolus are severe aortic stenosis, hypertrophic obstructive cardiomyopathy, hypotension, and concurrent use of phosphodiesterase type 5 inhibitors.

Other strategies to treat acute pulmonary oedema include supplemental oxygen and/or ventilatory support, morphine, diuretics (eg, loop diuretic or thiazide), fluid restriction, inotropic agents in cases of co-morbid hypotension or hypoperfusion, and treatment of any underlying aetiologies.[5] Non-invasive positive pressure ventilation improves pulmonary compliance and reduces atelectasis. This can reduce the work of breathing and decrease the need for endotracheal intubation.

The effect of NTG is rapid with an immediate peak effect and short half-life of 3 to 5 minutes. It offers greater efficacy, safety, and a faster onset of action than diuretics or morphine administered to cause rapid improvement of pulmonary congestion. In our case, the patient did not experience significant improvement after diuretics so an intravenous bolus of high-dose NTG was administered along with bi-level positive airway pressure support. As a result, while awaiting the fluid removal effect of diuretic therapy, high-dose NTG helped improve the respiratory distress and avoid an immediate need for intubation.

Our case findings suggest that high-dose NTG can be used to treat gynaecological TURP syndrome–induced acute pulmonary oedema and obviate the need for endotracheal intubation. As far as we know, this is the first case report of the application of high-dose NTG to treat gynaecological TURP syndrome. The success in this case may offer new insight for recommended treatment and further research.

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