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Editor's Welcome

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Welcome to the first issue of the Kansas Journal of Medicine (KJM). We have developed this Journal for several reasons. First, we are engaging resident physicians in the art of publishing manuscripts. My editorial board has spent many hours reviewing and recommending manuscript changes. Our first issue focuses on some of our own department's research activities, but future issues will include submissions from other departments at the University of Kansas Medical Center and beyond as our editorial board grows and broadens its expertise. Second, we hope physicians practicing in Kansas will access the Journal for articles that enhance the delivery of patient care. We encourage submission of relevant manuscripts from practicing physicians. Also, readers may recommend topics for articles written by invited authors. Topics for recommended articles may be submitted to the Kansas Journal of Medicine by email to the Managing Editor at kkallail@kumc.edu. Finally, we expect KJM to stimulate translational research opportunities for physicians, resident physicians, medical students, and other health care researchers. We hope you enjoy this new online journal for and by Kansas physicians.

Educating General Internists for Kansas

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Abstract

Background. The demand for primary care is increasing, yet fewer medical school graduates are entering primary care residencies and fewer residency graduates are targeting their careers toward primary care. The University of Kansas School of Medicine-Wichita (KUSM-W) was created specifically to enrich primary health care in the state. This project examined the impact of the Department of Internal Medicine's postgraduate education program on the supply of generalist physicians to Kansas.

Methods. Residency match data over 19 years were analyzed to determine the number of KUSM-W medical students who matched in internal medicine. Practice setting data for internal medicine and internal medicine/pediatrics residency graduates over 16 years were analyzed to determine type and location of practice.

Results. Over 19 years, an average of 20% of KUSM-W medical students matched to an internal medicine residency. A downward trend toward an internal medicine residency was noted over time. Over 16 years, 54% of residency graduates entered primary care practice with the majority staying in Kansas. Twelve percent entered primary care practice in rural communities under 20,000 population.

Conclusions. KUSM-W provides a valuable service by graduating a respectable proportion of medical students to internal medicine residencies and a large proportion of residents to primary care practice. Many of the issues that attract students and residents to primary care, however, are not under the direct influence of the medical education system. Changes in health policy, particularly reimbursement and health care delivery issues, will have more impact on primary care workforce than changes in medical education. *KJM 2007; 1(1):2-6.*

Introduction

Primary care in the US is on death row declared a recent editorial.[1] An American College of Physicians report stated that primary care is at grave risk of collapse.[2] The demand for primary care is increasing, yet fewer medical school graduates are entering primary care residencies and fewer residency graduates are targeting their careers toward primary care.[1,2] The proportion of physicians seeking sub-specialty training has increased.[3] Further, the attrition rate of primary care physicians due to career options and retirement is expected to increase. Money and lifestyle are the main culprits behind the decline in primary care.

National data on third-year internal medicine residents' career choices showed the trend away from primary care.[4] Since 2000, the percentage of graduates entering generalist practices declined by half from over 40% to about 20%. Over half of the graduates selected sub-specialty careers. The number entering hospital medicine practices has grown to over 10%.

Shortages of primary care physicians in Kansas are long-standing problems. As of the latest state report[5], 82 of 105 Kansas counties were designated, wholly or partially, as federal Primary Medical Care Health Professional Shortage Areas. In addition, 53 of 105 counties are designated

by state definitions as medically underserved. Finally, 90 counties are eligible for federally-designated Rural Health Clinics, an effort to increase recruitment of physicians to medically underserved areas. In total, 99 of 105 Kansas counties are designated, wholly or partially, by one of the various definitions as medically underserved for primary care.

The University of Kansas School of Medicine-Wichita (KUSM-W) was created specifically to enrich primary health care in the state. KUSM-W is a community-based medical school whose primary mission is to train physicians for the state of Kansas. The purpose of this project was to examine the impact of the Department of Internal Medicine's postgraduate education program on the supply of generalist physicians to Kansas.

Methods

Residency match data from the KUSM-W Office of Academic and Student Affairs was compiled on graduating medical students for the years 1988 through 2006. In addition, graduate data for the Department of Internal Medicine residency program for the years 1991 through 2006 were obtained from departmental records. Graduates from the Internal Medicine/Pediatrics (Med/Peds) combined program were reviewed beginning in 1998 through 2006. The first Med/Peds graduation occurred in 1998. The initial practice location following residency was reviewed to determine the geographic setting and community size. In addition, the type of initial practice was assessed.

US Census Bureau data were used to determine community population and rural status.[6] Data from the 1990 and 2000 census were used as appropriate. Rural status was defined as a non-metropolitan statistical area with a population less than 20,000. A primary care practice was defined as a generalist practice with continuity of care. Practices specifically excluded from the primary care category included emergency medicine, occupational medicine, hospital medicine, and fellowship training.

Results

From 1988-2006, an average of 20% of KUSM-W medical students matched to an internal medicine residency (regardless of location). The percentages ranged from a high of 30% in 1992 to a low of 11% in 1997. Figure 1 reveals a downward trend in the percent of students who matched to an internal medicine residency.

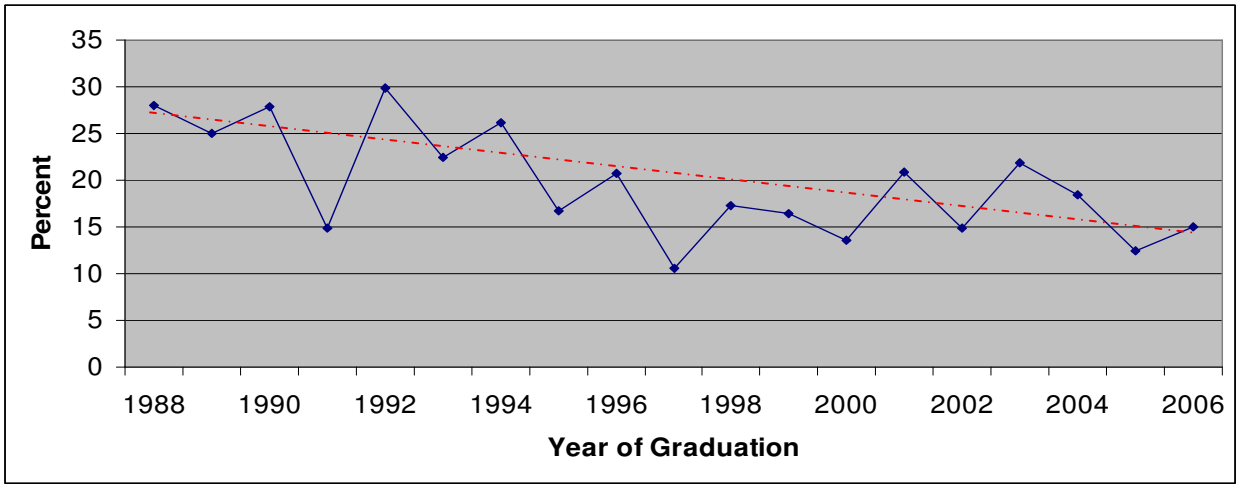

Figure 1. Percent of KUSM-W Medical Students Who Matched to Internal Medicine Programs by Year. (The red line is only an aid to visualize the decline over time.)

From 1991-2006, 169 individuals graduated from the KUSM-W residency program in either internal medicine (n = 155) or Med/Peds (n = 14). Table 1 shows the breakdown of graduates by practice setting. Of the 169 graduates, 92 (54%) entered primary care practice. Approximately one-third (35%) entered subspecialty fellowship training. The remainder (11%) practiced emergency medicine, occupational medicine, or hospital medicine.

Figure 2 shows the practice setting of the internal medicine residency graduates by year. Generally, the number of graduates entering primary care practice shadowed the total number until 2005. The number of graduates entering fellowship showed an upward trend since 2005 when more graduates entered a fellowship than primary care practice.

Internal medicine graduates who practiced primary care tended to stay in Kansas. About three-quarters of the graduates (78%) who practiced primary care stayed in Kansas. Of those who practiced in rural communities, 73% stayed in Kansas. A smaller percentage of Med/Peds graduates entered primary care practices than internal medicine graduates. More entered other practice settings, particularly in hospital medicine. Although the total number of Med/Peds graduates is small, all who entered primary care practice stayed in Kansas.

Table 1. Breakdown of KUSM-W Internal Medicine and Internal Medicine/Pediatrics Graduates by practice setting.

Practice Setting	Internal Medicine Residency n = 155 (%)	Internal Medicine/ Pediatrics Residency n = 14 (%)	Total n = 169 (%)
Primary Care Practice	88 (57)	4 (29)	92 (54)
Primary Care Practice in Kansas	69 (45)	4 (29)	73 (43)
Rural Primary Care	26 (17)	1 (7)	27 (16)
Rural Primary Care in Kansas	19 (12)	1 (7)	20 (12)
Fellowship program	57 (37)	2 (14)	59 (35)
Other Practice Settings	11 (7)	8 (57)	19 (11)

Discussion

The demand for general internists will increase by 38% by 2020.[2] Thirty-five percent of physicians nationwide likely will retire in the next ten years. Yet, changes in the health care system have made primary care less satisfying for physicians and less attractive for students and residents.[2,4,7] The KUSM-W Internal Medicine program is experiencing declining interest in primary care as are programs nationwide. However, the long-term view of the KUSM-W Internal Medicine program reveals that the decline is more recent and less severe than the national data suggest. The 16-year average for

residency graduates to enter a primary care practice is 57% with the majority staying in Kansas. The 2005 national data revealed that 20% of internal medicine residency graduates entered primary care. The comparable KUSM-W value was 40%. During the same year, 58% of graduates entered fellowship nationally. Only 50% of KUSM-W graduates entered fellowship that year. Certainly, these data revealed some success in preparing general internists for practice in Kansas.

Although the downward trend for KUSM-W medical students to enter an internal medicine residency is concerning, the 19-year average of 20% was near the national percentage of available positions. In 2006, 22% of all available residency positions were designated for internal medicine.[8] KUSM-W medical students, therefore, matched in internal medicine, on average, at about the same proportion of total residency positions available for internal medicine.

The decline in interest for primary care is multifactorial. The system for financing health care and the health care delivery system are problematic. Specialist income in 2004 was approximately double that of primary care physicians.[4] This gap is unlikely to narrow anytime soon. The reimbursement system favors procedural skills and quantity, rather than quality, of care.[4] Medicare, the largest purchaser of health care in the US, undervalues evaluation and management services.[2] Generalist physicians are frustrated by the growing demand for services without inadequate reimbursement.

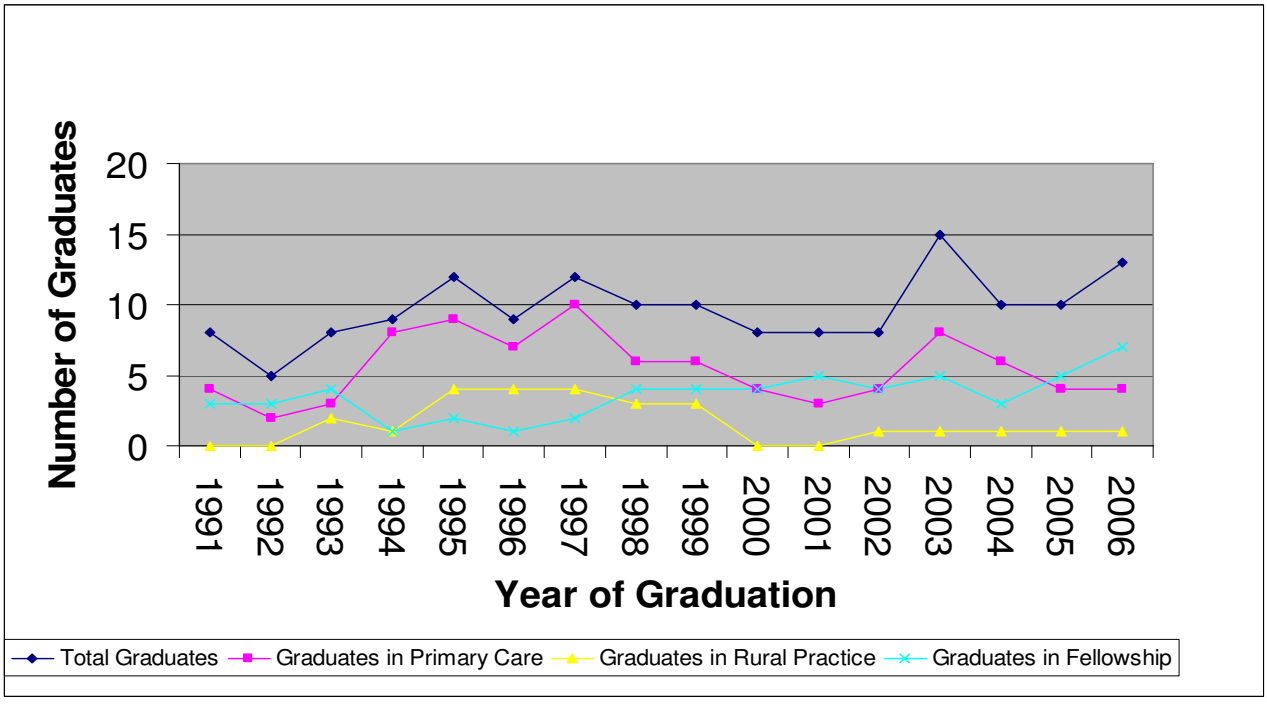

Figure 2. Number of KUSM-W Internal Medicine residency graduates over 16 years by practice setting.

Yet, the demand for primary care services is increasing. The increased prevalence of chronic disease and the aging of the population are factors driving the need for primary care.[4] Patients want a personal, comprehensive, and longitudinal

relationship with their doctors, especially within a fragmented health care system.[7] Primary care services reduce health care costs and improve quality of care.[2,7] Further, primary care services may result in more appropriate use of specialists who focus on their specific area of expertise.[2]

Educating general internists for Kansas is one responsibility of KUSM-W. Many of the issues that attract students and residents to primary care, however, are not under the direct influence of the medical education system. Changes in health policy, particularly reimbursement and health care delivery issues, will have more impact on primary care workforce than changes in medical education. Given the current health care environment, KUSM-W provides a valuable service by graduating a respectable proportion of medical students to internal medicine residencies and a large proportion of residents to primary care practice. Although the trends are concerning and must be addressed, the long-term outcomes have been positive.

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Keywords: medical education, graduate medical education, internal medicine, primary care, Kansas

Acute Middle Cerebral Artery Thrombosis

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Introduction

A CT of the head is commonly the first neuro-imaging test of acute stroke in the emergency department.[1] Usually, it may take up to 24 hours for signs of acute ischemia to be notable on CT.[2-3] "Dense MCA" or acute middle cerebral artery thrombosis is a valuable finding on noncontrast CT scan of the head when correlated with the appropriate clinical symptoms of acute stroke. A hyperdense artery sign of the middle cerebral artery (MCA) in the setting of acute cerebral infarction strongly indicates thrombo-embolic MCA occlusion.[4-5]

Case Report

A 59-year old women presented to the emergency department after falling in the bathroom due to left-sided weakness. Her family stated that she was asymptomatic earlier. On examination, she had right gaze preference, apraxia of eyelids opening, left cranial nerve palsy, flaccid left hemiplagia, and a left extensor plantar response.

A noncontrast CT scan of her head revealed a hyperdense tubular region in the proximal right middle cerebral artery (figure 1) consistent with acute thrombosis. Effacement of the right cerebral sulci also was present. Taken together, these findings were consistent with acute middle cerebral artery infarction.[1]

Discussion

Early goal therapy in patients with strokes is essential in identifying candidates for emergent therapy such as thrombolysis.[2] CT scan often is favored as a neuro-imaging diagnostic tool because of its widespread availability and rapid acquisition time.[3,6-7] CT is used widely in acute stroke to rule out any intracranial hemorrhage. However, changes due to brain tissue infarction usually take up to 24 hours to be seen on CT, thus it has limited power to detect any ischemic lesion early when emergent therapy could be beneficial.[3,8-9] Therefore, any early CT indicators of acute cerebral thrombosis have important value.[4-5,10-11]

The finding of increased density of the MCA main stem, or the hyperdense MCA sign, is highly suggestive of acute thrombosis when correlated with appropriate clinical findings.[4-5,11] This sign has been correlated angiographically with embolic or atherothrombotic MCA occlusion.[5,10-12] The hyperdensity is most likely due to either calcific or hemorrhagic components of the acute plaque. This sign is non-specific when it is present in isolation and not correlated with the clinical setting.[4-5,10] False-positive hyperdense MCAs have been noted in asymptomatic patients with high hematocrit or calcific atherosclerotic disease.[4,11-13]

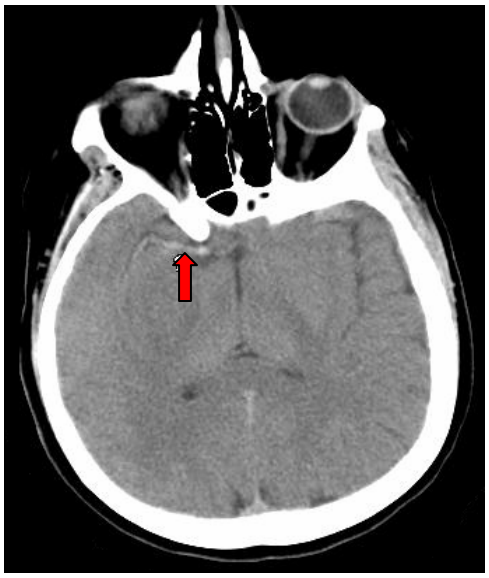

Figure. 1: Noncontrast brain CT scan showing a tubular hyperdense structure consistent with acute middle cerebral thrombosis (arrow).

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Keywords: middle cerebral artery thrombosis, case report

Tetanus: The Forgotten Disease

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Introduction

Tetanus was well known to the ancient physicians of Egypt and Greece, but since institution of the active immunization in 1940, it has become an old forgotten disease in developed countries.[1] Tetanus is a nervous system disorder characterized by prolonged contraction of the skeletal muscles. The disease is caused by tetanospasmin, a neurotoxin produced by the anaerobic bacterium *Clostridium tetani.*

Case Report

A 74-year-old Caucasian female presented to the emergency department complaining of trismus. The patient had not been evaluated by a physician for several years. Her symptoms started 10 days prior with difficulty opening her mouth, generalized muscle stiffness, and episodic muscle spasms. She denied fever and chills, was not taking any medications, and had no history of alcohol or illicit drug use. She did not remember ever receiving tetanus vaccination.

On physical examination, the patient was unable to open her mouth more than 1 cm and the masseter muscle was contracted. She had a generalized increase in muscle tone and episodic painful muscle spasms involving the entire body. Multiple scars in different healing stages over the lower extremities had been caused by scratches from her 13 cats.

The diagnosis of generalized tetanus was suspected. Five hundred units of human tetanus immunoglobulin were given intramuscularly and active immunization was started. Although there was no active wound infection, the patient was started on intravenous metronidazole. Muscle spasms and rigidity were controlled with a scheduled dose of intravenous diazepam. Over the next three weeks, the patient's symptoms gradually improved, and she was discharged to a skilled nursing facility.

Epidemiology

The incidence of tetanus in developed countries dropped dramatically since 1940 mainly due to the widespread use of active immunization.[1] Better hygiene and wound care also have played a major role. The annual incidence of tetanus in the US has fallen from 4 to 0.2 per million since 1947.[1] The decline in incidence was accompanied by a decrease in mortality from 50 to 30%.

Between 1998 and 2000, the annual incidence of tetanus was 0.16 cases/million representing an average of 43 cases per year.[2] The majority of cases were in patients older than 60 years, reflecting the waning of immunity with age or inadequate immunization of the elderly.[1] Injection drug abuse and the increase of non-immunized immigrants are responsible for most cases of tetanus in younger individuals aged 25 to 59. According to the CDC[2], 15% of cases of tetanus in the US occurred in intravenous drug users.

Acute injuries account for 70 to 80% of US cases; 23% of cases are associated with chronic decubitus ulcers, gangrene, burns and abcesses.[1] About 7% of cases have no apparent source. Neonatal tetanus is rare in the US.[1,2]

Tetanus is common in developing countries. The worldwide incidence is about 1,000,000 cases per year.[3,4] It is a major cause of death especially in neonates. Tetanus accounted for 300,000 deaths in 2000, including 200,000 neonatal deaths.[5]

Pathogenesis and Pathology

Clostridium tetani is an obligatory gram positive anaerobic bacillus. The bacteria are usually found in contaminated soil and animal gut in the sporiform phase. When these spores contaminate a wound, they germinate under anaerobic conditions, transform into a rod-shaped bacterium, and produce tetanospasmin. Tetanospasmin is an exotoxin produced by mature bacteria, then cleaved intracellularly into light and heavy chains before exocytosis. The heavy chain appears to mediate binding to cell surface and transport proteins, while the light chain is responsible for the clinical manifestations of tetanus.[1]

After its excretion, tetanospasmin gains entry into the central nervous system (CNS), both locally and distally, via the presynaptic terminals of lower motor neurons. Tetanospasmin is transported intra-axonally in a retrograde fashion to the cell body, the ventral horns of the spinal cord, and to the motor nuclei of the cranial nerves where it exerts its major pathogenic action. After reaching the spinal cord and the brain stem, tetanospasmin binds tightly and irreversibly to specific receptors, inhibiting the release of inhibitory neurotransmitters (glycine and γ-amino-butyric acid) from presynaptic inhibitory neurons.

The net effect is the disinhibition of motor excitatory neurons resulting in increased muscle tone and painful spasm. The autonomic nervous system also is affected, manifested by a hypersympathetic state due to failure to inhibit the adrenal release of catecholamine. Tetanospasmin interferes with pre-synaptic acetylcholine release at the neuromuscular junction and disinhibits sympathetic reflexes at the spinal level.[1] The inhibitory effect of tetanospasmin is irreversible. Recovery requires the growth of new axonal nerve terminals in 4 to 6 weeks. Clostridium tetani produces another toxin called tetanolysin that apparently has no pathogenic role.[1]

Clinical Features

The incubation period of tetanus usually lasts between 3 and 21 days with a range of one day to several months.[1] A correlation exists between the distance of the injury from the CNS and the duration of the incubation period.[1,5] The closer the distance, the shorter the incubation period. Both the incubation period and the time of onset (time from the first symptom to the first generalized spasm) inversely correlate with prognosis.[1]

Tetanus has four clinical forms: generalized, localized, cephalic, and neonatal. Generalized tetanus is the most common and severe. The generalized form typically starts with spasm of the masseter muscles, causing trismus or "lockjaw" and increased tone of the orbicularis oris resulting in the characteristic "risus sardonicus". The patient experiences diffuse spasms involving muscle groups in the neck, back, abdomen, and extremities. These spasms are painful, intermittent, and unpredictable. They often are triggered by noise and light.

Patients with generalized tetanus typically have symptoms of autonomic overactivity that manifest as sweating, tachycardia, cardiac arrhythmia, labile hypertension or hypotension, and fever.

Respiratory muscles also are affected causing periods of apnea. During generalized tetanic spasms, patients characteristically clench their fists, arch their back, flex their arms while extending their legs, and become apneic.[1] Death is due to respiratory failure or to autonomic dysfunction. Recurrent tetanus may occur if the patient is not immunized since the infection does not confer immunity. Recovery is typically 2 to 6 weeks.

Localized tetanus is usually a prodrome of the generalized form. It presents with localized rigidity and spasm of a muscle group near the site of injury. Subsequently, patients develop generalized tetanus. In some cases, localized disease reflects partial immunity with resolution of the spasms without generalization.[6] Cephalic tetanus is a form of localized tetanus involving the cranial nerves in a patient with head and neck injuries.[1]

Neonatal tetanus is related to the inadequate use of aseptic techniques in non-immunized mothers. It usually occurs within 14 days after birth.[1,7] Infants present with generalized weakness and failure to nurse, followed by rigidity and spasms. Mortality exceeds 90%. Apnea and sepsis are the leading causes of death.

Diagnosis

The diagnosis of tetanus is based on clinical symptoms and a high index of suspicion in susceptible individuals.[1] Anaerobic cultures from the wound are usually negative. A positive culture does not confirm the diagnosis. Anti-tetanus antibodies are undetectable in the majority of patients with tetanus. However, the presence of antibodies at low titer does not confer immunity and cannot be used to rule out the disease. Electroencephalography and electromyography can be helpful in ruling out other conditions.

Differential Diagnosis

Strychnine poisoning may produce a clinical syndrome similar to tetanus. Toxicologic screen of urine and blood should be performed if a suspicious injection is suspected or the history is not typical for tetanus (e.g., no injury or adequate immunization). The treatment of tetanus and strychnine poisoning is similar.

Trismus due to dental infection may be confused with cephalic tetanus. Drug induced dystonia and malignant neuroleptic syndrome can mimic tetanus.

Treatment

The treatment of patients with tetanus requires a multidisciplinary approach in the intensive care unit.[1] The mainstay of treatment consists of eliminating the source of toxin production, neutralizing unbound toxin, and managing symptoms and complications.

Halting toxin production

Wound debridement is important to eliminate the source of toxin production. All foreign bodies, including spores and necrotic tissue, should be removed. Metronidazole and penicillin G are the drugs of choice, but studies have shown better outcomes with metronidazole.[1,8] Intravenous metronidazole should be given initially at a dose of 15mg/kg followed by 20 to 30 mg/kg/day intravenously for 7 to 14 days or until there is no visible sign of active local infection.[1] Alternatives include cefazolin, cefuroxime, and ceftriaxone.

Neutralizing of unbound toxin

Tetanospasmin is bound irreversibly to tissues and only unbound toxin is available for neutralization. Passive immunization with human tetanus immune globulin (HTIG) shortens the course of tetanus and improves survival. A dose of 500 units of

HTIG should be given intramuscularly as soon as the diagnosis of tetanus is considered.[1,9] The use of intrathecal HTIG is not indicated.[1]

Management of muscle spasms

Generalized spasms are life threatening. Patients should be placed in a quiet dark room to avoid provoking muscle spasms. Spasms can cause exhaustion when severe.

Benzodiazepines are the mainstay in the symptomatic therapy of tetanus. They are usually effective in controlling muscle spasms, and patients may benefit from their amnestic effect. These drugs are gamma-aminobutyric acid (GABA) agonists. They have the potential of antagonizing the effect of the toxin.

Treatment with diazepam, lorazepam, and midazolam appear to be equally effective. Oral administration of these drugs is possible, but some patients do not absorb the drugs well and develop gastrointestinal motility disorders. Diazepam 10 to 30 mg intravenously is the usual starting dose. The intravenous formulation of diazepam and lorazepam contains propylene glycol that can cause lactic acidosis at high doses. Intravenous midazolam does not contain propylene glycol, but must be given as a continuous infusion of 5 to 15 mg/hour.

Intravenous propofol infusion is effective in controlling spasms and rigidity. It is expensive and associated with lactic acidosis, hyper-triglyceridemia, and pancreatic dysfunction.[1]

Neuromuscular blocking agents can be used in severe cases. Pancuronium (intermittent injection) and the shorter acting vancuronium (continuous injection) are usually used. Vancuronium is preferred over pancuronium because of the worsening of autonomic instability observed with the latter. Both agents should be used cautiously and the patient should be sedated adequately. These agents should be stopped daily to assess the patient's progress, and to observe for possible complications.

Intrathecal baclofen was found to control spasms and rigidity in a few small studies.[10]

Management of autonomic instability

Magnesium sulfate is effective in the treatment of autonomic dysfunction in patients with tetanus.[1,11] It also has a potential role in controlling muscle spasms. Magnesium sulfate blocks cathecholamine release from the nerves and reduces receptor responsiveness to cathecholamine.

Labetolol (0.25 to 1 mg/min) could be used because of its dual alpha and beta blocking properties.[1] Beta blockade alone should be avoided because the resulting unopposed alpha effect may produce severe hypertension and even death. Other useful agents include morphine sulfate by continuous intravenous infusion, atropine, and clonidine.

Supportive Care

Patients with tetanus are at risk of respiratory failure and aspiration. Many require endotracheal intubation to maintain adequate ventilation and preserve the airway.[1] Early tracheostomy usually is preferred in patients with tetanus who develop respiratory failure because of the likelihood of prolonged mechanical ventilation.

Nutritional support should be started as soon as possible because energy demands and protein requirements are very high. Physical therapy should be started as soon as the spasms have ceased.

Active Immunization

Tetanus is one of the rare bacterial infections that do not confer immunity following recovery. All patients with tetanus should receive active immunization with three doses of tetanus and diphtheria toxoids spaced at least two weeks apart, with

the first dose administered upon diagnosis. It should be presumed that patients who are inadequately immunized to tetanus are inadequately immunized to diphtheria as well.[12] Subsequent doses should be given at 10-year intervals throughout adulthood.

Prophylaxis

Tetanus is a preventable disease.[1] A series of three monthly intramuscular injections of tetanus toxoid provides adequate immunity for at least 5 years. Patients younger than 7 years of age and those never immunized to pertussis should receive the combined diphtheria-tetanus-pertussis vaccine (Tdap). Tetanus-diphteria (Td) booster injections are indicated every 10 years. Mild reaction to tetanus toxoid is common including local tenderness, edema, and low grade fever. Severe reactions are rare. Guillain-Barre syndrome was linked to tetanus toxoid in some reports but this was not confirmed in subsequent epidemiologic analysis.[1]

The CDC[13] has recommended administering tetanus toxoid-containing vaccine and tetanus immune globulin (TIG) as part of standard wound management to prevent tetanus (see Table 1). Tdap is preferred to Td for adults vaccinated less than five years earlier who require a tetanus toxoid-containing vaccine as part of wound management and who have not received Tdap previously. For adults previously vaccinated with Tdap, Td should be used if a tetanus toxoid-containing vaccine is indicated for wound care. Adults who have completed the 3-dose primary tetanus vaccination series and have received a tetanus toxoid-containing vaccine less than five years earlier are protected against tetanus and do not require a tetanus toxoid-containing vaccine as part of wound management.

Persons with unknown or uncertain tetanus vaccination histories should be considered to have had no previous tetanus toxoid-containing vaccine. Persons who have not completed the primary series might require tetanus toxoid and passive vaccination with TIG at the time of wound management (see Table 1). When both TIG and a tetanus toxoid-containing vaccine are indicated, each product should be administered using a separate syringe at different anatomic sites. Adults with a history of Arthus reaction following a previous dose of a tetanus toxoid-containing vaccine should not receive a tetanus toxoid-containing vaccine until at least 10 years after the most recent dose, even if they have a wound that is neither clean nor minor. In all circumstances, the decision to administer TIG is based on the primary vaccination history for tetanus (see Table 1).

Table 1. Recommendation for use of tetanus prophylaxis in wound management.

Immunization with tetanus toxoid	**Clean, minor wounds**		**All other wounds**	
	Tetanus toxoid	Immunoglobulin	Tetanus toxoid	Immunoglobulin
Unknown or less than three doses	Yes	No	Yes	Yes
Three or more doses	No, unless >10 years since last dose	No	No unless > 5 years since last dose	No

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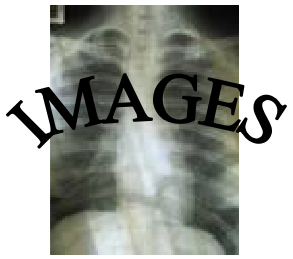
Keywords: tetanus, Clostridium tetani, case report



Chemotherapy–Induced Acral Erythema

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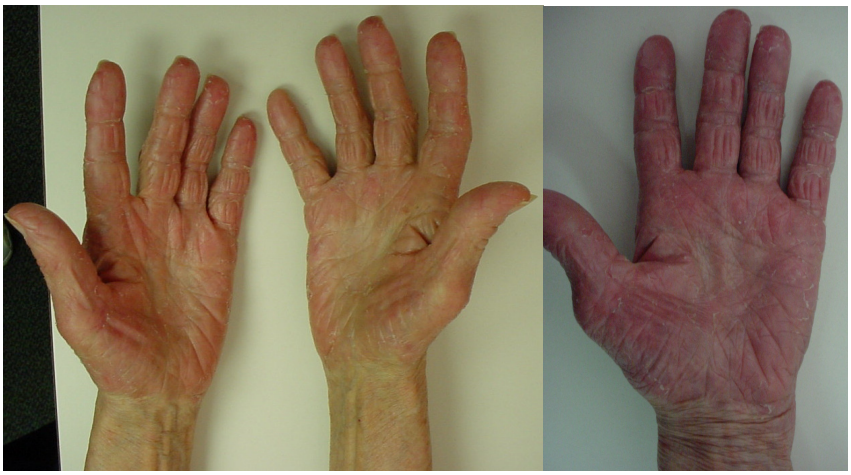

Cutaneous reactions to chemotherapeutics are common and may contribute significantly to the morbidity, and rarely to the mortality, of patients undergoing such treatments. Recognition and management of these reactions is important to provide optimal care.[1] A 79-year-old woman was diagnosed with mycosis fungoides after a tissue biopsy of a lesion in her mid-back. She received vincristine and cytoxan chemotherapy. She presented with erythema, desquamation of both hands, and very stiff underlying dermis. Symptoms were thought to be due from her original disease of mycosis fungoides, but histological examination demonstrated vacuolar degeneration of the basal cell layer and spongiosis in the epidermis. The diagnosis was consistent with chemotherapy-induced acral erythema. Chemotherapy-related cutaneous toxicity includes generalized rashes such as the spectrum between erythema multiforme and toxic epidermal necrolysis, and site-specific toxicity such as mucositis, alopecia, nail changes, extravasation reactions, or hand-foot syndrome. Most of the toxicity is reversible with chemotherapy dose reductions or delays. Early recognition and treatment of the toxicity facilitates good symptom control, prevents treatment-related morbidity, and allows continuation of anti-cancer therapy.[1-3]

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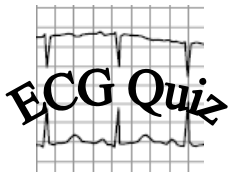
Keywords: acral erythema, cutaneous, chemotherapy, case report



Funny-Looking Ventricular Tachycardia or Something Else?

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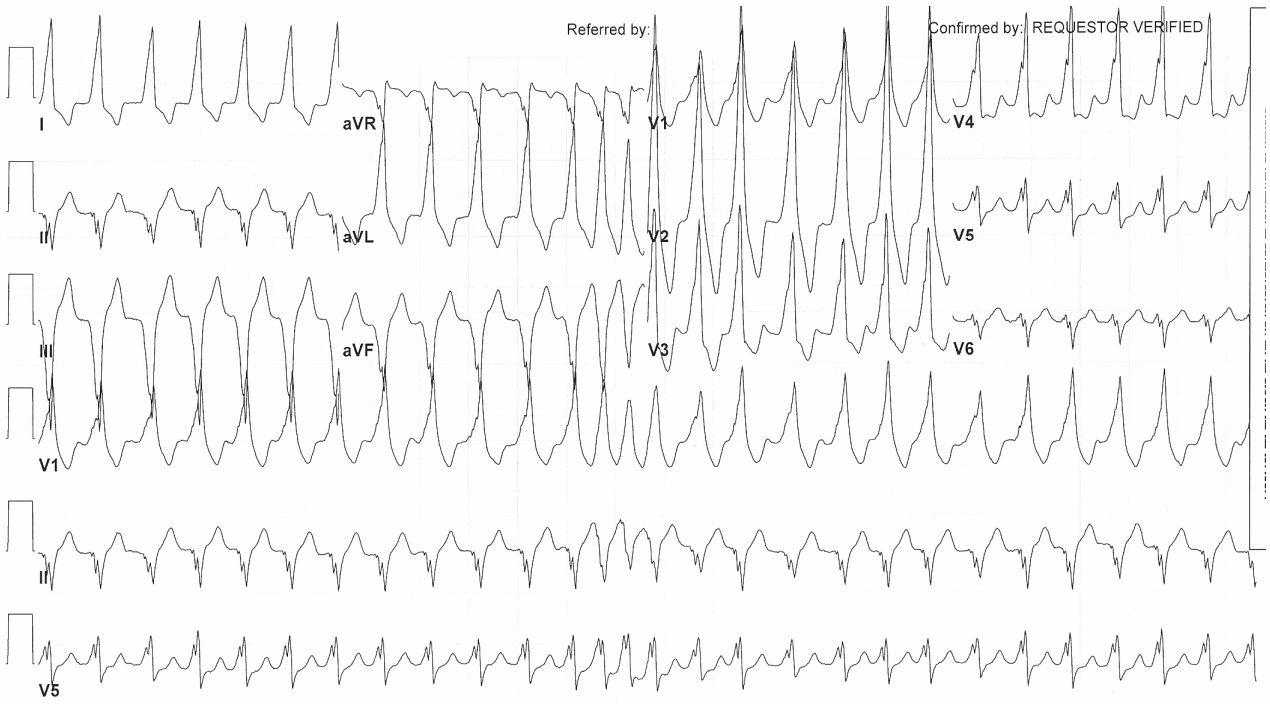
A 32 year-old white male presented to the emergency room with complaints of sudden onset dizziness, new syncope, and palpitations for the last 30 minutes. On arrival, his pulse was approximately 250 bpm and feeble. His blood pressure was 84/35. He denied any chest pain but was short of air.

His ECG is shown below:



What is the diagnosis?

(A) AV nodal Re-entrant Tachycardia

(B) Ventricular Tachycardia

(C) Atrial Fibrillation with accessory pathway (WPW Syndrome)

(D) ECG Artifact

(E) Ventricular Fibrillation

Correct Answer: C

Atrial Fibrillation (AFib) with accessory pathway (WPW Syndrome)

- AFib in patients with WPW Syndrome is potentially the most lethal arrhythmia for these patients due to its potential to deteriorate into ventricular fibrillation (Vfib).

    - In normal patients with an accessory pathway, the heart is protected from exceptionally high ventricular rates by the relatively long refractory period of the AV node. This generally limits the maximum ventricular rate. In patients with WPW, however, the accessory pathway commonly has a very short anterograde refractory period. This allows for much faster transmission of impulses from the atrial and correspondingly much higher ventricular rates can be reached.

    - The rapid ventricular rate may not allow for adequate diastolic filling of the ventricle and this in turn can predispose to hypotension. In addition, sympathetic discharge secondary to hypotension potentially can lead to an even shorter refractory period of the accessory pathway and subsequently increase the ventricular rate further. If the ventricular rate becomes too high, this can predispose to Vfib.

    - AFib usually does not conduct at a rate of more that 180 bpm through the normal AV node. On the other hand, conduction through an accessory pathway often results in more rapid ventricular rates. This usually appears as a bizarre, wide-complex, irregular tachycardia on ECG, with rates often in the 250 bpm range or higher as was noted in this patient.

What is the best initial treatment for the patient?

(A) IV Adenosine

(B) IV Lidocaine

(C) IV Diltiazem

(D) IV Amiodarone

(E) Cardioversion

(F) IV Digoxin

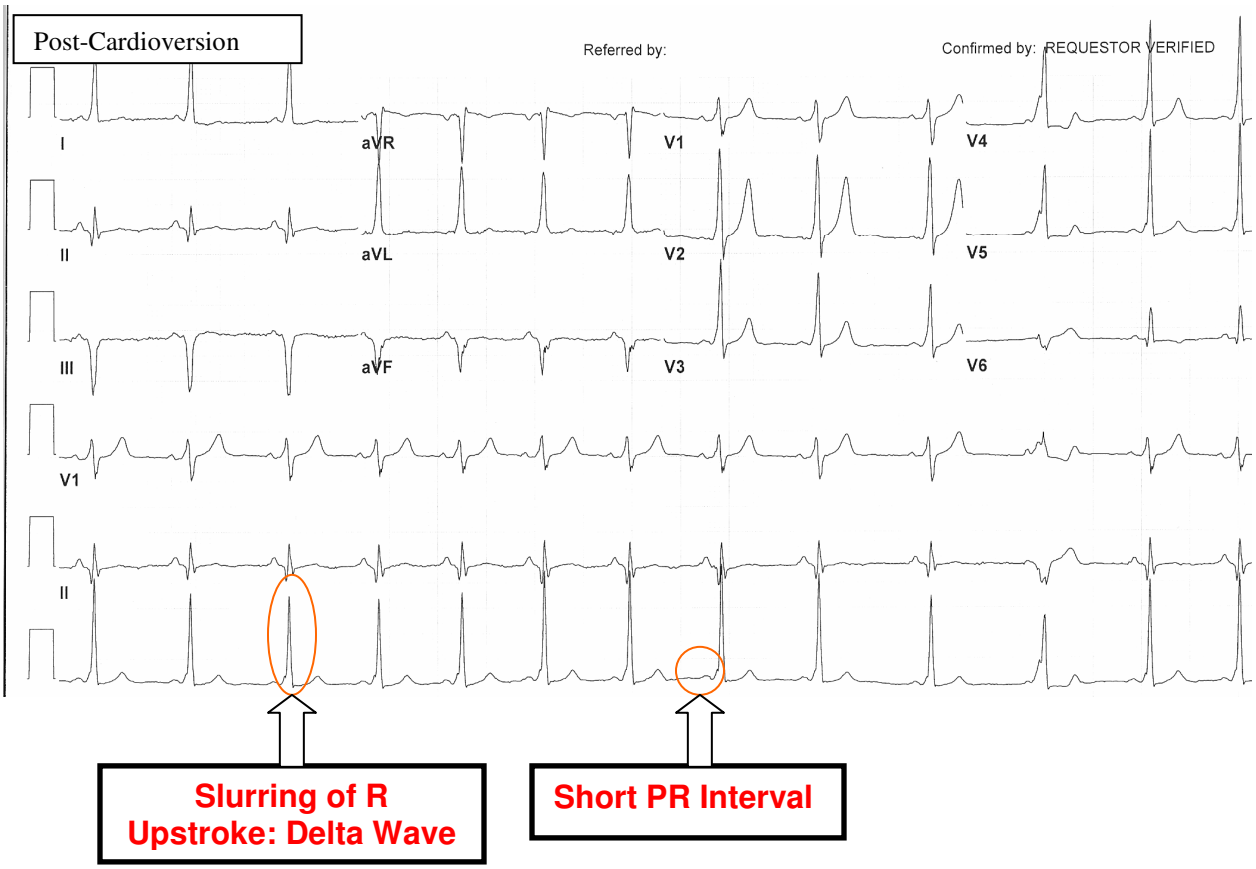
(G) IV Metoprolol

Correct Answer: E

Cardioversion

- Treatment of AFib associated with WPW is necessarily different than for a patient without an accessory pathway.
    - In WPW-associated AFib, the goal is to prolong the anterograde refractory period of the accessory pathway relative to the AV node. This slows the rate of conduction through the accessory pathway, thus the ventricular rate slows down.
    - In patients with non-WPW-associated AFib, the goal is to slow the refractory period of the AV node.
    - Standard rate control by drugs that prolong the refractory period of the AV node (e.g., calcium channel blockers, beta-blockers, digoxin, and even adenosine) conversely result in a higher rate of transmission through the accessory pathway and paradoxically increase the ventricular rate. This could have disastrous consequences possibly causing the arrhythmia to deteriorate into Vfib. Thus, such drugs are contraindicated in WPW-associated AFib. Similarly, lidocaine and amiodarone typically are not effective in this situation either.
    - If the patient is unstable with evidence of hypoperfusion, primary synchronized cardioversion should be the first-line treatment.
    - If the patient is stable, medical therapy with procainamide may be tried. Procainamide is given with a 17 mg/kg IV infusion up to a maximum of 50 mg/min. Procainamide is useful as it blocks conduction via the accessory pathway, but it has the added effect of increasing transmission through the AV node which occasionally may create a conventional atrial fibrillation that may require treatment with other medications and/or cardioversion.
    - In this case, the patient was deemed unstable and treated with synchronized cardioversion. His post-cardioversion ECG is shown below exhibiting the characteristic short PR interval and delta wave of WPW syndrome. He subsequently was referred for EP ablation of the accessory pathway.

Post-Cardioversion ECG



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Commentary

Quality and Patient Safety

Venous Thromboembolism

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Venous Thromboembolism (VTE) is a collective term to describe deep vein thrombosis (DVT) and pulmonary embolism (PE). These entities have a high prevalence in the hospitalized patient and are collectively the most common cause of preventable hospitalized death.[1-3] Approximately 10% of hospital deaths are attributed to PE.[3] Further, VTE in hospitalized patients is increasing.[4] Current hospitalized patients may be at greater risk than previous studied patients as more elderly patients are receiving advanced care (i.e., ICU, cancer, and surgical care).

Appropriate prevention measures have been underutilized nationally with a performance gap in knowledge to practice of appropriate VTE prophylaxis.[5-8] However, a reduction in morbidity and mortality with VTE prophylaxis was demonstrated.[3] Unfortunately, these data are not translating to evidence-based practice for patients. In a study of 152 MICU patients, only one-third of the patients received VTE prophylaxis.[7] There was an average two-day delay before prophylaxis was given. The Worcester DVT Study demonstrated that only 32% of high risk hospitalized patients received prophylaxis.[8]

The Agency for Healthcare Research and Quality and the Society of Hospital Medicine are taking steps to narrow this performance gap.[9-10] These steps require local action from each healthcare institution to establish a multidisciplinary team to champion VTE prophylaxis. Improving prevention of VTE should be an area of focus for healthcare leaders and providers.

The three key components to eliminate the performance gap in VTE prophylaxis are:

(1) Each institution should have a process for identifying at risk populations for VTE and instituting appropriate prophylaxis. The most effective methods minimize reliance upon a single individual to achieve desired outcomes. A recent systematic review reported that passive dissemination of guidelines to clinicians was unlikely to improve prophylaxis.[11] Clinician reminders with appropriate recommendation were more effective.[11]

(2) Once a process has been outlined, outcome measures will establish its viability. The optimal desired outcome is 100% of hospitalized patients without new VTE. A more realistic goal is 100% of hospitalized patients assessed for VTE risk with the most appropriate prophylaxis given. Each institution must review outcomes data frequently to evaluate achievement toward this goal.

(3) Refinement of the process will be necessary. Identification of road blocks preventing achievement of institutional preferred outcomes is important. A perpetual barrier is unexplained practice variation. Clinical variance only can be eliminated by standardization of practice. Evidence-based standardization will give institutions the opportunity to achieve desired outcomes. The best available evidence on practice standardization is the 7th ACCP consensus statement.[3]

A good process with good outcomes, based on evidence derived standardization is necessary to decrease VTE death in hospitalized patients. A physician champion with the appropriate support team can use these steps to produce an effective VTE prevention strategy. The current national campaign to eliminate this performance gap is called the Society of Hospital Medicine VTE Prevention Collaborative.[10] This collaborative is a good resource and could bolster individual institutional campaigns.

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Keywords: venous thromboembolism, deep vein thrombosis, pulmonary embolism, prophylaxis

Communicating with Patients at Risk for Low Health Literacy

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Introduction

Health literacy is the capacity to obtain, process, and understand basic health information and services needed to make appropriate health decisions.[1] Fundamentally, health literacy is an issue of ethics and equality. It is essential to reducing health disparities.[2] Many skills are necessary for adequate health literacy, including reading, writing, speaking, listening, and being able to advocate for oneself in the health care system.[1,3]

If the general public was health literate, better health outcomes would be expected.[2] Patients would seek care earlier because they recognize warning signs. They would read and comprehend instructions. They would understand their physician's advice and ask questions when they do not understand. Effective health communication contributes to all aspects of disease prevention and health promotion.[1] Effective communication between physician and patient is essential for the prevention, diagnosis, and treatment of illness and disease.[4] It assists patients in making complex medical care decisions and to manage their own health concerns better.

Health Literacy in the Elderly

According to the Institute of Medicine[3], almost half of all adults in the US have difficulty understanding and acting upon health information. Low health literacy is an increasing problem in the elderly.[5] Elderly individuals have more complex disease management and decision-making.[6] They are at-risk for cognitive and functional decline. Health literacy decreases with age beyond differences expected in education levels.[7]

Approximately 25% of community-dwelling older persons have limited health literacy.[6] The rates of limited health literacy were higher in individuals from more traditionally disadvantaged groups (e.g., poor and minority status).[6-8] Low health literacy often is accompanied by shame and embarrassment.[9]

Health Literacy and Health Outcomes

A US Agency for Healthcare Research and Quality study[10] concluded that low reading skill and poor health were "clearly related". Individuals with low health literacy used an inefficient mix of health care services.[7] Limited health literacy was associated with health disparities and lower healthcare access[6], poorer physical and mental health[8,11], non-adherence to preoperative medication instructions[12], hospital admissions[13], and higher medical costs[7]. These characteristics place individuals with limited health literacy at risk for poor clinical outcomes.

More direct teaching by health care professionals is needed to help patients understand their diseases and treatment program.[14] Materials with excellent content have limited value until patients and family members understand them.[15] Yet, it is difficult to identify patients with inadequate health literacy without formal testing.

Assessing Health Literacy

The Test of Functional Health Literacy in Adults – Short Version (STOFHLA)[14]

measures the ability to read real passages using materials from the health care setting. It is a reliable and valid test instrument. The STOFHLA, however, takes seven minutes to complete, too long for most medical encounters. Further, it measures reading skill and not all domains of health literacy.

Individuals with inadequate health literacy have been identified by as little as a single-item screening question.[16-17] A single question to assess health literacy easily can be asked during most medical encounters. Questions such as, How often do you have someone help you read hospital materials?, How confident are you filling out medical forms by yourself?, and How often do you have problems learning about your medical condition because of difficulty understanding written information?, were effective in detecting inadequate health literacy.[16]

Although reading ability is one of the most fundamental components of health literacy, strategies to educate patients cannot depend on written materials alone. Many domains must be evaluated for a complete assessment of health literacy. The communication gap with vocabulary and language structure probably is greater for patients with low literacy.[9] Physicians often overestimate the literacy skills of patients, particularly those at the lowest levels.[18-19]

Doctor-Patient Interactions

Patients report difficulty with communicating with their physicians.[4] In a qualitative study of patients being consulted about heart surgery, five important themes resulted.[20] The themes were (1) fear of missing information, (2) difficulty understanding information, (3) difficulty recalling information from the physician, (4) not recalling what the patient himself/herself said, and (5) difficulty communicating information to family and friends.

Physicians can enhance patient understanding with simple modifications to their communication style.[21] These modifications include taking time to assess patient understanding, avoiding medical terminology, using pictures, limiting information provided, repeating instructions, asking patients to demonstrate understanding, and acting respectfully and sensitively.

Health Literacy in Individuals with Hearing Loss

Intuitively, difficulty communicating with a physician would be expected to increase if the patient had a communication disorder. In fact, communication problems with deaf and hard-of-hearing adults may compromise health care quality.[22] Individuals with hearing loss cited risks for medical errors, misdiagnoses, and difficulty obtaining complete and accurate information from physicians.

Patients with hearing loss may not realize the amount of information they miss in the physician's office.[15] Use of a hearing aid may not compensate for hearing loss adequately during doctor-patient communications. Further, the exchange of written notes is not sufficient for effective communication.[23] Physicians should not assume that they are understood just because a hearing impaired patient nods in acknowledgment or agreement.

The number of Americans with a hearing loss has doubled during the past 30 years.[24] Three out of 5 older Americans with hearing loss and 6 out of 7 middle-aged Americans with hearing loss do not use hearing aids. The usefulness of self-assessment of hearing loss in the elderly has been reported by several authors.[25-37] Even a self-reported answer to a single question about hearing loss was shown to be an effective screening tool.[26,28,37]

Summary

Poor health outcomes are associated with low health literacy. Low health literacy may be manifested by difficulty reading health information. However, health literacy also is impacted by the doctor-patient interaction. Patients may manifest difficulty understanding verbal health information or expressing health information to the physician. In each instance, the quality of medical care may be compromised.

Given that almost half of adult patients may have difficulty understanding and acting upon health information, health care providers have an obligation to enhance communication with patients, particularly those most at risk. Patients who are elderly, have cognitive or sensory deficits, are communicatively impaired or lack a command of the spoken language, and come from traditionally disadvantaged populations are at higher risk for low health literacy levels.

Simple modifications to the doctor-patient interaction can impact the understanding of health information and encourage the patient to act upon it appropriately. Individual care plans for each patient should be developed. Brief, but effective, assessments of health literacy and hearing loss can be accomplished by a single question addressing the relevant issue. Within the exam room, physicians should take time to assess patient understanding, avoid medical terminology, use pictures, limit information provided, repeat instructions, and ask patients to demonstrate understanding. Physicians must be sensitive to recognize and address these issues. An improvement in the patient's overall health outcomes is the expected result.

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Keywords: health literacy, physician-patient relations, communication