Confirmation of suspected anaphylaxis by measurement of serum tryptase

Sir,

A 56-year-old male weighing 78.5 kg was scheduled for percutaneous nephrolithotomy for left renal calculus under general anaesthesia. He was a known diabetic, well-controlled on oral hypoglycaemic agents and with no history of allergies. As effort tolerance was poor on history, preoperative 2-D echocardiography was performed that showed normal cardiac function. His anaesthetic induction was as per standard institutional protocols that included intravenous midazolam 2 mg, fentanyl 160 µg, propofol 160 mg and intubation after 8 mg cisatracurium. Following positioning in lithotomy, a retrograde pyelogram was performed using meglumine iodide 76% (Urografin) at which time his heart rate was 68/min, blood pressure at 140/70 mm Hg and a saturation (SpO₂) of 100% on 50% oxygen. He had undergone a previous intravenous pyelogram using iodixanol (Visipaque) uneventfully. A few minutes after the instillation of the dye, a fall in saturation to 40% with hypotension (50/30 mm Hg) was observed which was managed with 100% oxygen, intermittent boluses of ephedrine, phenylephrine and fluid boluses. An improvement in blood pressure (BP) was seen only after the administration of incremental doses of adrenaline, and a total of 50 µg was administered. On suspicion of anaphylaxis to the contrast, 100 mg hydrocortisone, 50 mg injection ranitidine and one vial containing 2 mL of 45 mg of pheniramine maleate were administered intravenously. Arterial and central venous lines were secured. An intravenous infusion of noradrenaline (0.05 µg/kg) was started to maintain blood pressure. Intraoperative 12 lead electrocardiogram and echocardiogram were performed to rule out myocardial event and were normal. The planned procedure was deferred, and the patient was shifted to the intensive care unit with a heart rate of 84/min, BP of 124/72 mm Hg and SpO₂ of 100% on 40% oxygen. He was ventilated for 2 h and extubated shortly afterward.

Tryptase levels sent immediately following the incident and at 24 h were 66 µg/L and 14.3 µg/L, respectively, which were consistent with the diagnosis of an anaphylactic reaction, most probably to the contrast used for pyelogram. As this test was unavailable at our centre, we needed to send it to another laboratory involving a processing duration of about 10 days. We believe that this testing confirmed our suspicion of anaphylaxis.

Recommendations suggest measurement of tryptase level at 1 h after reaction, another at 2 to 4 h with a third at 24 h post reaction that serves as a control. The serum tryptase is <12.5 µg/L normally, and an increase of (1.2 × baseline) +2 µg/L is considered clinically relevant.

The incidence of reported anaphylaxis varies from 1 in 363 to 1 in 18600. Common causes of anaphylaxis are antibiotics and neuromuscular blocking drugs, sugammadex, latex, dyes and chlorhexidine. The key to successful management of anaphylaxis is a timely diagnosis, appropriate dosing of epinephrine and adequate intravascular volume replacement. There are reports of severe and sometimes life-threatening anaphylactic reactions to non-vascular administration of iodinated contrast media. It occurs due to the absorption of contrast at the mucous membrane. The European Society of Urogenital Radiology suggests the need for precaution even with non-intravenous contrast administration. Planning for future surgery in such patients should include skin prick or intradermal testing of all drugs to be used during surgery. Tests to quantify specific IgE antibodies to select drugs in the perioperative period like chlorhexidine and latex are available. Drug provocation tests are tests that can be used to identify the culprit drug in non-IgE–mediated allergic reactions. In case of emergency surgery, consider regional anaesthesia, minimise the use of drugs for induction, use inhalational agents, avoid muscle relaxants, latex, disinfectants, penicillin and cephalosporins along with a high index of suspicion for early treatment to avoid adverse outcomes.

It is concluded that serum tryptase levels should be estimated more often in patients with suspicion of anaphylaxis to confirm the diagnosis following which we need to do specific tests to identify the possible causative drug and design a safe future anaesthetic plan.

**Declaration of patient consent**

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The
Adrenal insufficiency as a post-COVID-19 sequela

Sir,

Novel coronavirus outbreak was declared as a ‘pandemic’ by the World Health Organization on 11 March 2020.[1] Patients with severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) may have had multiorgan involvement including involvement of adrenal glands.[2] Structural adrenal gland changes with histological features showing focal necrosis have been reported. Hypothalamic–pituitary–adrenal (HPA) axis has shown the presence of SARS-CoV-2 ribonucleic acid suggesting hypophysitis.[3]

We report a case of adrenal insufficiency (AI) in a 62-year-old man who presented with shock. This patient had persistent vomiting and imbalance while walking since the previous 2 days. He was a known hypertensive, diabetic and hypothyroid taking regular treatment. He was hospitalised 2 months back with coronavirus disease (COVID)-19 pneumonitis and had received oral steroids, remdesivir and supplementary oxygen.

Clinical examination revealed high-grade fever with a heart rate of 136/min, blood pressure of 86/60 mm Hg and mild tachypnoea with oxygen saturation of 90% on room air. He was restless, irritable and confused with cold clammy dry skin. Blood investigations revealed a high white blood cell count (16,000/ mm$^3$), raised renal parameters (Blood urea nitrogen 86 mg/dL, serum creatinine 2 mg/dL) and random blood sugar 386 mg/dL. Serum electrolytes, blood gases, electrocardiogram, two-dimensional echocardiogram and computerised tomography chest were normal. Inferior vena cava was seen collapsed on echocardiography. Our provisional diagnosis was post-COVID-19 septic shock with prerenal azotemia.

He was treated aggressively with intravenous (IV) antibiotics: meropenem, aztreonam, IV infusion of crystalloids (normal saline) and IV insulin. He needed noradrenaline IV infusion (0.2-1 µg/kg/min) to maintain haemodynamic stability. Despite being treated

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