How to Write the Cause of Death Statement in the Death Certificate for Natural Causes
By Lei Shao, MD

The death certificate serves two basic purposes: it documents the fact of death for legal and other uses and it provides data for vital statistics and public health policy. A cause of death statement consists of two parts. **Part I** is a series of lines in which causes of death can be entered. The first line is the immediate cause of death followed by intermediate causes. The underlying cause of death is the last line in the part I. **Part II** is a list of significant conditions, preexisting or coexisting, that contributed to death. Part II is used only when it is applicable and the most important contributory condition should be list first.

The **underlying cause** of death is the disease or condition that started the train of morbid events leading to death. It is the disease or condition that occurred first in time and that initiated and was ultimately responsible for any subsequent diseases, condition or complications that resulted in death. It is “the bottom line” for the death. The underlying cause of death should be as etiologically specific as possible.

In neonatal and infant deaths, the underlying causes of death usually fall into one of the following categories:
- Congenital and genetic disorders (e.g., trisomy 18, hemophilia, DiGeorge syndrome)
- Placental/amniotic disorders (e.g., chorioamnionitis, placental abruption)
- Maternal-fetal disorders (e.g., hemolytic disease of the newborn)
- Maternal disorders (genital herpes infection, diabetes, pre-eclampsia, drug toxification)
- Complications of labor and delivery (peripartum asphyxia, trauma during delivery)

The **immediate cause** of death is the final disease or complication that results from the underlying cause of death. The **intermediate cause** of death, if present, is a single or multiple diseases or complications that occur between the underlying and the immediate cause of death.

Mechanistic terminal event, such as respiratory failure, cardiac arrest, asystole, ventricular fibrillation, have almost limitless differential diagnosis. They are the terminal pathophysiologic or biochemical derangements that are common final pathways to deaths. They explain how a cause of death exerts its lethal effect. In general, mechanistic terminal events should not be used in the cause of death statements because they are extremely nonspecific and are of little value for mortality statistics.

**Case examples**

Case 1: A two-day old premie born at 28 weeks of gestation developed severe respiration distress and was diagnosed of hyaline membrane disease. Echocardiograph revealed a ventricular septal defect. He was on a ventilator, developed pneumothorax and died of respiratory failure. The pathologic examination of the placenta revealed acute chorioamnionitis.

**Part 1**
A. Peritherapeutic ventilator-induced pneumothorax
B. Hyaline membrane disease
C. Prematurity
D. Acute chorioamnionitis

**Part II**
Congenital ventricular septal defect

Case 2: A five-year-old girl with history of acute lymphoblastic leukemia, CNS relapse and allogenic bone marrow transplant. She was admitted for elevated LFT and a skin rash. Biopsy of the skin was consistent with GVHD. She later had a second marrow transplant. Three weeks after BMT, she had ascites, hepatosplenomegaly and markedly elevated LFT. Veno-occlusive disease was suspected. She later developed hepatic encephalopathy. The family decided to withdraw life support. She died of cardiac arrest.

**Part I**
A. Peritherapeutic ventilator-induced pneumothorax
B. Hyaline membrane disease
C. Prematurity
D. Acute chorioamnionitis

**Part II**
Congenital ventricular septal defect

There is often more than one acceptable way to write a cause of death statement. A good rule is to include significant information to tell a story about the sequence of diseases (conditions) and complications leading to deaths. The statement represents your opinion based on the best of your knowledge. If your opinion should change, the death certificate can be changed at a later time.

You can contact Dr. Shao at X 3234 to obtain a copy of the protocols for writing cause of death statement by College of American Pathologists.
Increase in Local Adenoviral Activity.
By Rangaraj Selvarangan PhD

Adenovirus is associated with many clinical syndromes including upper and lower respiratory tract infection, pertussis-like syndrome, pharyngoconjunctival fever, acute respiratory distress, conjunctivitis, cystitis, and gastroenteritis in children. Respiratory disease is the most common form of infection with increased incidence during late winter, spring and early summer. Infection is more common between 6 months and 5 years of age. Nosocomial transmission of the adenovirus in hospitalized children may account for up to 10% of pneumonia cases. Incidence of adenoviral infections in solid organ and bone marrow transplant patients varies from 3 to 20% and is usually associated with high mortality. Laboratory diagnosis of adenoviral infection is accomplished by cell culture of respiratory, conjunctival and stool specimens among others. The number of adenovirus isolated by viral cell culture has increased in the last year. The CMH virology laboratory has isolated 36 isolates in 2002, 26 isolates in 2003, 60 isolates in 2004 and 21 isolates in the first two months of 2005. Majority of the isolates were obtained from respiratory specimens.

Adenovirus is a DNA virus, classified in to six subgenera (A-F) based on hemagglutination properties. Among the 51 serotypes described the most common types are 1 through 8, 11, 21, 35, 37 and 40. Usually specific serotypes are associated with certain clinical syndromes. Adenovirus serotypes 3, 7 and 21 have caused severe disease in children. Two possibly hyper virulent genotypes of adenovirus (Ad7d2 and Ad7h) emerged in 1990 and have been associated with severe disease and have recently been found in US populations. These strains have been previously reported from epidemics in pediatric populations in South America and associated with increased disease severity and mortality. Community wide outbreaks of Ad7 have been well documented in crowded institutions such as day care centers, schools, hospitals and military quarters. Epidemics of Ad7 reported in US since 1996 have been primarily due to Ad7d2. All civilian Ad7d2 epidemics have occurred among institutionalized children.

Our laboratory, 1 of 15 in United States that collaborates with University of Iowa in a nation wide surveillance study to characterize the emergence of adenovirus through out the United States. Seventeen adenovirus isolates from 2004 season of CMH have been genotyped by the Iowa laboratory as follows: 8 Ad3, 3 Ad12, 3 Ad4, 2 Ad1 and 1 Ad6. On few occasions more than one virus isolate have been obtained from different sites of the same patient; three Ad4 isolates were obtained from eye, respiratory and stool specimens from one patient, while three Ad12 isolates were obtained from stool and urine from one patient, and two Ad3 isolates were obtained from a nasal and stool sample from a third patient. Many of the adenoviral infections are subclinical and prolonged viral excretion can occur in asymptomatic individuals. While a respiratory or conjunctival isolate is more suggestive of recent infection, a stool isolate may indicate a prolonged carriage or recent infection. Hence, significance of an adenovirus isolate is based on the specimen type from which the virus was isolated and the clinical picture of the patient.

Comparison of Respiratory Viral Season 2004/2005

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