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Editorial

Hypercapnia: A Serious Indicator of Clinical Deterioration

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Advances in intensive respiratory care, non-invasive ventilation (NIV), and chronic respiratory disease management have significantly improved outcomes over recent decades. However, a delayed clinical diagnosis of carbon dioxide (CO_2) retention continues to contribute to avoidable morbidity, mortality, prolonged hospitalisation, and intensive care admissions. Hypercapnic respiratory failure continues to remain one of the most overlooked, yet potentially reversible causes of clinical deterioration in hospitalised patients. In most clinical settings, it is the ward where the initial clinical deterioration begins, much before it has snowballed into a full-fledged medical emergency.

Even for the experienced clinician, it is sometimes quite a challenge diagnosing hypercapnia clinically and diagnosing those at potential risk before physiological compensation fails.

Patients with obstructive sleep apnoea syndrome (OSAS), chronic obstructive pulmonary disease (COPD), chest wall diseases, neuromuscular disorders such as Guillain-Barre Syndrome, and advanced sleep-disordered breathing may get admitted to medical and surgical wards for illnesses other than of respiratory origin. In such cases, excessive oxygen therapy, fluid overload, presence of infection, postoperative analgesia, medications such as sedatives and fatigue of respiratory muscles may transform a chronic case of compensated hypercapnia into an acute-on-chronic respiratory failure over a short period of a few hours.

In such cases, clinical detection of the deteriorating condition occurs late, usually after severe respiratory acidosis has set in, with consequent respiratory depression, fatigue and failure.

This lack of early clinical detection of physiological deterioration is a main underlying cause of morbidity and mortality in these patients.

Hypercapnia and its consequences

Raised $PaCO_2$ is indicative of inadequate alveolar ventilation relative to the production of metabolic CO_2. Even though a rising arterial carbon dioxide ($PaCO_2$) level is the determining physiological abnormality, the consequences extend far beyond pathological parameters.

Rising $PaCO_2$ levels cause cerebral vasodilatation which results in increased cerebral blood flow. This results in reduced mentation and depressed consciousness, with consequent reduced respiratory muscle performance, impaired airway protection, and eventual respiratory acidosis accompanied by cardiovascular disfunction.

Clinically, symptoms of hypercapnia evolve and include headache, somnolence, confusion, drowsiness, a dusky look or frank cyanosis, tremors, metabolic flaps, conjunctival oedema, and a bounding pulse, with progressively worsening respiratory failure.

Unfortunately, most clinicians are mainly concerned with monitoring oxygen saturation, failing to realise that while this is an important blood gas determinant, oxygen saturation provides no information regarding the state of ventilation.

A patient receiving high-flow oxygen therapy may demonstrate oxygen saturations of 96 - 100% while simultaneously developing life-threatening hypercapnia. This disconnect is particularly relevant in patients with COPD and OHS, who, having chronic CO_2 retention have a depressed hypercapnic drive and in such instances excessive oxygen administration worsens ventilation-perfusion mismatch and further depresses the hypoxic respiratory drive in these patients, resulting in further CO_2 retention.

Hence, a discerning clinician should read the arterial blood gas (ABG) report in its entirety, laying equal stress on all parameters while also corelating the ABG findings clinically.

The Harbingers of deterioration

Most signs of hypercapnic respiratory failure usually begin in the general medical wards rather than in high-dependency units (HDU) or the Intensive Care Unit (ICU).

Many past clinical studies have shown that changes in vital parameters such as the heart rate, respiratory rate, and a deteriorating level of mentation often precede serious adverse events by many hours. Yet respiratory rate remains one of the least accurately measured vital signs in routine practice.

Unfortunately, at times, vital parameters are clinically overlooked due to automated monitoring of the patient's condition. Therefore, slight increases in the heart rate and the respiratory rate are commonly attributed to other conditions such as pain and anxiety, thereby missing the diagnosis of impending ventilatory failure.

The underlying physiology

As alveolar ventilation reduces, respiratory rate begins to increase before the tidal volume falls. Accessory muscles become active and in due course the patient becomes progressively fatigued and increasingly drowsy and dusky looking.

Arterial blood gas analysis show a rising titre of $PaCO_2$ with compensatory bicarbonate (HCO3) elevation in chronic disease. However, in acute respiratory failure, a rise in the $PaCO_2$ levels is not accompanied by a compensatory rise in the HCO_3 levels.

If the clinical symptoms and signs are not corelated with the ABG findings, by the time altered consciousness develops, significant hypercapnia is already established.

Early warning scores, including the National Early Warning Score (NEWS2) help in early detection of deteriorating patients. However, these scores are primarily driven by abnormalities in respiratory rate, blood pressure, oxygenation, and consciousness. They are not direct measures of ventilation. Hence, patients with compensated hypercapnia may initially generate relatively modest scores despite progressive physiological decline towards acute-on-chronic respiratory failure. Clinical co-relation is therefore of utmost importance in detecting early respiratory fatigue and failure.

Which patients are at high-risk of developing hypercapnic respiratory failure?

In order to determine which patients are at high-risk of developing hypercapnic respiratory failure, it is essential that risk stratification be done at the time of admission.

Known cases of COPD are highest at risk, particularly those with a previous history of acute hypercapnic respiratory failure needing perpetual NIV.

Previous hypercapnic episodes are a good indicator of future episodes. Another important subset of patients at risk include patients with obesity hypoventilation syndrome (OHS), which has now emerged as an increasingly prevalent cause of patient decline in the medical wards.

With a rise in obesity the world over, many patients getting admitted under various medical specialities have, as yet, undiagnosed chronic alveolar hypoventilation.

Bedside clinical findings indicative of chronic hypercapnic respiratory failure include:

- Body mass index >35 kg/m^2.
- Loud snoring with apnoeic spells.
- Daytime sleepiness.
- Elevated serum bicarbonate (> 27 mmol/L).
- Polycythaemia.
- Chronic hypoxaemia requiring home oxygen therapy.
- Active NIV (BiPAP or CPAP) use at home.

Restrictive impairment due to neuromuscular and chest wall deformities can also further aggravate CO_2 retention. This is seen in patients with muscular dystrophies, cervical spinal cord injuries, motor neuron disease, neuromuscular disorders and severe kyphoscoliotic deformities. If patients afflicted by these disorders develop a superadded bacterial infection or are administered strong sedatives such as opioids, they may develop acute CO_2 retention.

Post-operatively, hypercapnia can occur following major surgery, primarily due to impaired cough reflex, atelectasis, residual anaesthesia and restriction of thoracic wall movements resulting in hypoventilation. This is especially seen in obese patients and those with obstructive sleep apnoea syndrome (OSAS).

Mainstay in treatment: Oxygen

Oxygen is the drug which is a mainstay in the treatment of this condition. Like all pharmacological medications, oxygen should be used as judiciously as any other drug.

Many clinical trials have shown that uncontrolled high-flow oxygen can increase morbidity and mortality in acute exacerbations of chronic obstructive pulmonary disease, especially chronic bronchitis, by worsening the hypercapnia, as compared to controlled oxygen therapy. This is because, in chronic bronchitis, the hypercapnic drive in the respiratory centre is blunted due to chronically high $PaCO_2$ levels. The only drive available to these patients is the hypoxic drive which too can get depressed due to uncontrolled high-flow oxygen therapy.

Hence, current guidelines have recommended target oxygen saturations of 88 - 92% for patients at risk of developing hypercapnic respiratory failure [1,2].

Hence, due care must be taken while prescribing oxygen therapy especially in chronic bronchitis and other conditions with chronic CO_2 retention.

A vital bedside investigation: Arterial blood gas (ABG) analysis

Arterial blood gas (ABG) analysis is the diagnostic test for detecting hypercapnia. When in doubt, never hesitate to perform ABG analysis as it is a pivotal investigation in reducing morbidity and mortality in patients with hypercapnic respiratory failure. This is especially true when patients exhibit cyanosis, drowsiness, altered mentation and tachypnoea along with clinical signs of conjunctival oedema, asterixis and a bounding pulse.

Venous blood gas (VBG) analysis too plays a useful role in this condition. There appears to be a good correlation between venous and arterial blood gas findings. Hence, venous blood gas examination by the bedside helps in detecting early CO_2 retention which can then be confirmed by the more invasive ABG procedure. VBG determines oxygenation at the tissue level more accurately. Hence, it is a very useful investigation in conditions with low blood pressure, such as cardiogenic shock.

Serial blood gas analysis over hours and days is normally needed in order to confirm acute and/or chronic CO_2 retention and thereby distinguish chronic bronchitis from emphysema. A diagnosis should never be made on a single ABG report as it may be fraught with errors.

Importance of capnography in early diagnosis of hypercapnia

Continuous capnography has significantly helped in monitoring patients in the operating theatre during surgery and also in the intensive care unit. However, it has remained underutilised on the hospital ward floors.

End-tidal CO_2 monitoring gives a constant assessment of ventilation and identifies early hypoventilation before oxygen saturation declines, especially among postoperative patients receiving opioids. Transcutaneous CO_2 monitoring provides constant estimates of arterial CO_2 levels.

Studies should be conducted to determine if routine ward capnography improves clinical outcomes and reduces morbidity and mortality in patients with CO_2 retention. However, capnography is recommended in high-risk patient populations such as postoperative patients, patients on strong analgesics, severely obese patients, and those requiring long-term NIV.

Future studies should evaluate whether continuous CO_2 monitoring can reduce emergency ICU admissions and cardiac arrests.

Non-invasive ventilation

The effectiveness of Non-Invasive Ventilation (NIV) depends upon timely intervention.

Timely use of NIV significantly reduces intubation rates, morbidity, length of hospital stay and mortality. If NIV is delayed, its efficacy significantly reduces as respiratory muscle fatigue and failure worsens.

Hence, NIV should be instituted before severe respiratory acidosis sets in.

Consequently, hospitals lacking proper NIV pathways experience substantial delays between clinical and blood gas confirmation and treatment initiation.

Modern technology versus clinical assessment

Though technologies such as artificial intelligence, wearable sensors, and continuous physiological monitoring offer exciting opportunities, technology cannot replace clinical assessment at the bedside.

Discerning physicians frequently identify subtle early clinical signs such as somnolence, increasing drowsiness, patient struggling to complete sentences, conjunctival oedema and a dusky look which often represent early CO_2 narcosis, and which technology cannot assess accurately.

Future expectations

As hospital populations become increasingly older, more severely obese with multiple co-morbidities, the incidence of hypercapnic respiratory failure will continue to increase. Simultaneously, this demands early recognition so that intervention can be instituted before organ failure becomes established.

This entails wider implementation of oxygen stewardship programmes, earlier blood gas analysis and evaluation of technologies for continuous CO_2 monitoring.

Hypercapnia is rarely a sudden phenomenon. Most patients exhibit early warning signs for hours or sometimes days before drastic deterioration in their clinical condition occurs. The challenge for the clinician and support staff is ensuring those clinical signs are detected early, interpreted correctly, and acted upon without delay.

Hence, recognising hypercapnia at the bedside before patients deteriorate clinically remains the cornerstone in reducing morbidity and mortality in these patients [3-8].

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