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Predicting Bad Outcomes in ED Patients With TIA

ABSTRACT & COMMENTARY

Source: Johnston SC, et al. Short-term prognosis after emergency department diagnosis of TIA. *JAMA* 2000;284:2901-2906.

THIS LARGE COHORT STUDY FROM 16 HOSPITALS IN THE KAISER PERmanente HMO of Northern California involved patients identified by emergency department (ED) physicians as having presented with an acute transient ischemic attack (TIA). The main objective was to determine the short-term risk of stroke during the 90 days after the index TIA. Other outcome measures included death, recurrent TIA, and hospitalization for other cardiovascular events. Patients were followed up by searching computerized databases, reviewing medical records, and checking a separate database for hospitalizations outside the system.

Of the 1707 patients with a TIA diagnosis, more than 99% arrived within one day of symptom onset. Mean age was 72 years and the mean symptom duration was 207 minutes. Strokes occurred in 180 patients (10.5%) within 90 days of the index TIA, 91 of which occurred during the first two days. The combined risk of stroke, recurrent TIA, hospitalization for cardiovascular events, and death during the 90-day follow-up period was 25.1%. More than 50% of the combined adverse events occurred within the first four days.

Five factors were independently associated with stroke: age older than 60 years (OR 1.8; 95% CI 1.1-2.7); diabetes mellitus (OR 2.0; 95% CI 1.4-2.9); symptom duration longer than 10 minutes (OR 2.3; 95% CI 1.3-4.2); weakness (OR 1.9; 95% CI 1.4-2.6); and speech impairment (OR 1.5; 95% CI 1.1-2.1). A simple index (1 point for each risk factor) showed that the 90-day stroke risk varied from 0% in patients with no risk factors to 34% in patients with all five.

COMMENT BY STEPHANIE B. ABBUHL, MD, FACEP

If you have experienced concern for the TIA patients you've discharged from the ED, this study confirms your clinical instinct. A

INSIDE

TIA appears to be an ominous sign, conveying a substantial (25%) short-term risk of stroke, recurrent TIA, hospitalization for other cardiovascular events, and death. A 10.5% risk of stroke within 90 days (5% risk within 2 days) is significant and certainly underscores the importance of either admission, neurology consultation, or close follow-up and good communication with the patient and family.

One criticism of the study, noted by the authors themselves, is that the patient cohort was recruited from the EDs of hospitals in a large HMO system. It is possible that disincentives to using the ED led to a selection bias in favor of patients with longer duration of symptoms (the mean was quite long at 207 minutes) and at greater risk for adverse outcomes. On the other hand, at least one other, smaller study found a similar stroke risk.[1] In addition, it would have been interesting to know what percentage of the patients presented with their first TIA as opposed to a recurrent TIA, and if that was associated with the outcomes.

The problem for us, of course, is who to admit, who to hold for a neurology consultatation, and who to discharge. A prediction model based on risk factors would be helpful, and this study is a first step in creating such a tool. Unfortunately, until these risk factors are validated prospectively in an independent cohort, they are still in the development phase. In the meantime, having a low threshold for admission and/or consultation seems prudent. ❖

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The Question of Antibiotic Therapy in Pediatric Enteroinvasive Diarrhea

ABSTRACT & COMMENTARY

Source: Leibovitz E, et al. Oral ciprofloxacin vs. intramuscular ceftriaxone as empiric treatment of acute invasive diarrhea in children. *Pediatr Infect Dis J* 2000;19:1060-1067.

In an effort to optimize the outpatient management of dysentery in children, Leibovitz and colleagues studied 201 cases of invasive diarrhea seen in the pediatric emergency department (ED) in Beer Sheva, Israel. Children already on antibiotics for another reason; those with previous cardiac, renal, or hepatic disease; and those requiring hospitalization were excluded. Seventy-one (35%) were younger than 1 year of age, and 85% were younger than 5 years of age. Average temperature was 39.1° C. Eighty-three (42%) had more than seven bloody-mucoid stools in 24 hours. Mean symptom score was 6.0 (out of 10 maximum). All cases were documented to have greater than 15 white blood cells (WBCs) per high power field on microscopic examination of stool. Mean C-reactive protein was 6.8 (normal: < 1.0). Patients had clinical evaluation, stool cultures, and laboratory tests at enrollment, and on days 1, 2, 3, 5, and 21 during follow-up. In double-blind, prospective fashion, cases were randomized to either three days of oral ciprofloxacin (CIP) solution plus placebo intramuscular (IM) injection, or IM ceftriaxone (CTX) plus placebo oral solution. A pediatric rheumatologist examined each child on follow-up visits.

Overall, clinical "success" was achieved in 200 (99.5%) patients. The only failure involved a 5-year-old with *Shigella*, who was still febrile at day 4 of CIP. Of all isolates (n = 127), *Shigella* species were most frequent

(57%), followed by *Salmonella* (18%), *E. coli* (15%), and *Campylobacter* (10%). Eradication rates were not statistically different between CIP and CTX for infections with *Shigella* (100% vs 97%), *Salmonella* (73% vs 80%), and *Campylobacter* (71% vs 83%), respectively. There was no significant difference in symptom scores, temperature resolution, and C-reactive protein between treatment groups. Children in each group had normal pediatric rheumatologist joint examinations at each follow-up visit. Vomiting and dehydration occurred more often with CIP than CTX (8 vs 3 cases, respectively). The authors conclude that oral CIP is as effective and safe as IM CTX in pediatric dysentery cases.

COMMENT BY MICHAEL FELZ, MD

This study intrigues me for several reasons. First, detection of WBCs in stool wet mounts, as demonstrated by the authors, is simple, rapid, and helpful in distinguishing bacterial from viral diarrhea. Yet this test often is underutilized in acute care settings. I learned to personally inspect stool for WBCs during missionary work in rural Papua New Guinea, but find clinicians in the United States ill-prepared to take this five-minute step in the laboratory. Just this winter, I have seen two children with bloody diarrhea in which sheets of WBCs were visualized on microscopy by me; each grew *Salmonella*. Second, oral CIP achieved clinical success remarkably quickly and reliably in 99% of cases, echoing the data on dysentery in adults. This study advances CIP as an attractive oral option for ED management, pending cultures. Finally, no arthropathy was found in 21 days of follow-up, adding to the accumulated evidence that quinolones may well be safe in children, as documented in hundreds of cystic fibrosis, osteomyelitis, neutropenic, and chronic otitis media patients requiring prolonged therapy with CIP.

Several questions arise from this study, however. Can the results from Israel be generalized to the United States? How does one decide whether to use antibiotics at all, as opposed to watchful waiting, in children sick enough to appear in the ED? What is the risk of hemolytic uremic syndrome associated with empiric antibiotic therapy in cases where *E. coli* 0157:H7 eventually grows in culture (0 cases in this series)? (See also "Hemolytic uremic syndrome in children receiving antibiotics for *E. coli* infection," in *Emerg Med Alert* 2000;7:18-19.) Is 21 days long enough to observe for arthropathy from CIP?

Yet, on balance, I think this study strengthens the armamentarium for treating the wide range of pathogens responsible for invasive diarrhea in the pediatric ED, once clinicians decide that children are ill enough to require therapy, and wish to avoid painful IM injections. Treatment could be as easy as "a sip of CIP." ❖

Clinical Scores, Vegas, and Pulmonary Embolus: A Tale of Playing the Odds

ABSTRACT & COMMENTARY

Source: Wicki J, et al. Assessing clinical probability of pulmonary embolism in the emergency ward: A simple score. *Arch Intern Med* 2001;161:92-97.

THIS FIVE-YEAR STUDY FROM SWITZERLAND EXAMINED the clinical characteristics of patients who presented to the emergency department (ED) with complaints of possible pulmonary embolism (PE). Data were obtained from two prior reports that studied plasma D-dimer in PE. The objective was to develop a simple standardized clinical score to stratify ED patients with clinically suspected pulmonary embolism into groups with a high, intermediate, or low probability of PE to refine the diagnostic approach to be less invasive. A database of 1090 consecutive patients was subjected to a standard algorithm that established diagnostic criteria for PE. Logistic regression was used to predict clinical parameters associated with PE. Two hundred ninety-six patients (27%) were found to have PE. The optimal estimate of clinical probability was based on eight variables: recent surgery, previous thromboembolic event, older age, hypocapnia, hypoxemia, tachycardia, and band atelectasis or hemidiaphragm elevation on chest x-ray. A probability score was calculated by adding points assigned to these variables. The most heavily-weighted variables were recent surgery and room-air hypoxia. The surgery had to have occurred during the last month, and included the following procedures: orthopedic, hip, knee, or extensive

Table

Probability Score Conversion

Variable	Points
Age 60-79	1
Age ≥ 80	2
Previous PE or DVT	2
Recent surgery	3
Pulse rate > 100 bpm	1
$PaCO_2$ < 36 mmHg	2
$PaCO_2$ = 36-38.9 mmHg	2
PaO_2 < 48.7 mmHg	4
PaO_2 = 48.7-59.9 mmHg	3
PaO_2 = 60-71.1 mmHg	2
PaO_2 = 71.2-82.3 mmHg	1
Plate-like atelectasis	1
Hemidiaphragm elevation	1

pelvic or abdominal surgery. The scores have been converted from European units (*see Table*).

A cutoff score of 4 best identified patients with low probability of PE. A total of 486 patients (49%) had a low clinical probability of PE (score ≤ 4). This group had a 10% prevalence of PE. Patients with intermediate scores (5-8) represented 44% of the patients and had a 38% prevalence of PE; 6% of the patients had a score of 9-12 and an 81% prevalence of PE. When compared to the clinician's pretest suspicions (second- and third-year internal medicine residents), the scoring system did better at predicting high and intermediate probabilities, but both predicted low probability patients with similar frequency. The authors conclude that less invasive testing would be warranted in patients with low clinical probability, and that this system identifies those patients.

COMMENT BY RICHARD J. HAMILTON, MD, FAAEM, ABMT

A good friend of mine and I once decided that we'd rather have a successful gambler than a genius as our personal physician. The genius might be correct with a high probability—but one fatal mistake out of thousands is considered poor odds for the successful gambler, who would rather be wrong safely many times than fatally wrong once. Emergency medicine does force us to play the odds because we often must choose treatment and dispositions without complete answers. This dilemma plays out in PE in very concrete ways. We know from the Prospective Investigation of Pulmonary Embolism Diagnosis (PIOPED) study that patients judged to have a low pretest clinical probability of PE and a low probability lung scan have a 4% incidence of PE.[1] Other studies have determined that the addition of a normal lower limb venous ultrasound drives these odds even lower.[2] Even though we may be wrong one time out of 100, at those odds, applying a standard like pulmonary angiography to all low probability patients might increase the morbidity and mortality from procedural complications beyond that native to disease prevalence. Is that a gamble worth taking to make the diagnosis? The answer is not clear. The good news is that this clinical scoring system gives us a concrete method of measuring the odds before we decide how far to go to make this diagnosis or when to begin empiric therapy. I might guess that a particular patient has a particular probability, but the scores could change my suspicions and prompt me to be more or less aggressive, as appropriate.

One limitation is that this study assumes that physicians have performed an arterial blood gas on every patient. It may not be valid to assume that normal pulse oximetry and respiratory rates always mean a normal room air blood gas. While studies like PIOPED suggested that the PaO_2 alone was not helpful in predicting or excluding the diagnosis, this study suggests that it may provide useful information in playing the odds.

Bayes' rule also reminds us that since this study was done in a population with a PE prevalence of 27%, the outcomes are most reproducible in similar populations. In general, if the variables in this study are weighted properly, these findings will be more robust in populations with higher prevalence of PE, and less so when prevalence is lower.[3] As the bottle of hair tonic says: "Your results may vary." ❖

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Special Feature

Treatment of Refractory Sulfonylurea-Induced Hypoglycemia: Beyond Glucose

By Richard A. Harrigan, MD, FAAEM

THERE CURRENTLY ARE THREE GENERATIONS OF SULfonylurea (SU) agents available for the treatment of type 2 diabetes mellitus (DM) (*see Table*). These agents commonly are encountered in the practice of emergency medicine, especially the second- (glipizide, glyburide) and third- (glimepiride) generation drugs. The most common serious toxicity associated with the SUs is hypoglycemia, which occurs in adult and pediatric patients—usually due to accidental ingestion in the latter group. In adults, SU-associated hypoglycemia may be intentional, as in overdose scenarios, or unintentional. Kidney and liver disease, alcohol use, polypharmacy, poor nutrition, and advanced age are factors that have been associated with increased risk of SU-associated hypoglycemia.[1,2] Hypoglycemia recognition usually is not difficult, nor is the initial treatment: glucose administration. But what if your patient continues to be hypoglycemic after two amps of D_{50}? Recurrent or refractory hypoglycemia due to SU poisoning poses a therapeutic challenge; effective management requires a working knowledge of the pharmacology of both the SU agents and the pharmacologic adjuncts used to treat hypoglycemia. After briefly reviewing the pharmacology of SUs, the following discussion will focus on the emergence of octreotide as an attractive supplement to glucose in persistent hypoglycemia secondary to SU toxicity.

Pharmacology of Sulfonylureas

SUs increase the secretion, and amplify the effect, of insulin. In the pancreas, they stimulate insulin release, a mechanism that is enhanced in the presence of glucose. They decrease hepatic insulin clearance; the elevated circulating levels of insulin that result then suppress production of glucose by the liver. SUs also may decrease insulin resistance peripherally and thereby enhance its action.[3,4]

All SUs feature a relatively long duration of action—an attractive attribute from a treatment perspective, but a hindrance to treatment in a poisoning setting (*see Table*). Delays in time-to-peak effect may create an illusion of wellness at the time of initial emergency department (ED) evaluation, especially if the patient presents in the first hour or two after an intentional ingestion. All SUs undergo hepatic metabolism; thus, the presence of hepatic disease (or competition for hepatic metabolism by other drugs) may lead to a protracted duration of action. Chlorpropamide, glyburide, and glimepiride undergo renal excretion of either the parent drug (chlorpropamide) or active metabolites (glyburide and glimepiride [the significance with the latter drug is still unclear]); therefore, concomitant renal insufficiency portends prolonged toxicity. Chlorpropamide, glyburide, and the extended-action formulation of glipizide are the most likely to cause sustained hypoglycemia.[5] (*See Table.*) Glyburide recently was found to be the responsible drug in all study patients who developed protracted hypoglycemia from SUs in a retrospective investigation restricted to a population of end-stage renal disease patients.[6]

Treatment of Hypoglycemia: Beyond Glucose

As discussed above, hypoglycemia refractory to simple glucose administration may develop in cases of SU ingestion confounded by hepatic impairment, renal insufficiency (with some agents), or massive ingestion. In such cases, consideration of adjuncts to glucose is necessary.

Table

Sulfonylureas

Generic Name	Trade Name	Time to Peak (hours)	Duration of Action (hours)
First generation			
Chlorpropamide	Diabinase	2-7	60
Tolbutamide	Orinase	3-4	6-12
Acetohexamide	Dymelor	3	12-18
Tolazamide	Tolinase	4-6	12-24
Second generation			
Glipizide	Glucatrol	1-3	12-24
Glipizide	Glucatrol XL	6-12	24
Glyburide	Miconase, DiaBeta, and Glynase	2-6	12-24
Third generation			
Glimepiride	Amaryl	2-3	16-24

Octreotide

Octreotide, a somatostatin analog, is appealing mechanistically due to its ability to suppress the secretion of numerous endogenous hormones—one of which is insulin. It enjoys experimental and clinical data support for use in SU-induced hypoglycemia. Boyle and associates compared octreotide to diazoxide and glucose in a small (8 patients), simulated, sub-toxic glipizide overdose model using healthy non-diabetic volunteers.[7] The octreotide arm demonstrated significantly less need for supplemental glucose than the diazoxide and glucose arms; indeed, 50% of octreotide patients did not need any supplemental glucose.

Since that time, a number of case reports have attested to octreotide's clinical efficacy. These cases demonstrate successful use of octreotide in adults[8-12] and a child,[13] and include a wide variety of culprit SU agents. A uniformly diminished need for supplemental glucose is evident after octreotide administration (oftentimes no further need); four cases in which insulin levels were measured showed a profound drop in serum insulin levels (elevated due to SU ingestion and glucose administration) after administration of octreotide.[8-10,13] Paradoxically, it should be noted that the glucose that is administered to combat SU-induced hypoglycemia (secondary to SU-induced hyperinsulinism) "feeds the fire," in that glucose also is a potent insulin secretagogue.[8,9,14]

A recent retrospective case series involving nine patients demonstrated a significant reduction in the need for D_{50} and in the number of hypoglycemic events after octreotide administration.[14] The risk for recurrent hypoglycemia in the pre-octreotide phase was 27 times that of the risk in the post-octreotide period.[14]

Data thus far have not demonstrated toxicity with short-term use. Octreotide has been administered both subcutaneously and intravenously, and it has been given successfully via continuous infusion.[8-14] Ideal dosing is yet to be determined; adult dosing thus far has ranged from 40 mcg to 100 mcg/dose;[8-12,14] the lone pediatric case utilized a dose of 25 mcg subcutaneously in a 20 kg 5-year-old.[13] Octreotide has been administered safely in other pediatric scenarios at 1-10 mcg/kg.[15] Intravenous infusions in adults have been administered up to 100-125 mcg/hr.[12,14] Case series data reflect variable dosing quantities and intervals.[14]

Glucagon

Glucagon, which works by mobilizing hepatic glycogen stores and inducing gluconeogenesis, is a less-than-optimal supplement to glucose in the treatment of SU-induced hypoglycemia. First, its success is at least partially dependent upon the existence of adequate glycogen reserves in the liver. Furthermore, glucagon—at least theoretically—worsens the hyperinsulinemic state of SU poisoning in that it, too, stimulates insulin release.[15] It is a reasonable temporizing alternative to glucose if intravenous access is delayed, and thus has a role in the prehospital situation. However, it lacks the advantages of octreotide as a first-line supplement.

Diazoxide

Historically important as a potent vasodilator used in the treatment of hypertensive crisis, diazoxide has long been used in the treatment of SU-associated hypoglycemia due to its ability to raise the blood sugar.[15] In the study by Boyle and colleagues, it was outperformed by octreotide; of note, diazoxide did not suppress insulin levels in that model, and thus lacks the mechanistic appeal of octreotide.

Summary

Octreotide has been touted as a true antidote for SU-induced hypoglycemia. Recent case report and case series data suggest it is rapid-acting, highly efficacious, and well-tolerated. While clinical experience is still mounting, and optimal dosing guidelines are yet to be established, it appears to be a first-line supplement to glucose in the patient with refractory hypoglycemia due to SU poisoning. ❖

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CME Questions

25. The Johnston study followed up 1707 patients with the ED diagnosis of TIA to determine their short-term prognosis. The results indicated all of the following *except*:

a. Strokes occurred in 10.5% of patients within 90 days of the index TIA.
b. The combined 90-day risk of stroke, recurrent TIA, hospitalization for cardiovascular events, or death was 25%.
c. Of those patients who returned to the ED with a stroke, one-half occurred within the first two days of the index TIA.
d. One of the five independent risk factors associated with stroke was male gender.

26. In the Israeli study of pediatric diarrhea, what was the most common bacterial isolate?

a. *Salmonella*
b. *Shigella*
c. *Campylobacter*
d. *Yersinia*

27. Which of the following are possible pitfalls associated with empiric quinolone therapy for enteroinvasive diarrhea in children?

a. An association between antibiotic treatment and the development of hemolytic uremic syndrome in children, when *E. coli* 0157:H7 is the etiologic agent
b. Arthropathy, believed to perhaps occur in children taking quinolones
c. Quinolone-induced hyperparathyroidism
d. Both a and b
e. None of the above

28. In the PIOPED study, patients with a low probability lung scan and a low pretest probability of pulmonary embolism had what incidence of pulmonary embolism?

a. 4%
b. 16%
c. 40%
d. 44%

29. Which of the following sulfonylureas is a poor choice in type 2 diabetics with end-stage renal disease?

a. Glipizide
b. Glyburide
c. Tolazamide
d. Repaglinide

30. A patient presents after sulfonylurea overdose with hypoglycemia refractory to three ampules of 50% dextrose solution, with a bedside glucose of 18 mg/dL. All of the following are reasonable therapeutic adjuncts *except*:

a. octreotide.
b. diazoxide.
c. glucagon.
d. acetohexamide.

31. Octreotide:

a. is an insulin secretagogue.
b. is a synthetic antibody active against hepatic binding sites for glucose.
c. has been shown to decrease the need for glucose therapy in sulfonylurea overdose.
d. is dependent upon adequate glycogen stores for efficacy.

Seeing the Clue to Bradycardia

By Ken Grauer, MD

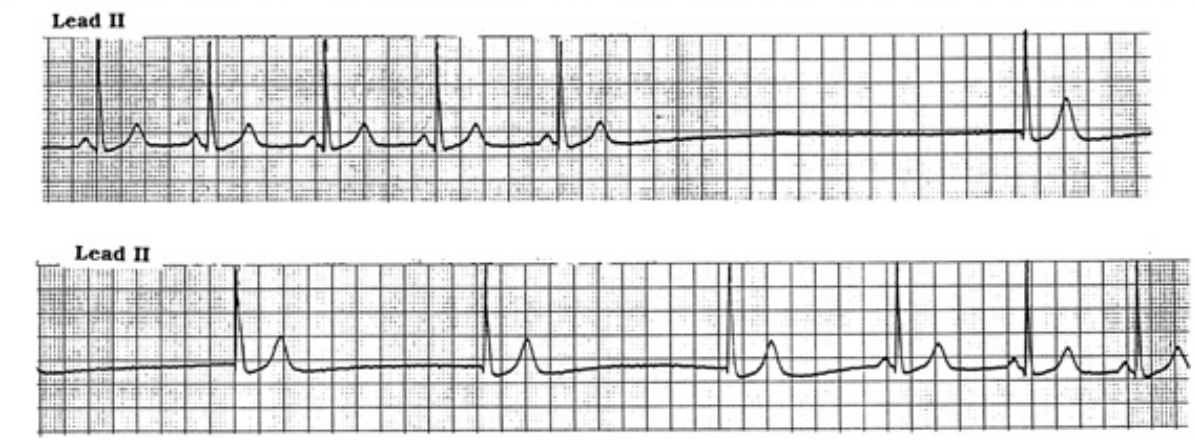

Figure. Telemetry strips from an elderly man taking lots of pills.

Clinical Scenario: The continuous telemetry strips shown here were obtained from an elderly man who was taking multiple medications. Digoxin, verapamil, diltiazem, and beta-blockers were not among the pills he was taking. How would you interpret the rhythm? Clinically, what would you do?

Interpretation: The tracing begins as a sinus rhythm that slows and then abruptly stops. The worrisome pause in the top tracing is just under four seconds long. Asystole is prevented by a junctional escape rhythm that itself is inappropriately slow (although much preferred to the alternative). Sinus node activity finally resumes with the last three beats on the tracing.

The first priority in management is to assess the patient and address immediate treatment needs of the rhythm disturbance. The patient in this case felt faint momentarily, but thereafter was not symptomatic. Recurrence of marked bradycardia to the degree shown in these tracings was not seen. Were bradycardia to recur, treatment with atropine and/or pacing would clearly be indicated.

Clinically, one should assess for potential causative factors. The rhythm strips seen in these tracings could result from a marked vagal response, as might occur after an episode of severe vomiting, or in an elderly patient following prolonged straining at stool. As noted in the history, the patient in this case was not taking any of the *pills* that are usually associated with drug-induced bradycardia. However, no mention is made of a number of other substances that also may produce rate slowing (e.g., clonidine, beta-blocker *eye drops* that are, at least to some extent, systemically absorbed, and certain herbal medicines such as cardioactive glycoside derivatives and veratrum). Finally, a 12-lead ECG should be obtained to rule out myocardial infarction as a possible cause of the bradycardia. If the above evaluation does not suggest a reason for bradycardia, the patient most likely has sick sinus syndrome that will probably require permanent pacing. In this particular case, further questioning revealed the patient was using beta-blocker eye drops for treatment of glaucoma. Episodes of bradycardia resolved completely once this medication was stopped. ❖

In Future Issues:

When to Use HCO_3 in the Treatment of DKA