Feature

Stroke as a Complication of H1N1 Influenza Infection: A Case Study

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In June 2010, the Centers for Disease Control and Prevention ended the US public health emergency for the H1N1 pandemic with estimates for total cases, hospitalizations, and deaths counted from April 2009 to April 2010. By the end of this period, the human H1N1 virus was estimated to have been responsible for 12 470 deaths in the United States. Most deaths associated with the seasonal flu or H1N1 infection are due to complications such as secondary infections. Experts are finding, however, that a small percentage of these deaths or comorbid conditions may be caused by disseminated intravascular coagulation, and stroke can be a sequela of disseminated intravascular coagulation. This case study describes the clinical course of a patient who had multiple strokes due to disseminated intravascular coagulation triggered by H1N1 infection. Useful clinical information about disseminated intravascular coagulation is detailed for nursing practice. Implications of the possible link between H1N1 infection (and influenza A and B) and stroke resulting from disseminated intravascular coagulation are discussed. (*Critical Care Nurse.* 2011;31[4]:e1-e8)

The human H1N1 infection (also known as swine-origin influenza virus, S-OIV, or swine flu) that recently swept across the globe is considered by experts as the most powerful pandemic threat since influenza A surfaced in 1968.[1] In April 2009, the first 2 cases of swine flu were identified in the United States by the Centers for Disease Control and Prevention (CDC). In the first 6 weeks of the swine flu epidemic in the United States, 642 cases were confirmed in 41 states; of the 642 cases, 40% were in patients from 10 to 18 years old.[1] Hospitalization status was known for 399 patients: 9% were hospitalized, 41% had chronic health issues, 36% were admitted to critical care units, and 18% required mechanical ventilation.[1] Two of the 36 hospitalized patients died.[1] The CDC's midlevel total estimates (April 2009-April 2010) for the H1N1 pandemic in the United States are 61 million cases, 274 000 hospitalizations, and 12 470 deaths.[2]

The course of severe illness and deaths associated with the swine flu is similar to the course associated with the seasonal flu, often involving viral and bacterial pneumonias that occur as secondary complications.[1] Ohuri and colleagues,[3] however, describe 2 cases of influenza that were diagnosed along with acute pulmonary microthromboembolism. The authors discuss the 1998-1999 influenza A virus epidemic in Japan that killed approximately 1300 people. Some of those patients were reported to have had sudden death from acute respiratory failure without evidence of pneumonia. The authors surmise that disseminated intravascular coagulation (DIC) induced by viral infection was the culprit.[3]

DIC is a systemic thrombohemorrhagic syndrome that results from single or multiple clinical conditions

 doi: 10.4037/ccn2011400

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that activate nonlocalized coagulation cascades, rapidly consuming clotting factors and platelets. Once clotting factors are depleted, the patient is at risk for uncontrolled hemorrhage. The result is end-organ damage.[4] Examples of conditions that trigger DIC include severe infection, malignant neoplasms, obstetric emergencies, trauma, transfusion reactions, acute hepatic failure, and rejection of transplanted organs or implanted devices.[4] These patients generally initially have signs and symptoms of bleeding (ie, hypovolemia or spontaneous hemorrhage) and/or thrombosis (ie, acral cyanosis or acute dysfunction of kidneys, liver, lungs, or brain).[4]

DIC is a rare condition that is estimated to occur in approximately 1% of all hospitalized patients; central nervous system dysfunction (such as stroke) in cases of DIC is equally rare, noted in 1 article to occur in about 2% of the study population.[4] Very little literature is published that links the flu, DIC, and stroke—the few published accounts on this topic are primarily case studies and literature reviews.[3,5,6] None of these accounts link the newest influenza threat of H1N1 to stroke. This case study describes the course of illness for a patient who had multiple strokes as sequela of H1N1 infection.

CASE STUDY

Patient X was a 34-year-old, African American woman with a medical history of mental retardation, hypothyroidism, hypertension, and appendectomy. Her hypothyroidism and hypertension were well controlled on her home medication regimen of levothyroxine, lisinopril, atenolol, and hydrochlorothiazide. She had no history of alcohol, illicit drug, or tobacco use. Her immunizations were up to date.

Patient X lived with her parents and was independent or needed minimal assistance with her activities of daily living. She spoke English, although she was difficult to understand at baseline because of her mental retardation. She appeared well nourished and cared for.

Four days before admission, patient X had dyspnea, cough, fever, and wheezing that led her mother to take her to another hospital's emergency department. According to the treating physician's summary note, she was given azithromycin for bronchitis. No chest radiography or nasal swab for influenza was done at that time. She was discharged to home. Two days later, patient X stopped the azithromycin because of diarrhea. Two days after that (day 6 of illness), her mother took patient X to a primary care physician when she experienced increasing shortness of breath and wheezing. The primary care physician noted that patient X was febrile, tachypneic, and tachycardic; therefore, she was sent directly to our emergency department for treatment.

Upon arrival, patient X's respirations were shallow and labored and she was tachypneic. Her initial vital signs were a heart rate of 117/min, a blood pressure of 149/87 mm Hg, a respiratory rate of 28/min, an oxygen saturation of 76% on room air, and a body temperature of 39.2°C. She was immediately fitted with a nonrebreather face mask with a fraction of inspired oxygen (FIO_2) of 100%. Her chest radiograph revealed mild to moderate bilateral infiltrates suggestive of an infectious process. Respiratory support was increased to an FIO_2 of 100% via bilevel positive airway pressure, but she was able to maintain an oxygen saturation of only 87%. Results of arterial blood gas analysis at that time were a pH of 7.27, a PCO_2 of 44 mm Hg, a PO_2 of 43 mm Hg, and a bicarbonate concentration of 21 mmol/L with an anion gap of 24.

Patient X was admitted to the medical intensive care unit with a chief complaint of hypoxic respiratory failure. She was immediately sedated and intubated. A notable laboratory test result was a normal white blood cell count of 5400/μL, which, when coupled with the presence of infiltrates, is indicative of an atypical pneumonia.[7] The hallmark of an atypical pneumonia is the presence of pulmonary infiltrates with extrapulmonary signs and symptoms; that is, evidence of involvement of organs such as the brain, heart, liver, kidneys, or gastrointestinal tract, often without an elevated white blood cell count.[7] Examples include confusion, diarrhea, bradycardia, elevated levels of liver enzymes, or shock.[7]

Other important laboratory test results for patient X were hyperglycemia (serum glucose level, 165 mg/dL; to convert to millimoles per liter, multiply by 0.0555), elevated level of aspartate aminotransferase (125 U/L; to convert to microkatals per liter, multiply by 0.0167), and a hemoglobin level of 11.5 g/dL. Table 1 summarizes laboratory values for patient X.

Patient X's list of problems at the time of admission included hypoxic respiratory failure, metabolic acidosis, hyperglycemia, liver dysfunction, and anemia. The immediate plan of care included respiratory support, broad-spectrum antibiotics (ceftriaxone, vancomycin, and azithromycin), oseltamivir phosphate (Tamiflu), testing for influenzas A and B, and correcting the metabolic acidosis. A goal was to rule out *Legionella* infection as well

Table 1 Selected laboratory results for patient X during 25-day stay

Result	Day 1[a]	Day 2	Day 3	Day 5	Day 8[b]	Day 15	Day 25	Normal[c]
pH	7.27	7.51	7.44	7.45	7.47			7.35-7.45
Pco_2, mm Hg	44	32.8	34.0	37.1	41.1			35-45
Po_2, mm Hg	43	81.0	66.7	83.0	72.5			80-100
HCO_3, mmol/L	21	25.2	22.4	24.9	29.8			21-28
Sao_2, %	76	96	94	97	95	99	96	95-100
Anion gap, mEq/L	24	6	9	11	8	9	11	8-16
Glucose, mg/dL	165	87	106	114	106	108	121	70-110
WBC, $\times 10^3/\mu L$	5.4	4.5	6.8	23.2	26.2	15.9	13.3	4.5-11
Hemoglobin, g/dL (female)	11.5	10.0	8.9	11.4	9.8	7.9	9.2	12-16
Hematocrit, % (female)	38.0	32.7	27.9	35.0	30.6	26.2	30.0	37-47
Platelets, $\times 10^3/\mu L$	152	58	53	88	299	646	279	150-350
INR	1.08	1.21	1.19	1.29		1.27		0.8-1.2
PT, s	14.2	14.4	15.4	16.4		12.8		10-13 s
aPTT, s	38.7	44.8	43.4	41.2		39.6		25-40 s
D-dimer, μg/mL		>20	>20					<0.5
Fibrinogen, mg/dL		183	193					200-400
FDP, μg/mL		160	320					<10
Heparin PF4 Abs		Negative						Negative
AST, U/L	125	125		49	87	58	29	10-30
ALT, U/L	26	24		16	32	17	11	10-40
Magnesium, mEq/L	2.23	2.27	3.01	1.98	2.42	2.06	1.88	1.3-2.1
Potassium, mEq/L	4.2	3.9	4.2	3.5	4.1	4.3	4.2	3.5-5.0
Influenza A	Negative			Positive				Negative
Influenza B	Negative			Negative				Negative
Subtype A				Positive				Negative

Abbreviations: ALT, alanine aminotransferase; aPTT, activated partial thromboplastin time; AST, aspartate aminotransferase; FDP, fibrin degradation products; HCO_3, bicarbonate; Heparin PF4 Abs, heparin platelet factor 4 antibody; INR, international normalized ratio; PT, prothrombin time; Sao_2, arterial oxygen saturation; WBC, white blood cell count.
SI conversion factors: To convert glucose to mmol/L, multiply by 0.0555; to convert D-dimer to nmol/L, multiply by 5.476; to convert fibrinogen to μmol/L, multiply by 0.0294; to convert AST and ALT to μkat/L, multiply by 0.0167; to convert magnesium to mmol/L, multiply by 0.5.

[a] Results of arterial blood gas analysis before intubation.
[b] Results of arterial blood gas analysis after extubation.
[c] Based on data from Maryland State Department of Health and Mental Hygiene Laboratories Administration[8] and Iverson et al.[9]

as viral infection such as H1N1 infection. A mechanical ventilation protocol for adult respiratory distress syndrome (ARDS) was initiated; the protocol included the use of low tidal volumes on an assist-control setting, increased positive end-expiratory pressure, and high FIO_2.

Patient X was tested for influenza A and influenza B, but the results were negative. On hospital day 4, another sputum sample was sent to the Maryland State Department of Health and Mental Hygiene to undergo more sensitive testing with the CDC's Human Influenza Virus Real-Time Polymerase Chain Reaction (RT-PCR) Detection and Characterization Panel. That sample was later ruled positive for "human swine-like influenza virus" or H1N1 virus.[8] This flu panel consists of influenza A PCR, influenza B PCR, and subtyping for influenza A. Diagnosis of H1N1 infection requires that the sample must test positive for both influenza A virus RNA and for subtype SWH1 (human swine-like influenza virus) RNA.[8]

Patient X received mechanical ventilation for the first 8 days of hospitalization. She had multiple episodes of desaturation associated with ARDS; therefore, the ventilator settings were manipulated many times to maximize oxygenation. Bilateral breath sounds were described as either "rhonchi," "coarse," or "crackles." Fluid balance

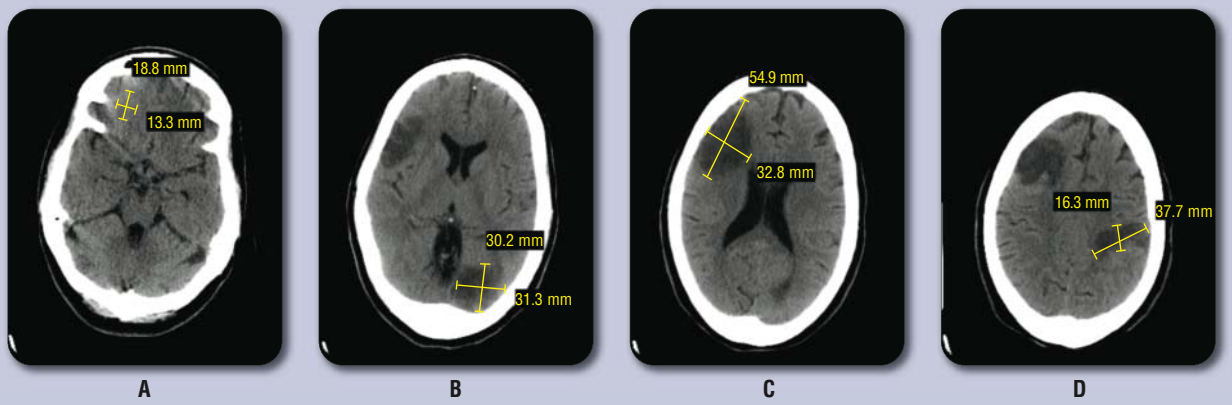

Figure Patient X's initial computed tomography scan without contrast material shows multiple infarcts in (A) right frontal lobe, (B) left occipital lobe, (C) right anteroparietal lobe, and (D) high left parietal lobe. Images are progressing (left to right) from the skull base toward the peak of the cranium.

was carefully managed by monitoring the central venous pressure, administering furosemide, and restricting fluid input to avoid further compromising her pulmonary function. Additionally, she occasionally experienced self-limiting bursts of ventricular tachycardia lasting approximately 10 beats per episode.

Antimicrobial therapies were adjusted as *Legionella* was excluded and H1N1 was verified, but the choice of agents was influenced by an effort to avoid any drugs associated with increased QT intervals on an electrocardiogram. Eventually the antimicrobial agents consisted of oseltamivir phosphate (for H1N1) and cefazolin (added because 1 tube of blood out of 2 full sets of pan cultures tested positive for *Staphylococcus hominis*).

The healthcare team was vigilant in monitoring and maintaining serum levels of magnesium at greater than 2 mEq/L and serum levels of potassium at greater than 4 mEq/L to prevent further episodes of cardiac arrhythmia. Additionally, the subclavian triple-lumen catheter was pulled back to avoid irritating the heart, but the occasional bursts of ventricular tachycardia continued. During this time, patient X remained sedated, except when sedation was temporarily lightened for examinations.

On hospital day 2, the physician team noted that patient X had become precipitously thrombocytopenic (platelet count, $58 \times 10^3/\mu L$), with an increase in the international normalized ratio, prothrombin time, and activated partial thromboplastin time. Heparin was temporarily discontinued while a heparin PF4 antibody test was completed; the result ruled out heparin-associated antibody syndrome (heparin-induced thrombocytopenia). Treatment with subcutaneous heparin was reinitiated for prophylaxis of deep venous thrombosis.

Her hemoglobin level was 10.0 g/dL and her hematocrit was 33%, which was indicative of anemia. On day 3, patient X's level of D-dimer was elevated at greater than 20 µg/mL and the platelet count was $52 \times 10^3/\mu L$. The team suspected DIC and gave her vitamin K with a plan to transfuse fresh frozen plasma and platelets if her platelet count decreased to less than $15 \times 10^3/\mu L$. The fresh frozen plasma and platelets were not transfused because her platelet count remained stable, but her hemoglobin level decreased to 8.9 g/dL and her hematocrit decreased to 28%, and therefore packed red blood cells were transfused instead.

On the same day, patient X had some minor oozing of blood from her triple-lumen catheter. For the next 2 days, she showed clinical signs of low-grade DIC (minor oozing of blood and ecchymosis at venipuncture sites), but the results of her laboratory tests were relatively stable and she continued treatment with subcutaneous heparin. Low-grade or chronic DIC represents a compensated state where the liver and bone marrow are able to produce clotting factors and platelets fast enough to not be overwhelmed by the rate of consumption by the hyperactive coagulation cascades of DIC.[4]

By day 5, her prothrombin time and international normalized ratio remained prolonged but her platelet count had increased to $88 \times 10^3/\mu L$ and the activated partial thromboplastin time had begun to improve. It appeared that patient X's DIC was resolving.

On hospital day 6, the direct care nurse discovered 2 cyanotic toes on patient X's right foot. On the same day, she reported that patient X was no longer following commands when sedation was lightened. An urgent unenhanced computed tomography scan of the head revealed areas of lucency in the right frontal (1.9 × 1.3 cm), right anteroparietal (5.5 × 3.3 cm), left occipital (3.0 × 3.1 cm), and high left parietal (3.7 × 1.6 cm) lobes without evidence of midline shift or hemorrhage (see Figure). The report

described these areas as being consistent with possible masses. A neurology consultation was obtained immediately. Patient X was given a heparin infusion to prevent thromboemboli from forming.

Although stroke was suspected, the healthcare team was diligent in ruling out any other reasons for the computed tomography results and the patient's corresponding decline. A lumbar puncture was performed in search of evidence of influenza encephalitis, aseptic meningitis, or other opportunistic infection; the results were negative. Magnetic resonance imaging confirmed that the same areas of abnormality on the computed tomography scan were multiple acute and subacute strokes throughout both hemispheres. The Figure shows the location and size of each of the infarcts on the computed tomograms.

Magnetic resonance angiography of the neck and brain showed no evidence of proximal vascular occlusion, but vascular abnormalities were present along the cortical surface at the sites of the infarcts. These abnormalities were indicative of subacute blood products from petechial hemorrhages. Carotid Doppler studies confirmed normal arteries bilaterally. Doppler imaging was used to test for normal and equal flow from the jugular veins; no signs of venous thrombus were apparent. Both transthoracic and transesophageal echocardiograms verified normal size of the left and right ventricles and normal systolic function with an ejection fraction greater than 55%. No evidence was found of a structural defect, shunt, thrombus, or vegetation that could result in cardioemboli.

Because patient X continued with somnolence and decreased responsiveness for several days, an electroencephalogram was obtained to rule out seizures. No indication of seizures was apparent on the electroencephalogram; however, independent areas of slowing that corresponded to the regions of infarct were evident on the imaging studies.

The overall clinical picture painted by the test and laboratory results strongly supported the idea that patient X had had multiple strokes due to DIC. Because H1N1 infection was the only condition this patient had at the time DIC occurred, the healthcare team felt strongly that H1N1 infection was the likely trigger. By day 9, no evidence of DIC was apparent on coagulation studies. Additionally, follow-up magnetic resonance imaging showed no new strokes, and no clinical signs of further bleeding or formation of thromboemboli were present.

Evaluation and treatment by physical, occupational, and speech and language therapists was initiated when patient X was medically able to tolerate these interventions. Patient X had spontaneous eye opening and was intermittently able to follow simple commands, but she was confused and at times her speech was incomprehensible. She had left-sided weakness and could barely move her left extremities against gravity. She had a weak gag reflex and delayed swallowing. Patient X's dysphagia was severe enough to require a percutaneous endoscopic gastrostomy tube, which was placed on hospital day 12. Care management worked with the family for support and discharge planning. Patient X became stable enough to be transferred to a telemetry unit on hospital day 13, where she stayed until she was ready for discharge.

Patient X's entire acute care hospital course lasted 25 days. Upon discharge, the family asked to take her home rather than have her sent to a neurological rehabilitation facility. She needed assistance with activities of daily living as well as additional therapy. Therefore, home health care nursing, physical therapy, and speech therapy were continued on an outpatient basis. The consensus of the team was that patient X did not need long-term anticoagulation because her coagulopathy was due to an infection that had resolved. However, daily aspirin 81 mg was prescribed as part of her home medications.

Approximately 1 month after her discharge from the hospital, she returned to have her percutaneous endoscopic gastrostomy tube removed. Although she was not back to baseline, patient X had improved; she was consistently following commands, ambulating with assistance, talking, and able to eat safely. Outpatient therapy continues.

Discussion

Patients with severe sepsis or trauma whose hospital course is complicated by DIC have a 1.5 to 2.0 times higher risk of death.[10] Nurses caring for such patients must be aware of conditions associated with DIC and the signs and symptoms of DIC (Table 2), as well as the diagnostic algorithm and treatment options for this rare complication.

DIC is diagnosed by examining the global clinical picture and abnormal results of laboratory tests. Levi and Schmair[10] describe an algorithm for diagnosis that is based on a retrospective study by a subcommittee of the International Society of Thrombosis and Haemostasis.[5] That algorithm (http://emedicine.medscape.com/article/199627

-workup) can be used as a scoring system for DIC.[5,10] It has been validated to have high sensitivity and specificity for the diagnosis of DIC as well as a high predictive value for fatal outcome. The key points of this algorithm are risk assessment and results of coagulation tests. Risk assessment identifies those patients who have an underlying condition that is associated with DIC; laboratory tests are focused on an overall coagulation panel.[5,10] Scoring assists with determining if the patient's condition is "compatible with overt DIC" or "indicative of nonovert DIC"; scoring tools for both conditions are available at http://www.isth.org.[5] Of course, differential diagnosis for any condition should always be considered. Examples of other conditions that resemble DIC are listed in Table 3.[4,5,10]

Treatment of DIC uses a 3-fold strategy: (1) treat the underlying disease that precipitated the DIC, (2) treat the coagulopathy, and (3) anticoagulate the patient.[5,10] Medical care and nursing interventions for patient X supported this strategy (Table 4). The most important part of the treatment triad is recognizing and treating the underlying problem.[5,10] In the case of patient X, the underlying disease was severe H1N1 infection complicated by ARDS. The ensuing coagulopathy was treated on the basis of the laboratory values, which were measured repeatedly to monitor her status and to give direction for further management. Administration of vitamin K to treat DIC is recommended, as well as transfusion of fresh frozen plasma, platelets, and red blood cells in response to laboratory findings.[4,10] Heparin infusion is often used for anticoagulation to avoid further formation of thromboemboli. However, the safety of heparin must be evaluated on an individual basis because patients who are prone to bleeding can have a catastrophic outcome.[4,10] Other anticoagulant agents are currently being investigated, as well as promising agents that restore physiological anticoagulant pathways. Examples include tissue factor pathway inhibitor, antithrombin, and activated protein C.[4]

Lindsberg and Grau[6] extensively reviewed literature dating back as far as the late 1800s and found that acute infection has been linked to increased risk for stroke, especially in the young and middle-aged, as well as other groups (odds ratio, 3.4-14.5). Evaluation of available scientific evidence revealed that a causal link of acute infection to stroke in terms of strength, consistency, and plausibility was "definitely present or strongly expressed."[6] Temporality, or the time elapsed between the acute infection and the onset of a stroke, was found to be "moderately or inconsistently expressed."[6] The literature suggests a pathological link between inflammatory mechanisms and prothrombic states due to a measurable activation of pathways that either reduces the anticoagulant effect of protein C or causes inhibition of the fibrolytic system, or some combination of both.[6]

Smeeth and colleagues[11] performed a retrospective self-controlled case-series investigation using the United Kingdom General Practice Research Database, which illustrates that the risk of first-ever myocardial infarction or stroke immediately after acute respiratory infection is

Table 2 Common conditions associated with and signs and symptoms indicative of disseminated intravascular coagulopathy[a]

Conditions	Signs and symptoms
Severe infection or sepsis	Spontaneous hemorrhage, hemorrhagic skin infarctions
Malignancy	Petechiae, especially on legs and soft palate
Obstetric emergencies	Ecchymosis at sites of trauma or venipuncture
Trauma	Hypovolemia or hypovolemic shock
Transfusion reactions	Venous thrombosis, acral cyanosis, or limb ischemia
Acute hepatic failure	Organ failure
Rejection of transplanted organs or implanted devices	

[a] Based on information from Becker and Wira,[4] Levi and Schmair,[10] and Taylor et al.[5]

Table 3 Differential diagnosis for disseminated intravascular coagulopathy[a]

Heparin-induced thrombocytopenia
Hemolytic-uremic syndrome
Human immunodeficiency virus–induced thrombotic thrombocytopenia purpura
Chemotherapy-induced microangiopathy

[a] Based on information from Levi and Schmair.[10]

Table 4 Nursing interventions for patient X

Problem	Nursing interventions	Rationales
Hypoxic respiratory failure Acute respiratory distress syndrome (ARDS)	1. Assist with endotracheal intubation for mechanical ventilation	1. Provides effective ventilatory support and oxygenation
	2. Institute evidence-based protocol for ARDS	2. Maximizes patient's outcomes for ARDS
	3. Administer sedation as ordered	3. Increases patient's comfort and safety while intubated
	4. Lighten sedation for examinations	4. Allows accurate neurological assessment
	5. Monitor oxygen saturation; report changes to the physician	5. Evaluates effectiveness of ventilatory support
	6. Monitor results of arterial blood gas analysis; report abnormal results to physician	6. Evaluates effectiveness of ventilatory support and guides ventilator changes
	7. Suction as needed via endotracheal tube	7. Maintains patent airway
	8. Administer diuretics as ordered	8. Decreases pulmonary fluid load to improve oxygenation
	9. Monitor central venous pressure	9. Evaluates effectiveness of fluid balance control and cardiac output
	10. Monitor fluid input and output	10. Evaluates effectiveness of fluid balance control and cardiac output
	11. Assess lung sounds	11. Evaluates for airway obstruction due to mucus and fluids
H1N1 infection	1. Test patient for influenzas A and B	1. Identifies pathogen and guides treatment
	2. Administer oseltamivir phosphate as ordered	2. Treatment of choice for H1N1
	3. Monitor for signs and symptoms of respiratory distress	3. Indicates possible need for mechanical ventilation or ventilator setting adjustment
	4. Wear a mask and gloves; wash hands	4. Prevents transmission of H1N1
Disseminated intravascular coagulopathy (DIC) Coagulopathy Decreased platelets Anemia	1. Monitor for clinical signs and symptoms of DIC; report signs and symptoms to physician	1. Diagnosis element for DIC; evaluates for worsening or resolving DIC
	2. Institute bleeding precautions	2. Protects patient with DIC from excess blood loss
	3. Monitor coagulation studies; report abnormal results or trends to the physician	3. Diagnosis element for DIC; evaluates for worsening or resolving DIC
	4. Monitor complete blood cell count; report abnormal results or trends to the physician	4. Diagnosis element for DIC; evaluates for worsening DIC and internal bleeding
	5. Transfuse fresh frozen plasma, platelets, or packed red blood cells as ordered; administer vitamin K as ordered	5. Replaces clotting factors, platelets, and red blood cells lost because of coagulopathy
	6. Administer heparin infusion as ordered	6. Prevents microthromboemboli formation
Ventricular tachycardia	1. Monitor cardiac rhythm; report arrhythmias to the physician	1. May cause ineffective cardiac output and hemodynamic instability
	2. Daily electrocardiogram or more often if needed; report abnormalities to the physician	2. Monitors for and aides in accurate diagnosis of arrhythmias
	3. Monitor vital signs	3. Alerts caregivers to hemodynamic instability
	4. Monitor serum levels of magnesium and potassium; report abnormal results to the physician	4. Electrolyte imbalance may cause dysrhythmias
	5. Administer magnesium as ordered	5. Hypomagnesemia causes ventricular tachycardia
	6. Avoid medications associated with increased QT intervals	6. Medications known to increase QT intervals are associated with ventricular tachycardia
	7. Pull back the central catheter	7. Alleviates mechanical irritation of the myocardium
Multiple strokes Impaired mobility Dysphagia	1. Arrange for urgent computed tomography of the head as ordered	1. Standard of care for any patient exhibiting new-onset stroke symptoms; rules in/out hemorrhage
	2. Perform neurological assessments as ordered; report changes to the physician	2. Alerts healthcare team to deterioration in patient's condition and possible need for repeat computed tomography or other interventions
	3. Turn and position every 2 hours	3. Helps maintain skin integrity
	4. Aspiration precautions	4. Stroke patients are at high risk for aspiration pneumonia developing
	5. Advocate for physical therapy, occupational therapy, and speech language pathology evaluations	5. Early therapy and multidisciplinary care maximizes functional outcomes for stroke patients
	6. Feed via percutaneous esophageal gastrostomy tube as ordered	6. Maintains optimal nutrition while reducing risk of aspiration
	7. Administer aspirin as ordered	7. Secondary stroke prevention

substantially elevated. Within the first 3 days after exposure, the rate of myocardial infarction increased approximately 5-fold (incidence ratio, 4.95; 95% confidence interval, 4.43-5.53) and the stroke rate increased 3-fold (incidence ratio, 3.19; 95% confidence interval, 2.81-3.62).[11] As demonstrated in other studies, the further out from the infection, the further the decrease in occurrence of myocardial infarction and stroke risk.[11]

Experts were forecasting a second wave of H1N1 to sweep across the United States in January and February 2010; however, that event never materialized and the CDC lifted the public health emergency for H1N1 in June 2010.[2] One could surmise that the second wave was avoided by extensive public immunization for H1N1. Although the association between acute infection and stroke seems apparent, the literature lacks specificity or experimental evidence; therefore we cannot adequately establish causality. More rigorous prospective investigations are needed. If causality is established, then in the future, influenza vaccination could be considered a component of the preventative treatment for stroke.

Conclusion

DIC is a rare condition. Nurses need to be alert to the signs and symptoms of this potentially deadly syndrome and understand the treatment plan. Patient X required a multitude of nursing interventions to ensure an optimized outcome. These interventions were based on the 3-fold treatment strategy for DIC, supportive care, and treatment of complications. Nurses are in a unique position to affect the outcome of these patients through diligent observation and monitoring, coordination of multidisciplinary care, and the application of evidence-based practice. CCN

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Financial Disclosures
None reported.

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