Normal pressure hydrocephalus

(MAY 2006)

TO THE EDITOR: It was refreshing to read Dr. Factora's article, "When do common symptoms indicate normal pressure hydrocephalus?" in the May issue (*Cleve Clin J Med* 2006; 73:447–457). Every week I face families who, because of "news" reports on normal pressure hydrocephalus and direct-to-consumer advertisements for shunts, wonders if their relative has the disorder. Even some of my physician colleagues are now more inclined to test for it. We tend to forget that this is a rare disorder with no evidence-based algorithms for diagnosis or management!

It is sobering to look at the little that is known. For instance, less than 50% of patients improve after a shunt is placed, and unfortunately, a substantial number experience significant adverse events, even death. Then there are some who respond incredibly well!

So what one should do when faced with a patient who may have normal pressure hydrocephalus is almost a guess. I always image the brain with magnetic resonance imaging and at the same time obtain cerebrospinal fluid flow measurements (for whatever that is worth). I often ask for neuropsychometric testing to look for subcortical abnormalities (although I wonder if that has any real value), and in the end, when other disorders have been excluded, the examination and history suggest normal pressure hydrocephalus, and the patient has no strong contraindications, I suggest a shunt.

I have seen "shunt failures" when "everything" pointed to "success" (eg, a positive drainage test, cisternography, positron emission tomography, or single-photon emission computed tomography). I have had "shunt success" when these tests were normal. I then often ask myself, would I shunt this patient if any of these tests came back normal? If the answer is yes, I no longer order any of these specialized tests.

As with much else in medicine, we badly need scientific studies (preferably not sponsored by shunt manufacturers) to guide us!

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Electrocardiography in acute pericarditis

(JANUARY 2006)

TO THE EDITOR: Diagnosing chest pain in patients with ST-segment elevation on an electrocardiogram (ECG) may be challenging.[1]

In the January 2006 issue (*Cleve Clin J Med* 2006; 73:49–50), Dr. Carvalho presented an ECG that was persuasive for pericarditis even though a classic clinical picture was not present. The ECG was obtained from a 32-year-old man presenting with a complaint of severe chest pain, and showed ST-segment elevation with upwards concavity without reciprocal changes.

We suggest that the depression of the PQ segment that was best visible in leads aVL, V_2, and V_3 (with a reciprocal elevation in lead aVR) could have been a useful diagnostic sign in this setting.

Depression of the PQ segment (below the TP segment) is a common ECG feature in acute pericarditis and reflects atrial involvement in the inflammatory process.[2]

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TO THE EDITOR: Dr. Carvalho interpreted ST-segment elevation of the ECG as pericarditis.[1] Several features in this ECG indicate that the ST-segment elevation is due to early repolarization. In nearly all patients with acute pericarditis, J-point height in lead V_6 measures more than 25% of the height of the T-peak from the baseline, which is not the case in the ECG presented.[1,2] ST-segment elevations in pericarditis are typically ubiquitous, whereas in early repolarization they have restricted distribution as in the presented ECG.[2] PR-segment depression is an important feature of pericarditis, though it may not always be present.[3] The heart rate is usually faster than 80 beats/minute unless autonomic nervous system problems coexist.[4] The absence of PR-segment depression and sinus tachycardia in the presented ECG does not support the diagnosis of pericarditis.

In patients with early repolarization, the degree of ST-segment elevation may vary from one recording to another.[5] In one series of 65 patients with the early repolarization pattern, chronological follow-up showed that ST-segment elevation disappeared in 17 (26%) in at least one follow-up ECG, and in 48 patients (74%) the degree of ST-segment elevation varied greatly from one recording to another.[6] During exercise, ST-segment elevation of early repolarization returns to normal baseline.[7]

Finally, the chest pain and elevated creatine kinase in the patient may be due to muscular causes such as trauma or muscle strain.

In conclusion, we believe that the correct interpretation of the ECG is the early repolarization pattern. Some ECG changes in acute pericarditis may resemble those observed in the early repolarization pattern, but numerous ECG criteria can usually distinguish between them.

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Editor's note: Dr. Carvalho could not be reached for a reply.