Usefulness of point-of-care ultrasound in venous thromboembolism related to COVID-19

Utilidad de la ecografía clínica a pie de cama en la enfermedad tromboembólica de la Covid-19

To the Editor,

Point-of-care ultrasound is gaining increasing importance in emergency departments due to its availability, accessibility, versatility, and lack of ionizing radiation exposure.[1] We present the case of a 54-year-old woman who came to the emergency department with a mild SARS-CoV-2 infection, confirmed by a positive polymerase chain reaction (PCR) test at low cycle thresholds, consistent with a high viral load. Three weeks later, the patient returned complaining of sudden-onset dyspnea, cough, and pleuritic pain. She was allergic to iodinated contrast media and had no other relevant medical history.

On examination, she exhibited tachycardia, tachypnea, and a baseline oxygen saturation of 94%, with pulmonary auscultation revealing only crackles in the right lower lung base. There were no signs suggestive of deep vein thrombosis (DVT). With a clinical suspicion of pulmonary embolism (PE), a chest X-ray was performed, showing interstitial infiltrates compatible with COVID-19 pneumonia, and blood tests revealed leukocytosis of 18,000/mm^3, C-reactive protein of 256 mg/L, and D-dimer of 10,820 µg/L. The SARS-CoV-2 PCR remained positive but at high cycle thresholds, indicating low viral load. Because of the patient's iodine allergy, CTPA (CT pulmonary angiography) was not performed. Instead, a point-of-care ultrasound was performed, revealing thrombosis of the right femoral vein (Figure 1). Focused cardiac ultrasound showed no evidence of right ventricular overload. The patient was admitted with a diagnosis of COVID-19-realted venous thromboembolic disease (VTE), and anticoagulant therapy was initiated, resulting in a favorable clinical course.

VTE is a relatively frequent condition in emergency departments, and its incidence rate appears to be increased in patients with COVID-19.[2] Diagnosis is based on clinical suspicion, supported by predictive scores and laboratory parameters, and is confirmed by imaging studies. CTPA remains the gold standard for the diagnosis of PE, while Doppler ultrasound is the technique of choice for diagnosing DVT.[3] However, predictive scales for DVT diagnosis, such as the Wells criteria, do not adequately predict disease in COVID-19 patients.[4] D-dimer maintains good sensitivity for detecting VTE, and it is proposed to use cutoff values similar to those in the general population for COVID-19, since raising the threshold to improve specificity would reduce its negative predictive value.[5] Although the CTPA is the diagnostic imaging modality of choice for PE, there are several limitations to its use — such as contrast allergy, pregnancy,[6] or renal failure — in addition to the risks associated with IV contrast administration and ionizing radiation exposure, and the fact that it may not be available in some health care centers. Ventilation–perfu-

Figure 1. The image on the left shows a cross-section of the right femoral vein and artery with an enlarged femoral vein. The central image shows the absence of collapsibility of the vein upon compression. The image on the right shows the absence of blood flow in the femoral vein with partial collateral vascularization.

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sion scintigraphy can be an alternative, but COVID-19–related pneumonitis limits its usefulness in these patients.[2]

Ultrasound can play an important role in diagnosing PE when CTPA cannot be performed.[1] Multiple organ ultrasound, including assessment of the heart, lungs, and venous system, has a sensitivity of 90% and a specificity of 86.2%,[7] although most studies assessing its diagnostic value in VTE were conducted before the COVID-19 era. Moreover, ultrasound can also be helpful in evaluating differential diagnoses with similar clinical presentations.[1]

We believe that a positive Doppler ultrasound, even in the absence of DVT symptoms, in a patient with high clinical suspicion of PE, as in our case, offers good diagnostic performance. In such patients, the need for CTPA could potentially be avoided,[2] eliminating its associated risks while improving accessibility and diagnostic speed.

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Hypothermia and the J wave of Osborn

Hipotermia y onda de Osborn

To the Editor,

We present the case of a 67-year-old male patient with a past medical history of hypertension and chronic obstructive pulmonary disease (COPD), who was found unconscious at home with a blood pressure (BP) of 90/65 mmHg, heart rate of 113 bpm, and oxygen saturation (SpO_2) of 83%. Peripheral body temperature was undetectable. Endotracheal intubation was performed due to a low level of consciousness, and the patient was transferred to the hospital emergency department. Upon arrival, the patient continued to have undetectable temperature (measured via rectal thermometer), BP of 84/57 mmHg, heart rate of 126 bpm, and SpO_2 of 99%. Core temperature, measured with an esophageal probe, was 32°C.

The initial electrocardiogram (ECG) (Figure 1) revealed the presence of prominent convex deflections at the junction between the QRS complex

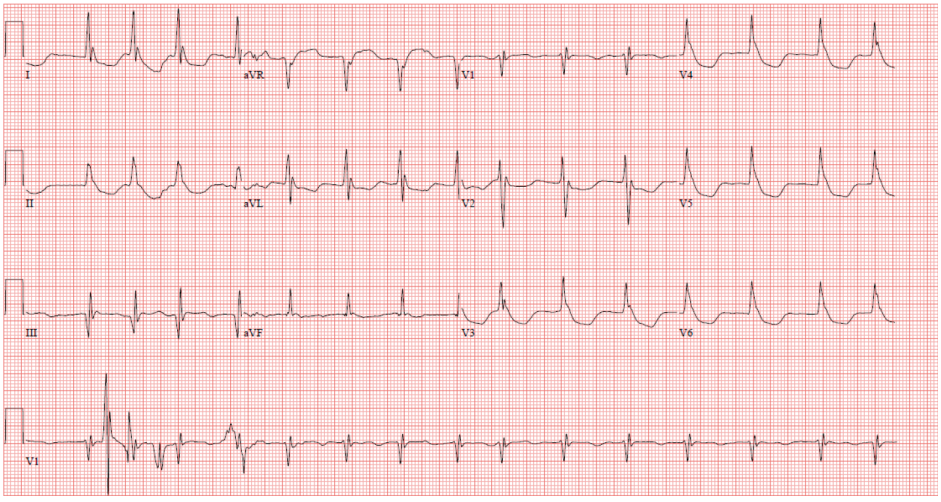

Figure 1. ECG upon admission: prominent convex deflections at the junction between the QRS complex and the beginning of the ST segment.

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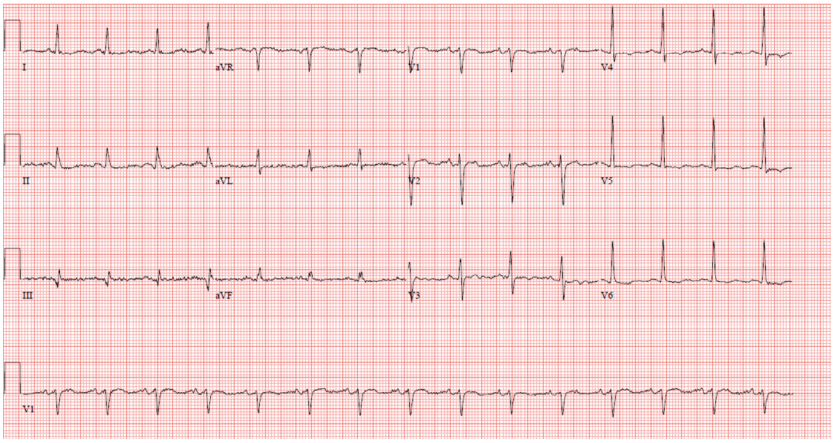

Figure 2. Follow-up ECG. Electrocardiographic normalization.

and the beginning of the ST segment. This alteration corresponds to the so-called J wave or Osborn wave, which typically appears in precordial leads as a positive deflection between the terminal portion of the QRS complex and the start of the ST segment.[1]

Vital support measures and empirical antibiotic therapy were initiated. After performing additional tests, which ruled out electrolyte disturbances and acid-base imbalance, the case was diagnosed as pneumonia-related septic shock associated with severe hypothermia due to cold exposure. Rewarming was initiated using thermal blankets and infusion of warmed fluids, leading to normalization of the ECG (Figure 2).

The Osborn J wave is associated with core body temperatures below 34°C, disappearing once normothermia is restored. It remains uncertain whether this ECG change results from ventricular depolarization abnormalities or early ventricular repolarization, but it shows high sensitivity and specificity for hypothermia. Moreover, several studies have correlated the degree of hypothermia with the presence of this wave, with the amplitude of the J wave being inversely proportional to the patient's core temperature.[3-5] However, the Osborn J wave is not pathognomonic of hypothermia and may also appear in other conditions such as hypercalcemia, Brugada syndrome, vasospastic angina, or certain cerebrovascular diseases.[2,3] This finding may be mistaken for acute ischemia, but it can be differentiated based on the clinical context, complementary test results, and ECG normalization after rewarming.

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On seeing revisiting as a red flag for risk of diagnostic failure

Sobre la revisita como red flag ante el riesgo del fracaso diagnóstico

To the Editor,

Within the field of health care management, the emergency department (ED) revisit rate — defined as patients who return to the ED within the first 72 hours after discharge — is considered a quality indicator used to assess the effectiveness of medical care.[1] However, if this indicator is not properly defined (e.g., revisit for the same reason for consultation, revisit for a different reason, revisit leading to hospital admission, or revisit requiring IV treatment, etc.) and exceeds the desired threshold, it risks providing misleading information about an emergency department's resolutive capacity. Nevertheless, in certain situations, it should instead be interpreted as a warning sign or red flag. As an example, we present the following case.

A 48-year-old man with a past medical history significant only for *Helicobacter pylori* infection presented to the hospital emergency department for diffuse abdominal pain. He was in a state of hemodynamic instability (tachycardic, hypotensive, febrile, and desaturated) with an impression of clinical severity. Furthermore, abdominal examination revealed colicky pain and tenderness in the upper abdomen, without palpable masses, organomegaly, or peritoneal irritation signs.

He had visited the emergency department 6 times over the previous 40 days

for the same complaint — specifically, 40 days prior, 34 days prior, 30 days prior, and twice within the past 24 hours prior to his final visit. Despite unremarkable blood test results, abdominal ultrasound had been performed on 3 occasions (at −34, −30, and −1 days prior to the final visit), without any pathological findings. Once stabilized through fluid resuscitation, electrolyte replacement, and management of sepsis with goal-directed therapy, including vasoactive agents, empirical antibiotic therapy, and hemostatic drugs, the patient was transferred to the radiology department for a contrast-enhanced thoraco-abdominopelvic computed tomography (CT) to determine the underlying cause.[2] CT findings revealed a colonic perforation due to an adenocarcinoma at the splenic flexure (T2N1M0), with associated fecaloid peritonitis.

The patient underwent emergency surgery, followed by favorable postoperative course, and was discharged 47 days after diagnosis and stabilization in the emergency department.

Based on this case, we believe that the revisit rate should not be used solely as a quality indicator for assessing health care efficiency, but considered a potential risk factor when making diagnostic, therapeutic, or disposition decisions.[3] Moreover, the fact that a patient reconsults does not necessarily imply that the prior management was inadequate.[4] Rather, it may indicate that the initial evaluation and treatment were appropriate at the time but later became insufficient due to the dynamic and evolving nature of the disease.[5]

Therefore, when properly defined, a revisit may serve as an important risk indicator for identifying missed diagnostic opportunities, allowing clinicians to adjust diagnostic and therapeutic measures during subsequent visits to achieve the most accurate diagnosis possible.[6,7]

As illustrated by this case, hospital emergency departments remain the final safety net for patients within the Spanish National Health System, particularly when other healthcare services fail to detect serious pathologies — or when patients simply do not seek care elsewhere.[8,9]

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Psychomotor agitation and hematuria in the context of an infectious process in a pediatric patient

Agitación psicomotriz y hematuria en contexto de proceso infeccioso en paciente pediátrico

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To the Editor,

Pediatric emergencies represent a significant challenge for emergency physicians, particularly those working in regional or community hospitals.[1]

We present the case of an 11-year-old boy brought to the emergency department due to disconnection from the environment, upward eye deviation, generalized rigidity, and subsequent hypotonia. Furthermore, he exhibited mild epistaxis and oral bleeding, both minimal in quantity. According to his parents, the previous day he had experienced malaise, asthenia, blurred vision, and hematuria. As background, the patient had been seen in the emergency department the previous week for fever, dyspnea, vomiting, and diarrhea, and was discharged with a diagnosis of acute gastroenteritis and respiratory infection. His 14-year-old sister had been evaluated the day before for suppurative pharyngotonsillitis. Other relevant history included obesity, bronchial asthma, and tonsillectomy.

On examination, the child was extremely agitated, unresponsive to external stimuli, and unable to fix his

gaze, with dry blood remnants in the mouth. His vital signs revealed afebrile status, elevated blood pressure (161/100 mmHg), and normal heart rate. Pupils were normal, and there was no neck stiffness. Rectal diazepam was administered without effect, followed by IV midazolam. The patient required oxygen via a reservoir mask to maintain SpO_2 > 90%. Bladder catheterization revealed overt hematuria.

Additional tests showed microcytic hypochromic anemia, leukocytosis with neutrophilia, normal platelet count, and normal coagulation studies. Glucose, renal function, acute-phase reactants, electrolytes, and proteins were all within normal limits. Chest X-ray showed a possible right basal infiltrate, and cranial computed tomography (CT) was normal. Since the hospital lacked a pediatric intensive care unit (PICU), the patient was transferred to a reference hospital, where he arrived with persistent hypertension, managed initially with labetalol (later replaced by nicardipine due to bronchospasm).

Further investigations revealed low complement C3 (with normal C4 and CH50) and elevated anti-streptolysin O (ASO) titer. Urinalysis showed hematuria, non-nephrotic proteinuria, and macroalbuminuria. Lumbar puncture turned out negative. Brain MRI revealed findings consistent with acute hypertensive encephalopathy vs posterior reversible encephalopathy syndrome (PRES). Electroencephalogram The EEG demonstrated mild generalized slowing of cerebral activity without persistent focal abnormalities.

The patient received antihypertensive therapy with enalapril and amlodipine, along with anticonvulsant prophylaxis using levetiracetam, with very favorable clinical progress. He was discharged with diagnoses of post-streptococcal acute glomerulonephritis (PSAGN), hypertensive emergency with central nervous system involvement, and PRES.

PSAGN is the most common cause of nephritic syndrome in childhood.[2,3] It presents a broad clinical spectrum, ranging from asymptomatic forms to severe cases such as encephalopathy or renal failure; however, when managed appropriately, it typically leaves no sequelae.[4,5] Although its incidence rate has dropped in recent years, poor hygienic conditions can increase the prevalence of nephritogenic streptococcal infections. The differential diagnosis should include cerebrovascular events, central nervous system infections, brain tumors, and toxic–metabolic disturbances.

We present this case to emphasize that PSAGN should be considered in any patient presenting with sudden neurological impairment associated with hematuria,[6,7] with or without hypertension, particularly in the context of a recent respiratory or skin infection.

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Asynchronous spontaneous rectus sheath hematomas

Hematoma espontáneo bilateral de la vaina de los rectos, asíncrono

To the Editor,

A spontaneous rectus sheath hematoma (RSH) occurs following the rupture of the epigastric artery or one of its branches. It has a low incidence rate, and its clinical presentation typically includes acute abdominal pain with a palpable mass effect. The main predisposing factor is anticoagulant therapy, in any of its forms.[1]

We report the case of a 76-year-old man with a past medical history of hypertension, diabetes mellitus, severe aortic stenosis, embolic stroke, chronic

Figure 1. Bilateral hematoma of the rectus sheath muscles of the abdomen.

obstructive pulmonary disease (COPD), chronic kidney disease (CKD), and permanent atrial fibrillation. His usual drugs included acenocoumarol, insulin, bisoprolol, and deflazacort. Twenty days prior to the current episode, the patient had experienced abdominal pain due to a right-sided RSH triggered by coughing fits, requiring a 7-day hospital admission. Upon discharge, anticoagulant therapy was discontinued, and he was prescribed subcutaneous enoxaparin (40 mg every 12 hours).

Eleven days later, he returned to the emergency department with a 48-hour history of intense pain in the left hemiabdomen, without trauma, along with a palpable mass and abdominal guarding. On examination, he appeared pale and generally unwell, with blood pressure of 114/52 mmHg and heart rate of 70 bpm. The abdominal examination revealed mild ecchymosis on the abdominal wall and a palpable mass in the mesogastrium and left flank with tenderness on palpation. Laboratory tests showed hemoglobin 8.3 g/dL, hematocrit 28%, glucose 156 mg/dL, urea 138 mg/dL, and creatinine 1.93 mg/dL, with normal coagulation parameters. Point-of-care ultrasound revealed a hypoechoic collection larger than 10 cm in the abdominal wall. Given the recent history of RSH, a computed tomography (CT) scan was performed, which confirmed a 10 cm × 10 cm × 20 cm hematoma within the left rectus abdominis muscle. A few hours later, due to clinical deterioration, the patient required transfusion of 2 units of packed red blood cells and embolization of the inferior epigastric artery.

RSH is generally unilateral and occurs more frequently in women, mainly due to anatomical differences in the rectus muscles between sexes and abdominal wall distension during pregnancy.[2] Other predisposing factors, in addition to anticoagulation, include corticosteroid use and chronic kidney disease,[2-4] all of which were present in this patient. The most common triggering factor is persistent coughing. In most cases, bleeding resolves spontaneously and remains contained within the muscular sheath, although in certain circumstances, urgent interventions such as volume replacement and embolization may be necessary.[5]

Bilateral RSH cases are relatively rare and have been associated with various clinical conditions, such as sepsis with secondary coagulopathy,[6,7] physical exertion,[8] and coughing episodes (as in our case).[9] Even rarer, however, is the sequential occurrence of hematomas, as seen in this patient. Finally, this condition should always be considered in the differential diagnosis of abdominal pain, particularly in elderly patients on anticoagulant therapy. Kidney failure can lead to anticoagulant overdosing, thereby increasing the risk of hematoma formation.

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