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ONE MINUTE CONSULT
BRIEF ANSWERS TO SPECIFIC CLINICAL QUESTIONS

Q: Should my elderly hospitalized patient with acute onset of altered mental status undergo stat head CT?

An 82-year-old woman presents to the hospital because of progressively worsening weakness. On hospital day 4, a nurse finds her with severe inattention, disorganized thinking, and an altered level of consciousness. The nurse initiates a rapid response. Computed tomography (CT) of the head without contrast is ordered, which reveals no acute intracranial process. Arterial blood gas measurement reveals respiratory acidosis. The patient is started on bilevel positive end-expiratory pressure ventilation. Repeat arterial blood gas measurement reveals that the acidosis has resolved, and the patient's mental status improves.

A: CT has become an integral tool in patient evaluation. Unfortunately, overreliance may have led to overuse.

In an article commemorating the 50th anniversary of the first CT scan,[1] Dr. Joel Howell reflected on the shift within medicine attributed to the new technology, specifically the ability to detect a lesion that may be contributing to disease. Howell asserted that the challenge of CT is "to determine when finding the lesion can help relieve symptoms and save lives and when it does little to improve the health of the patient."[1]

CT is used to look for intracranial hemorrhage if a patient is receiving anticoagulation. Additionally, it is commonly ordered to rule out a bleed or other intracranial process in hospitalized patients with delirium. However, a retrospective study found that of 220 CT scans performed for acute-onset delirium, only 6 (2.7%) had positive results.[2] This finding raises the question of whether CT is necessary in evaluating delirium, given its cost, radiation exposure, and allocation of a limited resource.

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DIAGNOSIS OF DELIRIUM

Delirium is an acute neurocognitive disorder characterized by sudden changes in attention and cognition. It has been reported to occur in 14% to 56% of hospitalized patients,[3] but it is recognized only 12% to 35% of the time.[4]

The *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, Text Revision* (DSM-5-TR) outlines 5 features that characterize delirium: (1) changes in attention and awareness that…(2) develop acutely from a patient's baseline and are… (3) associated with additional changes in cognition… (4) which are not better explained by another preexisting neurocognitive disorder and…(5) can be attributed to a medical condition elicited by history, physical examination, or laboratory data.[5]

There are many clinical methods for assessing delirium, such as asking the patient to count backward from 100 by 7s (serial 7s) or spell "world" backward, in addition to the following:

The Confusion Assessment Method[6] is a practical evidence-based tool to assess delirium at the bedside. Patients exhibiting acute or fluctuating inattentiveness accompanied by either an altered level of consciousness or disorganized thinking would be considered delirious as assessed by this tool.

The 4AT[7] rapid clinical test for delirium is a bedside screening tool that comprises 4 items: an assess-

ment of alertness, separate tests of cognition, and an assessment of changes in mental status.[7]

Electroencephalography. Interestingly, generalized slowing on electroencephalographic monitoring correlates with delirium and may be useful to assess delirium severity.[8]

BEDSIDE CLINICAL ASSESSMENT OF STROKE

Because "time is brain," speed is of the utmost importance when assessing and subsequently treating a potential "brain attack." As mentioned above, although CT is almost reflexively used in the setting of delirium, it rarely reveals a contributing process, suggesting that there is a better way to evaluate our patients.

The National Institutes of Health Stroke Scale (NIHSS) is one of the better known of the many stroke scales. Developed as a research tool to measure outcomes in the National Institute of Neurological Disorders and Stroke rt-PA Stroke Study,[9] over time the scale was truncated and further modified to its present-day version. The original NIHSS had 15 items and assigned a score based on level of consciousness, gaze, visual fields, facial palsy, extremity strength, ataxia, sensation, language, dysarthria, and extinction. Careful assessment of the scale whittled the original 15 items to an essential 11 by eliminating components that were deemed superfluous or poorly reproducible.

The modified NIHSS has been found to be both reliable (ie, different observers will calculate the same score for the same patient) and valid (ie, it correlates with both stroke volumes and clinical outcomes),[10] and it is clinically indicated in every "code stroke." However, it does not fully answer the question as to whether a stroke is occurring.

The 2CAN score[11] was developed as a way for clinicians who are not neurologists to recognize and distinguish inpatient strokes from stroke mimics. Recognition of inpatient stroke is challenging considering the confounding medical conditions and many medications given in the hospital. Possible 2CAN scores range from 0 to 6. Patients get 1 point if they have 1 of the following clinical deficits: asymmetric facial droop; asymmetric arm weakness; or slurred speech, inappropriate words, or inability to speak at all. They get 3 points if they have 2 or more of these deficits. In addition, they get 1 point for each of the following: cardiac surgery in the current hospitalization, history of atrial fibrillation, or being in the hospital less than 24 hours. However, the 2CAN score has not accumulated adequate evidence that it can accurately identify in-hospital strokes.[12]

DELIRIUM: LOOKING FOR OTHER CAUSES, WHILE KEEPING STROKE IN MIND

Regardless of whether the possibility of stroke was ruled out by history and physical examination or imaging, the underlying cause of delirium needs to be identified so that proper treatment can be started.

When approaching a patient in a delirious state, physicians can organize their thinking using the following framework[13]:

- **Neurologic causes:** cerebral hypoxia, seizure, traumatic brain injury, intracranial hemorrhage, brain tumor, hydrocephalus, central nervous system vasculitis, immune-mediated encephalitis
- **Toxic causes:** medications, alcohol, recreational drugs, poisons
- **Metabolic causes:** hepatic encephalopathy, uremia, hypoglycemia, hyperosmolality, electrolyte disturbances, vitamin deficiency, hypercarbia, thyroid disease, Cushing syndrome, hypothermia, hyperthermia
- **Infectious causes:** urinary tract infection, pneumonia, sepsis, meningitis, encephalitis, brain abscess
- **Other causes:** insomnia, hypertension, posterior reversible encephalopathy syndrome.

In the case of our 82-year-old patient, the delirium was most likely due to hypercarbia, a metabolic cause.

THE CASE AGAINST IMAGING

While the differential diagnosis for delirium is very broad, only a handful of the diseases are caused by processes that would require imaging. In fact, most treatable causes of delirium lie outside the brain.[14] By applying the framework described above and assessing the patient with a thorough history, focused physical examination, and appropriate testing reflecting the differential diagnosis, the underlying cause of delirium can be established accurately and would not require imaging.

Not only does excessive imaging weaken our diagnostic reasoning, it also delays proper treatment while we wait for the patient's return from the scanner and for the radiologist's report. This delays time to making a proper diagnosis, subsequently delaying treatment, which can increase morbidity in any medical condition, not just delirium. However, the concepts of stroke mimics[15] and "stroke chameleons" or "hidden

strokes"[16] further challenge our diagnostic abilities and require a heightened level of awareness and understanding that certain presentations may prompt neuroimaging on a case-by-case basis.

THE BOTTOM LINE

Since most causes of delirium are extracranial, imaging is not necessary for every hospitalized patient who develops delirium. Once the diagnosis of delirium is confirmed through the diagnostic criteria outlined in the DSM-5-TR or clinical scores such as the Confusion Assessment Method or the 4AT, the possibility of an underlying cerebral bleeding episode or ischemic process can be evaluated using widely adopted clinical scoring tools such as the NIHSS. These tools, accompanied by the clinician's clinical acumen, can obviate the need for CT, allowing the clinician to think through the differential diagnosis of delirium and narrow the range of potential causes for the individual patient. Subsequent tests and therapies can be ordered accordingly.

DISCLOSURES

The authors report no relevant financial relationships which, in the context of their contributions, could be perceived as a potential conflict of interest.

REFERENCES

1. **Howell JD.** The CT scan after 50 years—continuity and change. N Engl J Med 2021; 385(2):104–105. doi:10.1056/NEJMp2033374
2. **Theisen-Toupal J, Breu AC, Mattison ML, Arnaout R.** Diagnostic yield of head computed tomography for the hospitalized medical patient with delirium. J Hosp Med 2014; 9(8):497–501. doi:10.1002/jhm.2198
3. **Siddiqi N, Stockdale R, Britton AM, Holmes J.** Interventions for preventing delirium in hospitalised patients. Cochrane Database Syst Rev 2007; (2):CD005563. doi:10.1002/14651858.CD005563.pub2
4. **Inouye SK, Westendorp RG, Saczynski JS.** Delirium in elderly people. Lancet 2014; 383(9920):911–922. doi:10.1016/S0140-6736(13)60688-1
5. **American Psychiatric Association.** Diagnostic and Statistical Manual of Mental Disorders. 5th Edition, Text Revision. Arlington, VA: American Psychiatric Association; 2022.
6. **Inouye SK, van Dyck CH, Alessi CA, Balkin S, Siegal AP, Horwitz RI.** Clarifying confusion: the Confusion Assessment Method. A new method for detection of delirium. Ann Intern Med 1990; 113(12):941–948. doi:10.7326/0003-4819-113-12-941
7. **Bellelli G, Morandi A, Davis DH, et al.** Validation of the 4AT, a new instrument for rapid delirium screening: a study in 234 hospitalised older people [published correction appears in Age Ageing 2015; 44(1):175]. Age Ageing 2014; 43(4):496–502. doi:10.1093/ageing/afu021
8. **Kimchi EY, Neelagiri A, Whitt W, et al.** Clinical EEG slowing correlates with delirium severity and predicts poor clinical outcomes. Neurology 2019; 93(13):e1260–e1271. doi:10.1212/WNL.0000000000008164
9. **National Institute of Neurological Disorders and Stroke rt-PA Stroke Study Group.** Tissue plasminogen activator for acute ischemic stroke. N Engl J Med 1995; 333(24):1581–1587. doi:10.1056/NEJM199512143332401
10. **Kasner SE.** Clinical interpretation and use of stroke scales. Lancet Neurol 2006; 5(7):603–612. doi:10.1016/S1474-4422(06)70495-1
11. **Chang P, Ruff I, Bergman D, Mendelson S, Prabhakaran S.** Abstract 193: the 2CAN score: a novel inpatient stroke recognition instrument. Stroke 2018; 49(suppl 1):A193. doi:10.1161/str.49.suppl_1.193
12. **Parrino CR, Noles A, Lalla R, et al.** Optimizing the recognition and treatment of in-hospital stroke: evaluation of the 2CAN score. J Stroke Cerebrovasc Dis 2021; 30(10):106032. doi:10.1016/j.jstrokecerebrovasdis.2021.106032
13. **Mansoor AM.** Frameworks for Internal Medicine. Philadelphia, PA: Wolters Kluwer; 2018.
14. **Marcantonio ER.** Delirium in hospitalized older adults. N Engl J Med 2017; 377(15):1456–1466. doi:10.1056/NEJMcp1605501
15. **Libman RB, Wirkowski E, Alvir J, Rao TH.** Conditions that mimic stroke in the emergency department. Implications for acute stroke trials. Arch Neurol 1995; 52(11):1119–1122. doi:10.1001/archneur.1995.00540350113023
16. **Dupre CM, Libman R, Dupre SI, Katz JM, Rybinnik I, Kwiatkowski T.** Stroke chameleons. J Stroke Cerebrovasc Dis 2014; 23(2):374–378. doi:10.1016/j.jstrokecerebrovasdis.2013.07.015

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