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INSIDE

Medical Emergency Teams: Does Rapid Response Make a Difference?

ABSTRACT & COMMENTARY

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Dr. Kleinpell reports no financial relationship to this field of study.

This article originally appeared in the August 2010 issue of Critical Care Alert. *It was edited by David J. Pierson, MD and peer reviewed by Saadia Akhtar, MD. Dr. Pierson is Professor, Pulmonary and Critical Care Medicine, Harborview Medical Center, University of Washington, Seattle, and Dr. Akhtar works for St. Luke's Idaho Pulmonary Associates, Boise. Drs. Pierson and Akhtar both report no financial relationships relevant to this field of study.*

Synopsis: *In this before-and-after study of more than 275,000 patients admitted to a Swedish hospital before-and-after implementation of a medical emergency team, in-hospital cardiac arrests decreased and overall in-hospital mortality fell by 10% in the two years following the team's implementation.*

Source: Konrad D, et al. Reducing in-hospital cardiac arrests and hospital mortality by introducing a medical emergency team. *Intensive Care Med* 2010;36:100-106.

THE USE OF A RAPID RESPONSE SYSTEM (RRS), OR MEDICAL EMERGENCY team (MET), has become established as a patient safety measure to ensure early detection of patient compromise. It has been demonstrated that 50%-80% of in-hospital cardiac arrests are preceded by some clinical signs of instability, such as abnormalities in pulse rate, respiratory rate, mental status, or oxygen saturation (SpO_2).[1-3] Early detection and response to promote timely recognition of patients with physiological deterioration with the use of a RRS has been identified as a way to decrease mortality rates by intervening and potentially preventing a cardiac arrest.

This prospective before-and-after trial of implementation of an MET at a hospital in Sweden examined the impact on hospital mor-

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tality rates over a two-year intervention period that included 73,825 patients compared to a five-year pre-implementation period with 203,892 patients. The MET team consisted of an ICU physician and ICU nurse who responded to calls based on one or more standard triggers, including vital sign changes, changes in mentation, or staff concern about the patient. The primary outcome was the number of cardiac arrests per 1,000 admissions, and secondary outcomes included adjusted hospital mortality and 30- and 180-day mortality for patients receiving an MET intervention.

The number of MET calls was 9.3 per 1,000 hospital admissions. In comparing pre-MET and MET intervals, the number of cardiac arrests decreased from 1.12 per 1,000 patients to 0.83. MET implementation was associated with a reduction in total hospital mortality by 10% ($p = 0.003$). The results of the study indicated that the introduction of the MET improved outcomes for hospitalized patients.

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The findings of the study provide further evidence on the benefit of the use of RRSs in decreasing mortality rates in hospitalized patients. Support for the use of RRSs by quality organizations such as the Institute for Healthcare Improvement, in conjunction with the 2008 Joint Commission's designation of the use of systems to promote health care clinician response to a patient's worsening condition as a National Patient Safety goal have led to widespread implementation of RRSs. However, while studies have shown that an RRS may improve outcome, questions remain about the specific components, best format, and ultimate benefits of the RRS.[4]

Interpreting the Evidence Related to the Use of Rapid Response Teams. The findings by Konrad and colleagues support the results of other studies that have demonstrated a reduction in in-hospital cardiac arrests with the use of a RRS.[4-7] Yet, ongoing debate continues regarding the advantages of implementing a RRS as other studies have found no difference in reducing hospital mortality. The MERIT (Medical Early Response Intervention and Therapy) study, a large 23-hospital Australian study evaluated the use of METs with 12 hospitals receiving training in the MET process and implementing a program, compared to 11 hospitals that did not receive training and were asked to delay introduction of a MET system during the study period.[8] During a six-month follow-up period after implementation, no differences were found in cardiac arrests, unplanned ICU admissions, or unexpected death.[8] However, the authors cited inadequate utilization of the MET for patients who met clinical criteria and concluded that despite similar outcomes in both study groups, system-based interventions can have an impact on improving monitoring of patients and promoting appropriate clinician response. Additionally, the use of the randomized clinical trial design in demonstrating benefit of the RRS has since been debated.[9,10]

The findings by Konrad and colleagues also support prior research that has demonstrated a reduction in overall hospital mortality with the use of a RRS. An overall reduction in hospital mortality of 10% was found, lending support to other studies that have shown a reduction in hospital mortality of up to 26%.[6] A recent meta-analysis of 18 randomized clinical trials and prospective studies of the use of a RRS found a 33.8% reduction in rates of non-ICU cardiac arrests, but no significant differences in hospital mortality rates for adult patients.[11] However, for children, implementation of an RRS was associated with a 37.7% reduction in rates of non-ICU cardiac arrests and a 21.4% reduction in hospital mortality rates.[11]

To establish consensus on issues related to use of the RRS, a consensus conference of international experts examined characteristics of an "ideal" monitoring system.[12] Four specific components of the RRS were considered, including the afferent or crisis detection and response triggering mechanism, an efferent or predetermined rapid response team, the governance/administrative structure, and a mechanism to evaluate crisis antecedents and promote hospital quality improvement processes.[12] The findings designated sufficient data to support that hospitals implement a RRS and that outcome benefits exist in terms of reduced deaths, cardiac arrest, hospital length of stay, ICU length of stay, and costs. However, additional research was advocated to determine the magnitude and benefit.[12]

It becomes evident that hospital-wide initiatives to educate and assist clinicians in early identification and rapid treatment of life-threatening conditions that include a team response and protocols based on best practice guidelines can

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improve outcomes.[13,14] In addition, the use of track and trigger systems, combined with appropriate response algorithms, has been advocated to improve the recognition and management of critical illness.[1] As the severity of illness among hospitalized patients continues to increase, it becomes intuitive that RRS would be beneficial in promoting early detection of compromise. The results of research support that rapid response does make a difference, yet the quality of evidence that currently exists necessitates further work to validate the full utility of the RRS. ■

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PO Is OK for COPD — Follow the Guidelines!

ABSTRACT & COMMENTARY

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This article originally appeared in the August 15, 2010 issue of Internal Medicine Alert. *It was edited by Stephen A. Brunton, MD, and peer reviewed by Gerald Roberts, MD. Dr. Brunton is Adjunct Clinical Professor, University of North Carolina, Chapel Hill, and Dr. Roberts is Assistant Clinical Professor of Medicine, Albert Einstein College of Medicine, New York, NY. Dr. Brunton is a consultant for Novo Nordisk, Shionogi Pharma, and Takeda, receives grant/research support and serves on the speaker's bureau for Novo Nordisk. Dr. Roberts reports no financial relationships relevant to this field of study.*

Synopsis: *There is no difference in rates of treatment failure, death, or readmission for COPD between patients treated with oral or intravenous steroids for exacerbation of COPD, but the IV route may be associated with increased cost and length of stay.*

Source: Lindenauer PK, et al. Association of corticosteroid dose and route of administration with risk of treatment failure in acute exacerbation of chronic obstructive pulmonary disease. *JAMA.* 2010;303:2359-2367.

Although systemic steroids are widely used and accepted as part of the routine treatment of COPD exacerbations, very little is known about the best route of administration or optimal dose in patients hospitalized for COPD exacerbations. The aim of this study was to compare the outcomes of patients treated with low doses of oral steroids to those treated with higher doses of intravenous (IV) steroids. The study was carried out at 414 geographically diverse U.S. hospitals during 2006 and 2007. To be included in the study, patients had to be 40 years or older, have a principal diagnosis of COPD, or respiratory failure with acute exacerbation of COPD. They were excluded if they were admitted directly to the intensive care unit, had a secondary diagnosis of pneumonia or pulmonary embolism, were admitted for only only day, or were transferred to or from another acute care facility. In addition to patient age, sex, race/ethnicity, and insurance status, the authors were able to collect data about many other comorbidities and hospitalization history, as well as treatment during the hospitalization of interest for this study.

Patients were categorized in the high-dose IV therapy group if their first recorded dose of corticosteroids was given IV and was within a 120-800 mg/day range of the equivalent of prednisone. Patients were considered to be in the low-dose oral treatment group if they were initially treated with between 20 and 80 mg/day of prednisone by mouth. The main outcome variables were treatment failure (defined as requiring mechanical ventilation after the second hospital day), inpatient mortality, or readmission for acute exacerbation of COPD within 30 days of discharge. The authors also investigated length of stay and cost.

During the two years of study, 79,985 patients met criteria for inclusion in this analysis. A total of 73,765 (92%) were initially treated with IV steroids. There were no differences in outcomes according to route and dose of corticosteroid; 1.4% of the intravenously and 1.0% of the orally treated patients died during the hospitalization; 10.9% of the intravenously and 10.3% of the orally treated patients experienced at least one of the following: treatment failure, death during the hospitalization, or readmission for COPD within 30 days of

discharge. Adjusting for confounders did not change these findings. The authors also undertook an analysis based on the likelihood of being treated with low-dose oral steroids; this model included such things as attending specialty, treatment patterns by hospital, and other treatments. In this propensity-matched analysis, the risk of treatment failure, length of stay, and cost were significantly lower among orally treated patients. When compared with those in the high-dose intravenous group, patients treated with low-dose oral steroids were marginally older, included a lower proportion of white patients, and were less likely to have private insurance. Patients treated with low doses of orally administered steroids also were sicker; they were more likely to have diabetes, heart failure, anemia, and renal failure. When compared with those initially treated intravenously, those who got low-dose oral steroids were less likely to receive early treatment with antibiotics, methylxanthine bronchodilators, to undergo arterial blood gas analysis, and to receive non-invasive ventilation in the first 2 hospital days. Treatment with low-dose orally administered steroids was more common in the Northeast, at larger hospitals, and those with teaching programs. A total of 1356 patients (22%) initially treated with low-dose oral steroids were later switched to intravenous therapy.

Other interesting characteristics of this group of patients were revealed by this analysis. Their median age was 69 years, 61% were women, 73% were white, and Medicare was the most common form of health insurance. Hypertension, diabetes, heart failure, and depression were the most frequently recorded comorbidities. Of these patients, 17% had one admission for COPD in the year before the index hospitalization, and 12% had two or more. Eighty percent were admitted directly from the emergency department, and the vast majority was cared for by general internists or family physicians. The median length of stay was four days, median costs were $5,021, and 30% were hospitalized for six days or longer.

■ COMMENTARY

COPD exacerbation is among the top 10 leading causes of hospitalization nationwide,[1] and the data here demonstrate that it is usually managed by generalists. Standard treatment includes supplemental oxygen, short-acting bronchodilators, systemic corticosteroids, and often antibiotics. Steroids have been found to improve lung function, to reduce the risk of treatment failure, and to decrease length of hospital stay for patients with COPD exacerbation,[2-4] but very little work has been done comparing the efficacy of different doses and routes of administration. Clinical guidelines produced by leading professional organizations actually recommend the use of low doses of steroids given by mouth,[5,6] and this study indicates that these recommendations are appropriate. Amazingly, however, this study also indicates that an overwhelming majority (more than 90%) of practitioners caring for patients with COPD exacerbation in the "real world" are ignoring these recommendations. As the authors of the current paper put it, "This practice does not appear to be associated with any measurable clinical benefit and at the same time exposes patients to the risks and inconvenience of an intravenous line, potentially unnecessarily high doses of steroids, greater hospital costs, and longer lengths of stay." ■

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Blood Culture-negative Endocarditis: What Can the Laboratory Bring to the Table?

ABSTRACT & COMMENTARY

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Dr. Baron reports no financial relationships relevant to this field of study.

This article originally appeared in the August 2010 issue of Infectious Disease Alert. *It was edited by Stan Deresinski, MD, FACP, and peer reviewed by Timothy Jenkins, MD. Dr. Deresinski is Clinical Professor of Medicine, Stanford University; Associate Chief of Infectious Diseases, Santa Clara Valley Medical Center, and Dr. Jenkins is Assistant Professor of Medicine, University of Colorado, Denver Health Sciences Center. Dr. Deresinski serves on the speaker's bureau for Merck, Pfizer, Wyeth, Ortho-McNeil (J&J), Schering-Plough, and Cubist, does research for the National Institute of Health, and is an advisory board member for Schering-Plough, Ortho-McNeil (J&J), and Cepheid, and Dr. Jenkins reports no financial relationships relevant to this field of study*

Synopsis: *The etiology of blood culture-negative endocarditis was identified in 62.7% of 759 patients using combinations of serological, molecular, and histopathological assays. The majority of the agents not detected by conventional cultures were* Coxiella burnetii *(30%) and* Bartonella spp.

(11%), both diagnosed using serological tests, and Trophyrema whipplei*, other unusual bacteria, and fungi making up the remainder. For 14% of patients, diagnosis required PCR testing on valve tissue removed during surgery (not in paraffin). The cause of disease could not be determined for 264 patients; 2% of patients had non-infectious endocarditis.*

Sources: Pierre-Edouard Fournier, et al. Comprehensive diagnostic strategy for blood culture — negative endocarditis: A prospective study of 819 new cases. *Clin Infect Dis*. 2010;51:131-140.

SEVERAL YEARS AGO, TWO DIFFERENT PUBLICATIONS APPEARED SHOWing that prolonged incubation of blood cultures beyond the initial five-day protocol for patients with suspected fastidious bacterial endocarditis did not yield significant additional pathogens with today's modern blood-culture media and automated methods.[1,2] Many laboratories, however, still have problems convincing clinicians that such measures won't be productive. The current publication by the renowned Raoult group confirms the findings of those less sophisticated studies, and suggests an algorithm that will generate the diagnosis in the majority of circumstances in which a diagnosis is possible. Unfortunately, almost no other laboratories have the capabilities of the Raoult laboratory, so that clinicians outside France should not expect to see the same results from their local, or even their reference, laboratory.

This group has pioneered the use of cutting edge molecular technologies for detecting pathogens in the bloodstream and in tissues, and has actively pursued development and documentation of new tools for diagnosis of this syndrome for more than 15 years.[3-6] In my opinion, it is the most competent laboratory in the world to find an etiological agent of endocarditis when all else has failed. Unfortunately, laboratories in the United States are prevented from sending samples to France because of regulatory restrictions. Reference laboratories must be accredited by the state and CLIA, for example, in California, and laboratories accredited by the College of American Pathologists are supposed to send tests out only to other CAP-accredited laboratories. Neither regulatory body is recognized in France.

In this most recent publication, the group investigated the etiology of disease in 819 patients suspected of having blood culture-negative endocarditis (BCNE); approximately 213 involved native valves. Detailed clinical histories were obtained, along with several types of samples, when possible, including serum, EDTA and heparinized blood samples, excised valve tissue frozen at -80° C, and tissue in paraffin blocks, all received over the eight years of the study period. Sixty patients without a diagnosis of endocarditis, based on the modified Duke Criteria, were originally excluded.[7]

The agents tested for by serological testing were *Coxiella, Bartonella, Legionella,* and *Mycoplasma pneumoniae*. Blood, heart-valve tissue, and other tissues were extracted, and specific primers employed, for PCR testing after initial tests yielded no results. Primers for all bacteria, all fungi, and individual species *Coxiella, Bartonella, Tropheryma*, and *Chlamydia,* as well as cytomegalovirus and enteroviruses were used. Tissue cultures were set up on tissues, and if bacteria were seen by any of a number of fluorescent and histological stains, they were identified by universal primer amplification and sequencing. Tissues in paraffin block were stained with immunoperoxidase, acid-fast, and additional special stains such as Warthin-Starry and other silver stains. Autoimmunohistochemistry was used on tissues for which no etiology was detected by other means. A number of tests were employed to detect autoantibodies, such as rheumatoid factor, anti-DNA, and antinuclear antibodies.

Nineteen of the 759 patients included in the study group had non-infectious endocarditis, including marantic endocarditis (7), Libmann-Sacks endocarditis (4), and Behcet disease (1). Seven more patients had autoimmune disease diagnosed elsewhere by additional tests. By the Duke criteria, 549 of 740 remaining patients were designated as having definite endocarditis, and Raoult's group was able to detect an etiological agent in 476 (86.7%) of those. One-hundred ninety-one patients (~ 26%) were classified as "possible" endocarditis. Serologic tests provided the diagnosis in almost half of the patients, emphasizing the importance of this modality, often overlooked by clinicians in their zeal to order molecular assays.

Employing all possible tests, the authors detected 315 cases due to Q fever (*Coxiella*), 86 due to *Bartonella*, 12 due to *T. whipplei*, eight fungi, and 70 conventional bacteria, predominantly streptococci (many *S. gallolyticus*) and staphylococci. Readers of *Infectious Diseases Alert* may remember that *S. gallolyticus* was discussed in a previous *Infectious Disease Alert* article on the recent changes in the taxonomy of the *S. bovis* group.[8] PCR on EDTA blood samples provided the diagnosis in < 15% of all patients. In fact, PCR tests using blood detected only three of the seronegative patients, although valve biopsies were more fruitful, providing a bacterial diagnoses for 66.1% of samples and a fungal diagnosis for 3% among patients from whom valve samples were available. Cell culture never revealed an agent that was not detected by another method, for which microbiology laboratories can breathe a sigh of relief. Valve biopsies were positive by culture more often than blood as well, yielding an organism in 58 of 127 patients (46%). PCR of valves yielded a positive result in 157 of 227 cases (69%).

The authors summarized their findings with a proposed algorithm. It must be remembered that the majority of patients lived in France, other European countries, and Algeria, and only a few patients from the United States were studied, so the results of a similar series might be expected to have different outcomes if conducted in the United States. The authors of this series suggest that after the initial negative

blood cultures, serum should be the first specimen to evaluate for Q fever and *Bartonella*, followed by specific PCR assays for *Bartonella* species, *T. whipplei,* and fungi. For patients with negative results at this point, additional serological and PCR tests could be employed. They also suggested that tests for antinuclear antibodies and rheumatoid factor should be instituted at this point. The authors also recommend that heart valve biopsies should first be tested using broad-range PCR assays for bacteria and fungi, with autoimmuno-histochemistry only for patients with all other results negative. Although they had tested the commercial Roche SeptiFast blood culture multi-analyte PCR system, available in Europe but not FDA-cleared in the United States, they found its sensitivity to be so sufficiently poor that they did not recommend its use. Instead, they suggested that if laboratories had specific staphylococcal and streptococcal PCR assays available, they should be used. Of course, right now in the United States there are no commercial tests available for use on blood directly from the patient without the benefit of culture amplification in blood cultures.

In summary, serological tests are the second most productive modality for diagnosis of culture-negative endocarditis, followed by PCR, and then even more heroic measures, if they are available. Even then, one-third of cases remained for which an etiology could not be determined. ■

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Do Rapid Response Teams Reduce Hospital Mortality or Simply Increase Costs?

ABSTRACT & COMMENTARY

By Michael Young, MD

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Dr. Young reports no financial relationship to this field of study.

This article originally appeared in the August 2010 issue of Critical Care Medicine. *It was edited by David J. Pierson, MD, and peer reviewed by Saadia Akhtar, MD.*

Synopsis: *This meta-analysis casts serious doubt on the ability of rapid response teams to significantly reduce hospital mortality.*

Source: Chan P, et al. Rapid response teams: A systematic review and meta-analysis. *Arch Intern Med* 2010;170:18-26.

In the past decade, rapid response teams (RRTs) were broadly implemented to identify and treat patients on medical and surgical wards at risk for catastrophic deterioration and thus prevent death. The impetus to form RRTs came after multiple observational studies in the 1990s suggested that ward patients who experience a marked clinical deterioration often suffer excess morbidity and mortality from delayed treatment and underresuscitation. A number of single-center studies indicated that RRT implementation was associated with reductions in cardiopulmonary arrests outside the ICU and decreases in hospital mortality. However, two meta-analyses on RRTs published in 2007 were far more cautionary on their purported benefits. Despite equivocal evidence favoring RRTs, RRT implementation was an integral part of the "Save 100,000 Lives Campaign" lead by the Institute of Healthcare Improvement (IHI), and in 2008 the Joint Commission listed implementation of a RRT (or equivalent) as a National Patient Safety Goal.

The present meta-analysis by Chan and colleagues examines the effect of RRT implementation on cardiopulmonary arrest rates and hospital mortality. The authors' search strategy yielded 143 potentially relevant articles on the basis of title and abstract. Additional articles were excluded because they were simply review articles, lacked a control group, failed to evaluate mortality or CPR rates, were duplicates of other articles, or provided insufficient data. Eighteen articles, including 13 adult and five pediatric studies, published between 2000 and 2009, were included in the meta-analysis. Of the 6 studies classified as "high-quality," 2 were randomized clinical trials and 4 were observational in design.

Some of the adult RRT studies included in the meta-analysis

showed a reduction in cardiopulmonary arrest rates while others did not. Cumulatively, the adult high-quality studies identified a 21% decrease in non-ICU cardiopulmonary arrest rate compared to a 48% decrease in the other studies. In contrast, the pooled mortality rate for adult patients was unaffected by implementation of RRTs in both high- and low-quality studies, and in studies with high- vs low-RRT activation rates. In the pediatric population, there was a lower mortality rate (relative risk [RR], 0.79; 95% confidence interval [CI], 0.63-0.98) with RRT implementation, but the improvement was not robust. Furthermore, when the adult and pediatric results were pooled, mortality rate with RRT implementation did not change (RR, 0.92; 95% CI, 0.82-1.04).

Among the five studies that reported a lower mortality rate with RRT implementation, Chan et al measured whether the reduction in cardiopulmonary arrest rate could explain the improvement. Their analysis found that the observed reduction in cardiopulmonary arrest rates could explain < 1% to as much as 61% of the mortality reduction.

■ COMMENTARY

This study is now part of growing series of meta-analyses indicating that the benefit of RRTs may be more modest than widely believed. If RRTs' benefits are measured largely by their impact on hospital mortality, the enthusiasm for RRT formation by the Joint Commission and IHI is not easily supportable. On the other hand, the data do indicate that RRTs reduce the number of cardiopulmonary arrests outside the ICU among both adult and pediatric patients. Given the discouraging mortality and morbidity associated with in-hospital cardiopulmonary arrest, this is an important benefit. There may be other benefits that RRT implementation provides that have not been well quantified. Nurse and patient/family satisfaction are reported to be improved with RRT implementation. Clinician education may be improved and delays in transfer to the ICU from the ward may be decreased. More research in these areas is needed.

Perhaps equally important, this study reminds us how difficult it can be to interpret the results of single-center studies, especially those that use a "before-and-after" design. RRTs may be another example of where we, as clinicians, adopt therapies or interventions that simply make "sense" to us without waiting for well-done outcomes studies. Once rigorously studied, we may find the story more complex than we suspected and the therapy less helpful than we anticipated and sometimes even more harmful than helpful — for example, lidocaine post-myocardial infarction, aggressive use of total parenteral nutrition in the ICU, and routine use of pulmonary artery catheters among patients with acute lung injury and the ARDS.

Why the remarkable variation in outcomes seen in the before-and-after RRT implementation studies? There are probably a number of factors at play. RRT composition is widely variable. The clinical thresholds used to prompt RRT activation vary. What are the sensitivities and specificities of those clinical thresholds' ability to predict catastrophic deterioration? In about 50% of cases, RRT are activated because of a "worried clinician." When should clinicians become worried? What interventions should the RRT provide when the RRT arrives at the bedside of a patient suffering measurable physiologic or subjective deterioration? What is the right rate of RRT activation? Once activated, how long should the RRT stay at the patient's bedside? When and which patients should be transferred from the ward to the ICU? How often should the vital signs of patients on medical and surgical wards be measured and reported? How accurate are those measurements?

Should RRTs be disbanded? In my view, no. However, until we conduct serious research to find answers to these questions, the real potential of RRTs to prevent morbidity and mortality among patients on hospital wards will remain unknown. ■

Catching Dengue in Florida

ABSTRACT & COMMENTARY

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This article originally appeared in the August 2010 issue of Infectious Disease Alert. *It was peer reviewed by Timothy Jenkins, MD. Dr. Jenkins is Assistant Professor of Medicine, University of Colorado, Denver Health Sciences Center; he reports no financial relationships relevant to this field of study.*

Synopsis: *Autochthonous transmission of dengue virus infection has been detected in Florida.*

Source: CDC. Locally acquired dengue — Key West, Florida, 2009-2010. *MMWR*. 2010;59:577-581.

A PHYSICIAN IN NEW YORK NOTIFIED RELEVANT COUNTY AND state public health authorities in August 2009, of a patient from Rochester with suspected dengue, subsequently confirmed by CDC, whose only travel had been to Key West, FL. Confirmation at the CDC included both serum antibody testing and detection of dengue virus serotype 1 in cerebrospinal fluid. The patients subsequently completely recovered. Within two weeks, two residents of Key West who had not traveled were found to have dengue. Increased truck and aerial spraying and an intense door-to-door campaign to detect and eliminate mosquito breeding sites were instituted. Since enhanced and active surveillance was implemented by April 13, 2010, a total of 28 cases had been identified.

A serosurvey of residents found that 13 of 240 (5.4%) had evidence of recent dengue infection. Of 21 speci-mens from patients with suspected dengue submitted from Sept. 23, 2009-Nov. 27, 2009, nine (42.9%) were positive. An additional two cases were detected by review of medical records from three Key West Health facilities. The median age was 47 years (range, 15 -73 years), and approximately two-thirds were male. Fever occurred in all, and most had headache and myalgia, with or without arthralgia. Fifty percent had ocular pain and 54% had a skin rash. Six patients reported bleeding, including hematuria (4), gingival bleeding (2), vaginal bleeding (1), and epistaxis (1).

■ COMMENTARY

This is the first evidence of acquisition of dengue virus infection in the continental U.S. (dengue has occurred in Hawaii) since cases along the Texas-Mexico border in 1945 and the first in Florida since 1934. Dengue is endemic in Puerto Rico, the U.S. Virgin Islands, and U.S. territories in the Pacific. In 2007, more than 10,500 cases of dengue were reported among American citizens in the continental United States and its territories, mostly in Puerto Rico.[1] These cases, and the serosurvey data, indicate that dengue has established a footlhold in Florida. The widespread presence of *Aedes aegypti* throughout the southern U.S. and *Aedes albopictus* in the southeastern U.S. provide continuing cause for concern regarding its reemergence throughout these areas.

Dengue was added to the National Notifiable Diseases Surveillance System list of nationally notifiable infectious diseases in 2009.[1] Laboratory confirmaton of cases require one or more of the following:

• Direct identification of the virus by culture or PCR (requires collection of the specimen within the first five days of illness).

• Seroconversion between acute and convalescent serum specimens obtained within 30 days of symptom onset.

• Detection of NS-1 antigen (the test is, however, not FDA-approved).

• Virus-specific IgM antibody in cerebrospinal fluid.

The presence of dengue-specific IgM antibody in serum, in the presence of a compatible clinical picture, can only be considered probable (not confirmed) evidence of dengue because of the possibility of false-positive tests.

By the way, kudos to the anonymous infectious disease specialist in New York who made the diagnosis in the first case! ■

Reference

1. http://www.cste.org/ps2009/09-id-19.pdf

CME Questions

1. **In the retrospective, nonrandomized, chart review of over 79,000 people hospitalized for a COPD exacerbation, Lindenauer, et al., observed that treatment with low-dose oral steroids during hospitalization led to which of the following observations?**

a. No difference in the rate of treatment failure compared to high-dose IV steroids.

b. No difference in mortality compared to high-dose IV steroids.

c. Most patients during the study period where treated initially with high-dose IV steroids contrary to published guidelines.

d. All of the above

2. **According to the study from France by Pierre-Edouard Fournier and colleagues on patients with blood culture-negative endocarditis:**

a. Prolonged incubation of the blood cultures for fastidious organisms was the most useful step in detecting an etiology.

b. Serology for Coxiella burnetti and Bartonella spp. is the next most helpful step once blood cultures are negative.

c. Despite an extensive workup, the etiology in approximately one-third of cases remains unknown.

d. Both B and C are correct; A is incorrect.

3. **Based on the two recent studies on Medical Emergency/ Rapid Response Teams, which of the following statements is correct?**

a. The literature on the efficacy of these teams consistently demonstrates their clinical benefit.

b. Different hospitals use different physiologic criteria to trigger a rapid response.

c. In-hospital mortality is higher in patients who receive care from a medical emergency/rapid response team.

d. All of the above

Answers: 1. (d); 2. (d); 3. (b)

CME / Objectives

Upon completion of this educational activity, participants should be able to:

- discuss pertinent safety, infection control and quality improvement practices;
- explain diagnosis and treatment of acute illness in the hospital setting; and
- discuss current data on diagnostic and therapeutic modalities for common inpatient problems. ■

Dear *Hospital Medicine Alert* Subscriber:

This issue of your newsletter marks the start of a new continuing medical education (CME) or continuing nursing education (CNE) semester and provides us with an opportunity to review the procedures.

Hospital Medicine Alert, sponsored by AHC Media LLC, provides you with evidence-based information and best practices that help you make informed decisions concerning treatment options and physician office practices. Our intent is the same as yours — the best possible patient care.

Upon completing this program, the participants will be able to:

1. review pertinent safety, infection control, and quality improvement practices;
2. discuss diagnosis and treatment of acute illness in the hospital setting;
3. review current data on diagnostic and therapeutic modalities for common inpatient problems.

Each issue of your newsletter contains questions relating to the information provided in that issue. After reading the issue, answer the questions at the end of the issue to the best of your ability. You can then compare your answers with the correct answers provided in an answer key in the newsletter. If any of your answers were incorrect, please refer back to the source material to clarify any misunderstanding.

At the end of the semester, you will receive an evaluation form to complete and return in an envelope we will provide. Please make sure you sign the attestation verifying that you have completed the activity as designed. Once we have received your completed evaluation form, we will mail you a letter of credit. This activity is valid 24 months from the date of publication. The target audience for this activity is principal investigators and clinical trials nurses.

If you have any questions about the process, please call us at (800) 688-2421, or outside the U.S. at (404) 262-5476. You can also fax us at (800) 284-3291, or outside the U.S. at (404) 262-5525. You can also email us at: customerservice@ahcmedia.com.

On behalf of AHC Media, we thank you for your trust and look forward to a continuing education partnership.

Sincerely,

Valerie Loner
Director of Continuing Education
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EVIDENCE-BASED INFORMATION FOR HOSPITALISTS, INTENSIVISTS, AND ACUTE CARE CLINICIANS

HOSPITAL MEDICINE ALERT

HMED090110TM

CE/CME Evaluation

Please take a moment to answer the following questions to let us know your thoughts on the CE/CME program. Fill in the appropriate space and return this page in the envelope provided. You must return this evaluation to receive your certificate. Thank you.

CORRECT ● **INCORRECT** ⊘ ✓ ✗ ⚠

1. In which program do you participate? ○ CE ○ CME

2. If you are claiming physician credits, please indicate the appropriate credential: ○ MD ○ DO ○ Other _____

3. If you are claiming nursing contact hours, please indicate your highest credential: ○ RN ○ NP ○ Other _____

	Strongly Disagree	Disagree	Slightly Disagree	Slightly Agree	Agree	Strongly Agree
After participating in this program, I am able to:						
4. Review pertinent safety, infection control, and quality improvement practices;	○	○	○	○	○	○
5. Discuss diagnosis and treatment of acute illness improvement practices; in the hospital setting; and	○	○	○	○	○	○
6. Review current data on diagnostic and therapeutic modalities for common inpatient problems.	○	○	○	○	○	○
7. The test questions were clear and appropriate.	○	○	○	○	○	○
8. I am satisfied with customer service for the CE/CME program.	○	○	○	○	○	○
9. I detected no commercial bias in this activity.	○	○	○	○	○	○
10. This activity reaffirmed my clinical practice.	○	○	○	○	○	○
11. This activity has changed my clinical practice.	○	○	○	○	○	○

If so, how? _____

12. How many minutes do you estimate it took you to complete this entire semester (6 issues) activity? Please include time for reading, reviewing, answering the questions, and comparing your answers to the correct ones listed. _____ minutes.

13. Do you have any general comments about the effectiveness of this CE/CME program?

_____

I have completed the requirements for this activity.

Name (printed) _____ **Signature** _____

Please make label address corrections here or PRINT address information to receive a certificate.

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