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Infective Endocarditis Trends and Outcomes

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Dr. Watkins reports that he has received research support from Allergan.

SYNOPSIS: Using large databases from New York and California, investigators found the overall incidence of infective endocarditis remained stable between 1998 and 2013, and 90-day mortality declined. Changes were noted in pathogen etiology and patient characteristics over time.

SOURCE: Toyoda N, Chikwe J, Itagaki S, et al. Trends in infective endocarditis in California and New York state, 1998-2013. *JAMA* 2017;317:1652-1660.

Infective endocarditis (IE) remains an uncommon yet serious illness. In 2007, a major change occurred in the IE prophylaxis guidelines, with the recommendation of fewer indications for prophylaxis. In light of this change, Toyoda et al used large databases to examine trends in the epidemiology and outcomes of IE between 1998 and 2013.

Patients for the study were identified using ICD-9 codes in statewide databases from New York and California. These databases included information on every hospital discharge, ambulatory surgery, and emergency room visit in their respective state. IE was characterized as native valve, prosthetic valve, cardiac device-related, or drug abuse-associated. Primary and secondary diagnostic codes were used to identify causal microorganisms, including *Staphylococcus aureus* (methicillin-resistant [MRSA] and methicillin-susceptible [MSSA]), other *Staphylococcus* species, *Streptococcus* species, gram-negative organisms, fungi, and unknown, which included both culture-negative cases and those without a code. During the study period, 75,829 cases of first-episode IE were identified, 56% in California and 43% in New York. The crude annual incidence increased from 7.6 to 9.3 cases per 100,000 persons. However, after adjustment for age, sex, and race, there was no significant increase in IE over time (range, 7.6 to 7.8 cases per 100,000 annually), and 90-day mortality decreased annually by approximately 2%. During the latter part of the study, those diagnosed with IE tended to be older, more likely to be male, and more likely to have chronic obstructive pulmonary disease, cancer, or liver disease. Drug use-associated IE increased over the study period

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by 0.9% annually (95% confidence interval [CI], 0.4-1.3). There was a substantial increase in hemodialysis patients diagnosed with IE, from 14.9% to 17.9% and representing 35.0% of healthcare-associated cases of IE between 2010 and 2013.The proportion of patients with a history of valve surgery increased from 12.8% to 15.2%, and the proportion with implantable cardiac devices increased from 8.8% to 15.6%.

Overall, these trends resulted in a decreased proportion of patients with native-valve IE at the end of the study (74.5% to 68.4%) and an increased proportion with prosthetic-valve IE (12.0% to 13.8%) and device-related IE (1.3% to 4.1%). The proportion of healthcare-associated IE increased from 49.8% in 1998 to 51.2% by 2013. The standard incidence of *S. aureus* IE increased during the study period from 2.1 (95% CI, 2.0-2.2) to 2.7 (95% CI, 2.6-2.9) cases per 100,000 people annually, with MRSA increasing from 0.23 (95% CI, 0.19-0.27) to 1.13 (95% CI, 1.05-1.22). The incidence of oral streptococcal IE decreased from 0.84 (95% CI, 0.76-0.92) to 0.73 (95% CI, 0.67-0.80) cases per 100,000 people annually and there was not an increase after the prophylaxis guidelines were changed. More patients underwent cardiac surgery during or within 30 days of their index admission over the study period (10.6% to 13.3%). Finally, healthcare-associated IE was associated with greater mortality compared to community-onset IE (adjusted hazard ratio [aHR], 1.52; 95% CI, 1.48-1.56), and compared to streptococcal IE, mortality was greater with gram-negative IE (aHR, 1.22; 95% CI, 1.16-1.28), staphylococcal IE (aHR, 1.38; 95% CI, 1.34-1.42), and highest with fungal IE (aHR, 1.84; 95% CI, 1.72-1.99).

■ COMMENTARY

This is an important study for several reasons: the large number of patients that were included, the time frame studied, which ranged from before and after the guidelines changed for IE prophylaxis, and the findings about how IE has evolved in recent years. Indeed, there is reason for both optimism and concern. While the overall incidence of IE remained stable during the study period, it is encouraging that 90-day mortality decreased, perhaps as a result of improvements in diagnosis and management, such as earlier valve replacement.

There was a 38% increase in patients with IE who were hemodialysis-dependent, which could be interpreted as either hemodialysis patients have a higher risk for IE, or perhaps there are more hemodialysis patients now in the general population. Although further investigation about the risks associated with hemodialysis and IE is needed, clinicians currently caring for hemodialysis patients need to be vigilant and maintain a high index of suspicion for IE, especially in the setting of a bloodstream infection.

Regarding the decrease in oral streptococcal IE, this trend could mean more people are receiving better dental care. That no increase in IE cases was observed after 2007 lends further support to the current IE prophylaxis recommendations. Drug use-associated IE increased over the study period, which serves as an important reminder about how the ongoing epidemic of intravenous drug abuse (especially heroin) is a major and very costly problem for society. Thus, one could argue from a purely economic standpoint that more resources (e.g., needle-exchange programs and funding for substance-abuse programs) should be devoted to this issue. The increase in MRSA IE is not surprising, given the higher incidence of community-associated infections that began in the 1990s. What is unexpected given the high virulence of MRSA is that the overall mortality of IE declined during the study period. Perhaps the increase in MRSA cases was balanced by the decline of oral streptococcal ones.

As with all retrospective studies that use large databases, there is a chance that misclassification errors and unrecognized confounding variables affected the results. The IE data from New York and California might not be representative of other regions of the country, e.g., the Southern and Midwestern states. Moreover, the organisms were identified by ICD-9 codes and were assumed to be the causative pathogens, which may not have been the case. The data are already four years old and may not accurately reflect the current characteristics of IE. For example, there is evidence that the incidence of healthcare-associated MRSA infections is decreasing.[1] Finally, the investigators were not able to identify cases of IE acquired in skilled care facilities, which could have led to an underestimate of healthcare-associated cases.

The study by Toyoda et al presents a lot of interesting data that can serve as a starting point for many future investigations. IE is an important and dynamic disease whose trends must be monitored continuously to achieve optimal patient outcomes. ■

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Ready for Dengue in the United States?

By Julie L. Hanson, MD, and Philip R. Fischer, MD

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Dr. Hanson and Dr. Fischer report no financial relationships relevant to this field of study.

SYNOPSIS: Dengue is increasingly recognized in the southern United States. When recently surveyed, however, clinicians in Texas seemed incompletely prepared to understand and manage patients with dengue.

SOURCE: Adam JK, Abeyta R, Smith B, et al. Clinician survey to determine knowledge of dengue and clinical management practices, Texas, 2014. *Am J Trop Med Hyg* 2017;96:708-714.

The vast majority of dengue cases in the United States have been imported from endemic regions by U.S. travelers. However, in recent years there has been increasing evidence of autochthonous dengue transmission in Texas, Florida, and Hawaii. The southern border of Texas is at particular risk of recurrent dengue outbreaks given the close proximity to Mexican states with relatively frequent dengue epidemics. Given this continual threat of dengue outbreaks at the Texas-Mexico border, Adam and eight co-authors from the Centers for Disease Control and Prevention (CDC) and Texas Department of State Health Services developed strategies to reduce morbidity and mortality associated with local dengue infections. As part of this effort, they developed a clinician survey to further understand local Texas clinicians' knowledge of dengue presentation and clinical management.

The clinician survey was sent to 2,375 physicians, physician extenders, and physician assistants in south Texas and the Houston metropolitan area. Two hundred seventeen clinicians (9%) fully completed the survey and were practicing within the catchment area. The clinician group was stratified further by specialty, practice site (i.e., inpatient, outpatient, or acute care), years of practice, and number of dengue cases diagnosed in their career. The survey questions focused on three knowledge areas, including dengue prevention and anticipatory guidance, clinical presentation and course, and clinical management. All clinician sub-divisions and categories of experience with dengue displayed a deficiency in knowledge of dengue. Approximately half (56%) of participants were able to identify all clinical signs of dengue and could identify indicators of early shock. However, < 1% recognized all warning signs for severe dengue. Fifty-five percent of clinicians recognized that intravenous crystalloids should be the primary initial fluid replacement in patients with elevated hematocrit, but only 7% correctly identified all three indications for crystalloid use. A minority of clinicians (19%) correctly identified situations in which patients with suspected dengue should return for re-evaluation after discharge. This survey highlights the need for improved dengue education in the United States and, in particular, in regions at risk for continued dengue outbreaks.

■ COMMENTARY

As the continued geographic spread of dengue virus threatens the southern United States, it is helpful to review the basics of dengue virus presentation, management, and prevention. Returning to dengue disease basics will assist in closing knowledge gaps as displayed by the clinician survey conducted in Texas.

Epidemiology

Aedes aegypti and *Aedes albopictus*, the mosquitoes that serve as vectors for the four dengue virus types (DENV-1 to -4), have been able to reach subtropical and temperate regions, including North America. Dengue is thought to have been present in the United States since the end of the 18th century, with frequent dengue epidemics occurring up until the mid-1900s. After 1945, there was a relatively quiescent period of dengue cases in the United States until 1980 when an autochthonous case of DENV-1 was identified in south Texas. Since 1980, surveillance studies have reported several additional cases of dengue in southern Texas associated with epidemics in northern Mexico, including outbreaks in 1999, 2005, and 2013. During the most recent outbreak in 2013, 53 cases of dengue virus were identified, with 49% of

these patients acquiring the infection locally.[1] In addition to cases in southern Texas, an epidemic occurred on the island of Maui, Hawaii, in 2001, and the first autochthonous case of dengue in 75 years was identified in Florida in 2009.[2] Dengue also has been reported in several U.S. territories, with the largest number of cases reported in the U.S. Virgin Islands and Puerto Rico. While autochthonous dengue infections have been limited thus far to these three states and two U.S. territories, both dengue vector species are distributed widely throughout the southern parts of the United States, indicating that a larger portion of the continental United States may be at risk for dengue infection in the future.

Clinical Presentation

Infection by dengue viruses can lead to a wide variety of clinical presentations ranging from a mild, influenza-like illness to hypovolemic shock and death. This spectrum of dengue infection has been subdivided into three primary syndromes, including classic dengue fever (DF), dengue hemorrhagic fever (DHF), and dengue shock syndrome (DSS). Classic dengue fever, or "break bone fever," is characterized by onset of a high fever, headache, rash, myalgias, and arthralgias three to 10 days after sustaining a bite from an infected mosquito. As the fever begins to subside three to seven days after symptom onset, the patient may have complete resolution of symptoms, or go on to develop dengue hemorrhagic fever. DHF is defined by four characteristics: recent history of fever, any hemorrhagic manifestation, thrombocytopenia, and evidence of increased vascular permeability. The most common hemorrhagic manifestations are petechiae, a positive tourniquet test, and gingival bleeding. Increased vascular permeability can lead to an elevated hematocrit, presence of pleural effusion or ascites, or hypoalbuminemia. Cases of dengue shock syndrome meet the four criteria for DHF, but also show signs of circulatory failure, such as a rapid, weak pulse, narrow pulse pressure, or hypotension. The risk of progression to DHF or DSS is increased in secondary infections when the individual has been infected previously by a different virus serotype. The fatality rate of patients with DSS can be 10% or higher without proper recognition and management, but mortality can be decreased to < 1% with appropriate intervention.[3]

Diagnosis

Efficient and accurate diagnosis of dengue is important for prompt clinical care, disease surveillance, and outbreak control. In endemic regions, clinical diagnosis of dengue usually is sufficient, but laboratory testing can be useful when the diagnosis is uncertain or in regions where dengue is sporadic. Dengue can be diagnosed by isolation of the virus, molecular methods, or serologic studies. Virus isolation is the traditional diagnostic method for detecting dengue virus infection; however, it has been replaced by RT-PCR tests and the NS1 antigen ELISA. RT-PCR testing allows for viral identification from the onset of the illness and is fast, sensitive, and specific. Unfortunately, PCR-based testing requires specialized equipment and staff that may not be feasible in a resource-poor region.

In contrast, the NS1 ELISA has emerged as a simple, low-cost diagnostic tool to detect dengue virus in the early stage of infection. The qualitative NS1 ELISA has become the standard for dengue diagnosis worldwide, and quantitative NS1 ELISA continues to be researched as initial studies suggest a direct correlation between NS1 levels and the risk of progression to severe disease. Serological studies also are important tools in diagnosing dengue infection, especially outside of the acute phase. IgM antibodies typically are detectable three to five days after illness onset and peak several weeks after recovery. IgG is usually not present in the acute phase in a primary infection, but may appear as early as three days after illness onset in a secondary infection. The ratio of IgM and IgG in the acute phase of disease may provide an indication as to whether it is a primary or secondary infection. IgM and IgG serologies are susceptible to cross-reactivity with other flaviviruses, which can provide a diagnostic challenge in areas of the world where more than one flavivirus is circulating. Overall, no single assay can be used to definitively diagnose dengue at all stages of infection. Therefore, a combination of NS1 testing and IgM/IgG serologies is recommended to maximize disease detection. Several diagnostic kits, including rapid point-of-care devices, use this combination with nearly 100% detection sensitivity from disease onset through recovery.[4]

Treatment

While there are a wide variety of clinical manifestations of dengue, treatment is relatively simple and generally effective in reducing the morbidity and mortality associated with infection. Patients in the early febrile phase generally can be managed safely as outpatients with adequate follow-up and anticipatory guidance. Warning signs of severe disease that should prompt return for care include abdominal pain, persistent vomiting, signs of bleeding, and change in mental status. Acetaminophen is safe for symptomatic management of the febrile patient, but nonsteroidal anti-inflammatory drugs should be avoided given the increased risk of bleeding complications. If the patient begins to show warning signs suggestive of more significant disease, or if there are coexisting risk factors, the patient should be hospitalized for further management. Initial treatment focuses on fluid resuscitation with the use of isotonic crystalloids, as well as monitoring of hematocrit and signs of plasma leakage. Patients who present with signs of shock, severe hemorrhage, or severe organ impairment should be hospitalized in a facility with access to intensive care services and blood products. Isotonic crystalloid infusion is still the most effective intervention for patients with severe den-

gue; however, blood products and more intensive hemodynamic and electrolyte monitoring may be required.[5]

Prevention and Control

Dengue prevention and control methods have been focused largely on vector management as well as the development of dengue-specific vaccines. Vector control efforts include the reduction of mosquito breeding sites, application of insecticides to high-yield targets (e.g., bed nets, window curtains, school uniforms), use of bacteria or fungi to decrease mosquito survivability, and genetic modification of wild mosquito populations to reduce transmission. Vector control is particularly challenging given the varying efficacies and costs of mosquito control measures, but efforts such as the World Health Organization's (WHO) Integrated Vector Management strategy seek cost-effective, efficacious, and ecologically appropriate solutions to vector control.[6]

A considerable amount of research has been devoted to the development of a dengue vaccine. The live attenuated tetravalent vaccine, CYD-TDV, has been registered in several countries. CYD-TDV is indicated for individuals 9-60 years of age who are living in an area with endemic dengue. Mathematical models have been carried out that demonstrate the possible effect of the CYD-TDV vaccine over time. The greatest effect of vaccination is in settings with high transmission intensity (seroprevalence > 70% at 9 years) where the reduction in symptomatic and hospitalized dengue ranged from 10-30% over a 30-year period. In contrast, the models predicted an increase in dengue hospitalization rates in very low transmission intensity settings (seroprevalence 10% at 9 years). This finding suggests that the vaccine may act like an asymptomatic infection in a previously seronegative individual, setting up the individual for a secondary-like infection if he or she is exposed to the dengue virus. Therefore, the WHO takes a stance that the vaccine should be considered for regions with seroprevalence > 70%, but it is not recommended for regions with seroprevalence < 50%.[7] The CYD-TDV vaccine, as well as other vaccines in development, will continue to be researched to provide an efficacious and safe method for dengue prevention. ■

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Encephalitis Due to Powassan Virus

By Stan Deresinski, MD, FACP, FIDSA

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

SYNOPSIS: Powassan virus is transmitted by the same tick that carries the etiologic agent of Lyme disease and several other pathogens. The number of cases of encephalitis caused by this virus may be increasing in the endemic areas.

SOURCES: Tutolo JW, Staples JE, Sosa L, Bennett N. Notes from the field: Powassan virus disease in an infant — Connecticut, 2016. *MMWR Morb Mortal Wkly Rep* 2017;66:408-409.

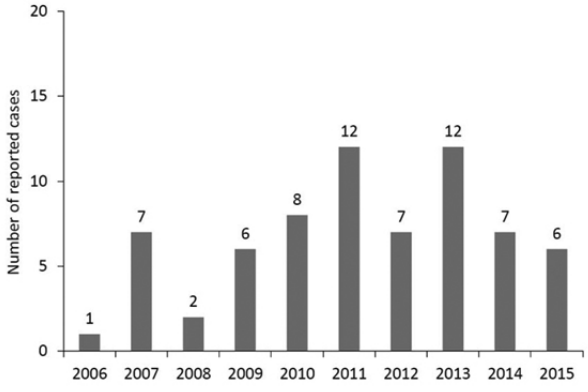
Doughty CT, Yawetz S, Lyons J. Emerging causes of arbovirus encephalitis in North America: Powassan, chikungunya, and Zika viruses. *Curr Neurol Neurosci Rep* 2017;17:12.

A 5-month-old infant living in eastern Connecticut was admitted to a hospital in November 2016 for evaluation and management of seizures that occurred a few days after onset of fever and vomiting. The history obtained at the time indicated that, two weeks earlier, the infant had been bitten by a tick, which was likely to have been attached for less than three hours before removal. CT of the brain was normal, and a lumbar puncture was performed; the cerebrospinal fluid (CSF) white blood cell (WBC) count was 125 cells/μL, with 81% lymphocytes.

Figure 1: Powassan Virus Neuroinvasive Disease Cases Reported by State, 2006-2015

Source: ArboNET, Arboviral Diseases Branch, Centers for Disease Control and Prevention.

Figure 2: Powassan Virus Neuroinvasive Disease Cases Reported by Year, 2006-2015



Source: ArboNET, Arboviral Diseases Branch, Centers for Disease Control and Prevention.

A symmetric pattern of restricted diffusion involving the basal ganglia, rostral thalami, and left pulvinar was detected on magnetic resonance imaging (MRI). Bacterial cultures of CSF yielded no growth, and testing failed to detect evidence of infection with a number of arboviruses. Respiratory viral cultures also were negative.

An infectious diseases consultant requested that CDC test for evidence of Powassan virus infection, and IgM antibody directed against this virus was detected in a CSF sample obtained four days after the onset of illness. In addition, the antibody neutralizing titer was 1:32. Seizure control was achieved, and the patient was discharged on anticonvulsant therapy after seven days. At age 10 months, he was no longer receiving anticonvulsants and was reported to have normal motor and verbal development. MRI, however, revealed gliosis with encephalomalacia in both thalami and basal ganglia, along with volume loss and early mineralization in the left basal ganglia.

■ COMMENTARY

Powassan virus is a tick-borne flavivirus that was first identified in 1958 in Powassan, Ontario, in the brain of a 5-year-old boy who died with encephalitis. Powassan virus and the tick-borne encephalitis virus belong to the tick-borne serocomplex, which causes encephalitis in parts of Eastern Europe, far eastern Russia, and Asia, in addition to North America. This complex is distinct from the mosquito-borne serocomplex, members of which include dengue virus, West Nile virus, Japanese encephalitis virus, and St. Louis encephalitis virus. Cases in the United States occur in the Great Lakes region and the Northeast (*see Figure 1*); although the virus has been identified previously in ticks in Connecticut, the case summarized above is the first human case reported.

Powassan virus is known to be transmitted by at least three distinct tick species: *Ixodes marxi*, *Ixodes cookei*, and *Ixodes scapularis*, the last of which also transmits *Borrelia burgdorferi*, *Borrelia miyamotoi*, *Anaplasma phagocytophilum*, and *Babesia microti*. There are two lineages of Powassan virus. Lineage 1, which includes the prototype virus identified in 1958, is transmitted by *I. cookei*, with skunks and groundhogs as the main reservoirs. Lineage 2 virus ("deer tick virus") is transmitted by *I. scapularis* and is maintained in the white-footed mouse.

Recognized infection is uncommon, with a median of seven cases reported each year from 2006 through 2015 in the United States (*see Figure 2*), although it has been suggested that the number of cases may be increasing, a conjecture that is consistent with a marked increase in seropositivity in deer in New England in recent decades. Approximately 3% of ticks examined in New York state are infected with Powassan virus. The virus is transmitted rapidly, requiring tick attachment for only 15 minutes and, as a consequence, one-half of infected patients remain unaware of having suffered a tick bite. In the case reviewed here, it is believed that the implicated tick likely was brought into the house on someone's clothing.

Most human Powassan virus infections are likely asymptomatic. Initial symptoms consist of fever and headache and, ominously, altered mental status. One-half have gastrointestinal symptoms and one-third have rash. Encephalitis, in some cases, may be delayed. In addition to encephalitis, meningitis, myelitis, or radiculitis is reported.

Examination of CSF demonstrates findings suggestive of a viral encephalitis, although occasional neutrophil predominance is seen early in the infection. T2 hyperintense lesions without enhancement commonly are seen on MRI and, as in the case reviewed here, diffusion restriction may be observed. Although lesions may occur elsewhere, involvement of the basal ganglia is

common, as is the case with other flavivirus encephalitides such as that due to Japanese B encephalitis virus. As with West Nile virus, PCR testing is insensitive and the diagnosis is best made by detection of IgM antibody, preferably in CSF, with confirmation using a plaque reduction neutralization test to deal with potential cross-reactivity with other flaviviruses.

There is no specific treatment available, and an estimated 10-15% of patients with encephalitis die, while approximately one-half of survivors suffer residual neurological deficits. Prevention consists of avoiding tick bites, which, in brief, consists of wearing long pants and long-sleeved shirts, using effective tick repellent, performing effective screening, and performing tick checks. A more detailed set of preventive measures is available at the CDC website.[3] ■

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ECG Review: Are There Definitive Clues?

By Ken Grauer, MD

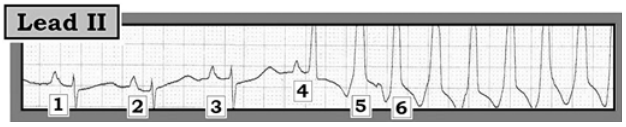
Dr. Grauer, Professor Emeritus in Family Medicine, College of Medicine, University of Florida.

Dr. Grauer is the sole proprietor of KG-EKG Press, and publisher of an ECG pocket brain book.

The lead II rhythm strip shown in the figure below begins with three sinus-conducted beats. There follows a run of a wide complex tachycardia (WCT). How certain are you that the run of WCT that begins with beat #4 is ventricular tachycardia (VT)?

The first three beats in the figure are sinus-conducted. The PR interval is upper normal at 0.20 seconds. The P-P interval changes slightly, which means there is underlying sinus arrhythmia. QRS morphology then abruptly changes beginning with beat #4. The QRS widens and is oppositely directed (all positive) compared to the narrow rS complexes of the first three beats. There is no reason for aberrant conduction to occur beginning with beat #4 because beat #4 occurs late in the cycle, at a time by which conduction properties that lead to aberrancy should have resolved. Instead, we can say with 100% certainty that the run of wide beats beginning with beat #4 is VT.

The first principle is that abrupt onset of a regular (or at least fairly regular) wide rhythm of different morphology than sinus-conducted beats predicts VT with > 90% likelihood. Consideration of clinical details (i.e., history of underlying heart disease and/or prior documented VT episodes), together with morphologic ECG features, often can increase certainty of our diagnosis beyond this level.



Beat #4 is a fusion beat. Note that the PR interval preceding beat #4 is shorter than the PR interval preceding each of the three sinus-conducted beats. This means that something else must have happened to produce the oppositely directed upright QRS complex of beat #4 because the on-time sinus P wave preceding beat #4 simply did not have enough time to complete its conduction through the ventricles.

Fusion beats manifest QRS and ST-T wave morphology intermediate between the QRS and ST-T wave morphology of sinus-conducted beats and ventricular beats. Depending on how deep in the ventricles the sinus P wave is able to penetrate, the resulting QRS and ST-T wave will look more like sinus beats or ventricular beats. Beat #4 is upright like the wide run that follows, but this beat is not quite as wide, nor is its negative T wave as deep because there is fusion (simultaneous occurrence) of supraventricular and ventricular activation.

AV dissociation also appears on this tracing, at least at the beginning of the run of wide beats. The P wave preceding beat #4 is on time. Note that another on-time P wave appears to notch the ST-T wave just after beat #5. But since these P waves do not conduct normally, there is AV dissociation. The abrupt onset of a different wide run with fusion beats and AV dissociation provides indisputable proof that the rhythm in the Figure is VT.

For more on this case, please visit: http://bit.ly/2mbW9Ah. ■

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CME QUESTIONS

1. Based on the study by Toyoda and colleagues of the changes in infective endocarditis from 1998 to 2013, which of the following statements is true?

a. The overall incidence of infective endocarditis has remained the same.

b. There was an increase in the proportion of cases of prosthetic-valve endocarditis.

c. The 90-day mortality has decreased.

d. The incidence of MRSA endocarditis has increased.

e. All of the above.

2. In patients infected with a dengue virus, which of the following criteria is not one of the four defining criteria of progression to dengue hemorrhagic fever (DHF):

a. Recent history of fever

b. Any hemorrhagic manifestation

c. Thrombocytopenia

d. Leukocytosis

e. Evidence of increased vascular permeability

3. In patients with a wide complex tachycardia, the presence of fusion beats and AV dissociation identifies the rhythm as:

a. Ventricular tachycardia

b. Atrial fibrillation with aberrancy

c. AV nodal re-entrant tachycardia

d. Wolff-Parkinson-White syndrome

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.

[IN FUTURE ISSUES]

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