Clinical Reasoning Case Study

Introduction and Patient Presentation

Joy Smith is a 70-year old lady living alone with her husband as a retired and independent couple. Her weight is 88 kg, and she has a history of hypertension, atherosclerosis, and osteoarthritis. She has undergone a percutaneous coronary intervention in 2009 when a stent was placed in her left anterior descending artery. She has also had a cholecystectomy in 1989 and a left total knee replacement in 2010. Her current symptoms began three days back when her husband noticed a loss of appetite, lethargy, and unwillingness to get out of bed. This morning she woke up at 4:30 AM with chills, and she was agitated and didn't know where she was. One hour later, her husband brought her to the ED agitated, confused, and fatigued. She had lost sense of time and place. She was reoriented by the triage nurse and then assessed. Her vitals were SpO_2 94% on room air, heart rate 105, temperature 38.3 ˚C, and respiratory rate 26. Joy also complained of a stinging sensation when passing urine. Her current medications include Aspirin 100 mg daily in the morning; Lipitor 40 mg daily in the evening; Panadol Osteo - 2 tablets TDS, and Carvedilol 50 mg BD. She does not have any known drug allergy. After the initial assessments, at 6.00 AM Joy was transferred to an acute care bed via a wheelchair where further assessments revealed her deteriorating condition.

Patient Assessment

Assessment	Primary (0530 Hrs)	Secondary (0600 Hrs)
	• Airway Patent	• Airway Patent

A		• Nil stridor sounds • Nil mouth secretions • Nil central cyanosis • Nil face, tongue and neck edema
B	• RR 26 • SpO_2 94% on Room Air	• RR 27 (Normal respiratory rate 12-20 per minute, Older adult 15-20) (Berman et al., 2015) • SpO_2 93% on Auscultation (Normal SpO_2 values vary between 95 and 100%) (Pretto, Roebuck, Beckert, & Hamilton, 2014) • Breathlessness • Mild use of accessory muscles on inspiration The patient's oxygen saturation is within the normal range at room air. However, she appears breathless and still uses accessory muscles.
	• BP 95/55 mmHg	• BP 90/55 mmHg (Normal is <120/80 mmHg) (Berman et al., 2015)

C	• HR 105	• HR 116 regular and weak (Normal heart rate is 60- 100 bpm (Berman et al., 2015) • Nil peripheral or central cyanosis • 18g cannula insitu on the right posterior forearm
D	• Confused, fatigued, and agitated. • Loss of appetite • Lack of energy to perform regular house duties • Stinging pain while passing urine	• GCS 14 (Eyes=4, Verbal=4, Motor=6) (GCS of 13-15 shows a good prognosis and mild brain injury (BrainLine, 2018)) • Alert and talking in full sentences • Pain score of 3/10 when passing urine • Fluctuating orientation • Lying in an acute care bed
	• Temperature 38.3 ˚C	• Temperature 38.3 ˚C (Normal 36.0 and 37.4 °C) (Berman et al., 2015) • Surgical scar on left knee and rest of skin appearance intact • Skin turgor is decreased while the skin

E	High temperature could be an indication of an infection.	is intact • Wearing a hospital gown, lying in the hospital bed and covered with a sheet and one hospital blanket. Joy is still hyperthermic and dehydrated. Left knee scar is from the TKR replacement.
F	_	• On Hartman's IV fluid running at 60 mls/hr via posterior right forearm cannula.
	_	• Venous blood: normal pH 7.36, normal pCO_2 43 mmHg, normal pO_2 40 mmHg, high glucose 9.0 mmol/L (normal 4.0 – 8.0 mmol/L) and high lactate 3 mmol/L (normal 0 – 2 mmol/L).

G		• Full blood count: Low Hb 115 g/L (normal 115-165 g/L for adult females) (MyDR, n.d.) • Urinalysis all normal within limits

Process Information: Pathophysiology

From the assessment, it is clear that the patient has two related conditions. She has Acute Respiratory Distress Syndrome (ARDS) caused by urosepsis.

Urosepsis

Urosepsis is a variant of sepsis caused by the infection of the urogenital tract and is a systemic response to an infection (Wagenlehner et al., 2013; Kalra, 2009). Mostly, urosepsis is caused by gram-negative bacilli such as *Escherichia coli* (50%), *Proteus* spp. (15%), *Enterobacter* and *Klebsiella* spp. (15%), and *Pseudomonas aeruginosa* (5%) (Dreger et al., 2015), which are also responsible for ARDS (Kim & Hong, 2016; Xu, Huang, Mao, Zhang, & Li, 2015).

In adults, lactate levels are an indicator of the severity of sepsis (Wylie-Cheer & Goodson, 2016) with a lactate level >4 mmol/L considered significant (Chamberlain et al., 2014). From the assessment, the patient has increased lactic acid (3 mmol/L) accumulation potentially due to the anaerobic respiration following reduced saturation of oxygen due to respiratory distress. Looking at the SIRS criteria, increased temperature (>38.5 ˚C), hyperglycaemia (blood glucose >7.7 mmol/L), tachycardia (heart rate >90), tachypnoea (RR >25), altered consciousness, dehydration, and hypotension indicate severe sepsis (Dreger,

Degener, Ahmad-Nejad, Wöbker, & Roth, 2015; Wylie-Cheer & Goodson, 2016). The patient's assessment values of RR 27, HR 116, increased temperature, hyperglycaemia, reduced skin turgor, and chills specifically point to urosepsis and ARDS.

Although urosepsis can be diagnosed using the SIRS criteria above, biological and other clinical symptoms must also be considered (Wagenlehner et al., 2013; Kalra, 2009). Apart from the already explored symptoms of sepsis, the patient has painful urination which is a sign of UTI, and in combination with other symptoms points to urosepsis (Kalra, 2009; Dreger et al., 2015). Another indication of sepsis is reduced brain consciousness or functionality. The patient's orientation shows drastic reduction and her GCS is 14, indicating mild brain dysfunctionality. Circulatory assessments initially show BP 95/55 mmHg, HR 105, and nil central or peripheral cyanosis (Sepsis Alliance, n.d.). However, after 30 minutes the BP suddenly changes to 90/55 mmHg from 95/55 mmHg, and the HR also increases from 105 to 116, which is a clear indication of sepsis (Thursky et al., 2018).Therefore, the most likely diagnosis is urosepsis that causes ARDS confirmed by the stinging sensation when passing urine with a pain score of 3/10.

ARDS

ARDS is responsible for fluid leaks into the air sacs, which limits the supply of oxygen to the blood. Its hallmark is increased permeability of the capillaries. The damaged capillary endothelium and alveolar epithelium are responsible for the accumulation of protein-rich fluid within the alveoli (Kim & Hong, 2016). This results into diffuse alveolar damage and release of pro-inflammatory cytokines. The cytokines recruit neutrophils and are activated to release toxic mediators, which leads to a cascade of events that result in oxidative damage (Kim & Hong,

2016). ARDS is a life-threatening condition that leads to the reduction of oxygen supply to the other parts of the body (Kim & Hong, 2016).

Breathing assessments indicate RR 27, SpO_2 93%, and on auscultation there was decreased air entry in both bases. The patient also uses accessory muscles on inspiration following breathlessness. When presented to the ED, she had increased breathing, shortness of breath, low blood pressure, confusion, and extreme fatigue, which are signs of ARDS (Sepsis Alliance, n.d.; Walkey, Summer, Ho, & Alkana, 2012; Chamberlain et al., 2014). All these symptoms and assessments specifically indicate that the patient has ARDS, which has stemmed from urosepsis.

Nursing Priority Problems, Interventions, Rationale, and Outcomes

1st Critical problem: Impaired gas exchange related to ARDS evidenced by shortness of breath, confusion, fatigue, agitation, use of accessory muscles on inspiration, decreased air entry in both bases, SpO2= 94% on RA, RR= 27 P/M, and pH = 7.36.		
1st SMART Goal: To increase and maintain the oxygen saturation to be over 95% on room air within 24 hours.		
Interventions	**Rationale**	**Outcomes**
Use of Acapella®, which is an airway clearance device that combines oscillations and the resistive features of	One of the complications of ARDS is a reduction in the SpO2 and reduced dynamic compliance (Sharm et al.,	After 60 minutes of oxygen therapy 15L/hr on NRM, it is expected that the patient's oxygen saturation will be in

positive expiratory pressure device to diminish mucus adhesive and decrease in the collapsibility of the airways (Sharma, Prem, & Jain, 2018). It is an evidence-based method in the improvement of oxygen saturation and respiratory mechanics (Sharm et al., 2018). The Acapella® is connected to the expiratory port of the mechanical ventilator with the frequency set at the minimum for 15 minutes. Increase in dynamic compliance of the lungs and SpO2 are always evident between 10-60 minutes. The patient's position on the bed needs to be changed in	2018). ARDS manifests through an inflammatory response that causes exudate formation and tissue edema (Walkey et al., 2012). During the acute inflammatory stages of ARDS, there is an influx of neutrophils, impaired endothelial cell barrier function, and release of proinflammatory cytokines (Walkey et al., 2012). Respiratory failure occurs due to the accumulation of protein-rich fluid in the distal airspaces. There is also reduced surfactant production by type II epithelial cells (Walkey et al., 2012).	the normal range between 96% and 100%. It is also expected that her breathing will get better as compared to an hour ago. The normal respiratory rate between 14 and 20 RR with no secretions obstruction in the upper airway is expected after 1 hour. Also, it is expected that the originally high lactate level will reduce from 3.0 mmol/L to 2 mmol/L.

four hour durations.		
Encourage patient to lie in prone position.	Repositioning from supine to prone positioning relieves lung compression. It also redistributes the lung edema to the less perfused areas (Walkey, Summer, Ho, & Alkana, 2012). Consequently, it results in improved oxygenation. Again, it also reduces the transpulmonary pressures, which are injurious. Postural prone positioning facilitates lung drainage as well as reduces ventilator-associated pneumonia (Walkey et al., 2012). However, extreme care must be taken to avert adverse events such as catheter or	

	tube dislodgement, endotracheal obstruction, and pressure ulcers. This has proven to be useful in improving airflow with ventilation in ARDS (Walkey et al., 2012).	

2nd Critical Problem: Fever (pyrexia) related to the UTI evidenced by decreasing skin turgor, hyperthermia, chills, RR 27, and HR 116.		
2nd SMART Goal: To maintain normal body temperature range (36.6-37.4) within 2-3 hours and reduce the impacts of the UTI.		
Interventions	**Rationale**	**Outcomes**
Immediate administration of ordered IV antibiotics and antipyretics within one hour. Closely monitor the vitals such as hourly urine output, heart rate, temperature,	Sepsis is caused by the invasion of the body by bacteria. In the patient's case, the major suspect of urosepsis is *E. coli* due to the presence of an inflammatory	Regulation of the high temperature from 38.3 ˚C to the normal range (36.6-37.4 ˚C) within the first 2 hours since the first assessment. The patient does not have a

oxygenation, respiratory rate, and blood pressure. Keenly check the IV cannula site for any signs of adverse drug reaction every 15 minutes.	response. Consequently, administering antibiotics and antipyretics should be a priority response in the first hour (Dreger et al., 2015). The mostly used antipyretics include acetaminophen or non-steroidal anti-inflammatory drugs (NSAIDs), which are effective in lowering body temperature through decreasing the threshold of body temperature control within the hypothalamus (Egi, Makino, & Mizobuchi, 2018). These drugs inhibit the production of prostaglandin E (Egi et al., 2018; Zhang, Chen, & Ni, 2015; Niven, Stelfox, &	fever anymore, and the pain during urination has reduced.

	Laupland, 2013b).	
External cooling mechanisms such as using cold bed-sheets, cooling blankets, or ice packs.	Studies have shown that the regulation of fever through external cooling helps control short term effects of sepsis (Schortgen et al., 2012). External cooling results in a rapid decrease in oxygen consumption that restores tissue oxygenation during septic shocks. It also helps in reducing body temperature. In older adults, fever makes them have chills if it worsens. Thus, cooling will help reduce the effects of fever (Schortgen et al., 2012). External cooling is associated with a decrease in early mortality in febrile patients	Reduced fever and its impacts such as dehydration. The patient does not have chills anymore.

	and decrease in the vasopressor dose required to maintain arterial pressure (Niven, Laupland, & Stelfox, 2013a).	

3rd Critical Problem: Hypovolemia related to sepsis and decreased cardiac output supported by BP= 90/55 mmHg, HR= 116 bpm, regular and weak pulse, elevated temperature T= 37.8 ˚C, hyperlactatemia, and reduced skin turgor.		
3rd SMART Goal: To restore the systolic pressure to be over 100 mmHg in 15 minutes. Reestablished electrolyte-fluid balance by improving and maintaining blood pressure between 85 and 120 systolic and heart rate to between 60 and 100 bpm in an hour. Improve the skin turgor status and mental health capacity in 2 hours, reduce the temperature to normal range between 36 to 37 ˚C, and reduce fluid perfusion.		
Interventions	**Rationale**	**Outcomes**
Fluid therapy with IV Hartman's 60 ml/hr, for half an hour. Continue monitoring the	Patients with hypovolemia, hypotension, and hyperlactatemia have a high risk of mortality.	Following an hour of administering fluid therapy, the patient is expected to show improved vitals.

vitals (BP, RR, HR, SpO_2, and T) every 30 minutes. Observe the urine output per hour, mental capacity, and skin turgor.	Hyperlactatemia, as in the case of the patient is caused by circulatory collapse and hypoperfusion of tissues, which leads to lactic acid fermentation, cell hypoxia, and disrupted mitochondrial respiration. Fluid resuscitation is an indispensable procedure in the acute management and restoration of tissue perfusion due to the inflammatory response, especially in splanchnic organs (Wu et al., 2015). Hypertonic saline (HTS), synthetic colloids, and crystalloids are preferred in fluid therapy in sepsis patients as they help achieve	The systolic blood pressure will improve from 90 systolic to a range between 110 and 120 systolic. Her heart rate will also be within the normal range of 60 and 100 bmp. Her radial pulse will be strong and stable compared to the current regular and weak status. Her respiratory rate is also expected to be between 14 and 20, showing a decrease from RR 27. Skin turgor will improve. Improvement on orientation to place and time after 2 hours but drowsiness and

	mean arterial pressure (Wu et al., 2015). They restore the cardiac output, oxygenation, and intravascular volume (Wu et al., 2015).	maintained GCS 14.
Administer blood transfusion of 1 pack RBC within 2 hours and closely monitor the vitals. Transfusion of RBCs should take place until haematocrit reduces to or equals 30%.	Sepsis patients experience hyperlactatemia, hypotension, decreased mixed venous oxygen saturation. Therefore, the administration of packed red blood cells (pRBCs) increases haemoglobin concentration that correspondingly increases the oxygen-carrying capacity of blood (Chan et al., 2017). Anaemia, which is common in sepsis patients, is a culprit for the lactatemia due to the	After 2 hours of administering the pRBC transfusion, the blood culture will indicate normalcy in haemoglobin to 130 g/L and absolutely no signs of fluid overload.

	collapse of circulatory systems and hypoperfusion of tissues (Chan et al., 2017). Blood transfusion is a sure way to restore and improve cardiac output. However, there is a need to closely monitor the vitals to avert any adverse events (Chadebech et al., 2017).	

References

Australian Broadcasting Corporation. (2011, August 17). Fever - Health & Wellbeing. Retrieved from https://www.abc.net.au/health/library/stories/2011/08/17/3294499.htm

BrainLine. (2018, July 25). What Is the Glasgow Coma Scale? Retrieved from https://www.brainline.org/article/what-glasgow-coma-scale

Chadebech, P., Bodivit, G., Razazi, K., De Vassoigne, C., Pellé, L., Burin-des-Roziers, N., … Pirenne, F. (2017). Red blood cells for transfusion in patients with sepsis: respective roles of unit age and exposure to recipient plasma. *Transfusion*, *57*(8), 1898-1904. https://doi.org/10.1111/trf.14170

Chamberlain, D. J., Willis, E., Clark, R., & Brideson, G. (2014). Identification of the severe sepsis patient at triage: a prospective analysis of the Australasian Triage Scale. *Emergency Medicine Journal*, *32*(9), 690-697. https://doi.org/10.1136/emermed-2014-203937

Chan, Y. L., Han, S. T., Li, C. H., Wu, C. C., & Chen, K. F. (2017). Transfusion of Red Blood Cells to Patients with Sepsis. *International journal of molecular sciences*, *18*(9), 1946. https://doi.org/10.3390/ijms18091946

Dreger, N. M., Degener, S., Ahmad-Nejad, P., Wöbker, G., & Roth, S. (2015). Urosepsis—Etiology, Diagnosis, and Treatment. *Deutsches Aerzteblatt Online*, *112*(49), 837–848. https://doi.org/10.3238/arztebl.2015.0837

Egi, M., Makino, S., & Mizobuchi, S. (2018). Management of fever in critically ill patients with infection. *Journal of Emergency and Critical Care Medicine*, *2*, 10-10. https://doi.org/10.21037/jeccm.2018.01.09

Kalra, O. (2009). Approach to a patient with urosepsis. *Journal of Global Infectious Diseases*, *1*(1), 57. https://doi.org/10.4103/0974-777x.52984

Kim, W., & Hong, S. (2016). Sepsis and Acute Respiratory Distress Syndrome: Recent Update. *Tuberculosis and Respiratory Diseases*, *79*(2), 53. https://doi.org/10.4046/trd.2016.79.2.53

MyDR. (n.d.). Full blood count (FBC). Retrieved from https://www.mydr.com.au/tests-investigations/full-blood-count-fbc

Niven, D. J., Laupland, K. B., & Stelfox, H. T. (2013a). External Cooling Applied to Febrile Patients with Septic Shock: Control for Cointerventions? *American Journal of Respiratory and Critical Care Medicine*, *187*(11), 1272-1273. https://doi.org/10.1164/rccm.201205-0877le

Niven, D. J., Stelfox, H. T., & Laupland, K. B. (2013b). Antipyretic therapy in febrile critically ill adults: A systematic review and meta-analysis. *Journal of Critical Care*, *28*(3), 303-310.https://doi.org/10.1016/j.jcrc.2012.09.009

Pierrakos, C., Karanikolas, M., Scolletta, S., Karamouzos, V., & Velissaris, D. (2012). Acute Respiratory Distress Syndrome: Pathophysiology and Therapeutic Options. *Journal of Clinical Medicine Research*, *4*(1), 7-16. https://doi.org/10.4021/jocmr761w

Pretto, J. J., Roebuck, T., Beckert, L., & Hamilton, G. (2014). Clinical use of pulse oximetry: Official guidelines from the Thoracic Society of Australia and New Zealand. *Respirology, 19*(1), 38-46.

Samaha, G., Casserly, B., & Stevens, J. (2012). Management of Severe Sepsis and Septic Shock. *Severe Sepsis and Septic Shock - Understanding a Serious Killer*. https://doi.org/10.5772/29876

Schortgen, F., Clabault, K., Katsahian, S., Devaquet, J., Mercat, A., Deye, N., … Brochard, L. (2012). Fever Control Using External Cooling in Septic Shock. *American Journal of Respiratory and Critical Care Medicine*, *185*(10), 1088-1095. https://doi.org/10.1164/rccm.201110-1820oc

Sepsis Alliance. (n.d.). Sepsis and ARDS. Retrieved from https://www.sepsis.org/sepsis-and/sepsis-and-ards/

Sharma, P., Prem, V., & Jain, S. (2018). Immediate Effects of Acapella® on Dynamic Lung Compliance in Mechanically Ventilated Patients with Acute Respiratory Distress Syndrome: A Case Series. *Indian Journal of Critical Care Medicine*, *22*(2), 100-102. https://doi.org/10.4103/ijccm.ijccm_157_17

Thursky, K., Lingaratnam, S., Jayarajan, J., Haeusler, G. M., Teh, B., Tew, M., … Slavin, M. A. (2018). Implementation of a whole of hospital sepsis clinical pathway in a cancer hospital: impact on sepsis management, outcomes, and costs. *BMJ Open Quality*, *7*(3), e000355. https://doi.org/10.1136/bmjoq-2018-000355

Wagenlehner, F. M., Lichtenstern, C., Rolfes, C., Mayer, K., Uhle, F., Weidner, W., & Weigand, M. A. (2013). Diagnosis and management for urosepsis. *International Journal of Urology*, *20*(10), 963-970. https://doi.org/10.1111/iju.12200

Walkey, A., Summer, Ho, & Alkana. (2012). Acute respiratory distress syndrome: epidemiology and management approaches. *Clinical Epidemiology*, 159. https://doi.org/10.2147/clep.s28800

Wu, C., Chan, K., Cheng, Y., Yeh, Y., & Chien, C. (2015). Effects of different types of fluid resuscitation for haemorrhagic shock on splanchnic organ microcirculation and renal reactive oxygen species formation. *Critical Care*, *19*(1). https://doi.org/10.1186/s13054-015-1135-y

Wylie-Cheer, B., & Goodson, H. (2016). Sepsis Down Under: Sepsis care and treatment in New Zealand and Australia. *Journal of Emergency Management Services*, *41*(9), 60-63.

Xu, Z., Huang, Y., Mao, P., Zhang, J., & Li, Y. (2015). Sepsis and ARDS: The Dark Side of Histones. *Mediators of Inflammation*, *2015*, 1-9. https://doi.org/10.1155/2015/205054

Zhang, Z., Chen, L., & Ni, H. (2015). Antipyretic Therapy in Critically Ill Patients with Sepsis: An Interaction with Body Temperature. *PLOS ONE*, *10*(3), e0121919. https://doi.org/10.1371/journal.pone.0121919

Kozier, Barbara, Erb, Glenora Lea, Berman, Audrey, Snyder, Shirlee, Levett-Jones, Tracy, Dwyer, Trudy, Hales, Majella, Harvey, Nicole, Moxham, Lorna, Park, Tanya, Parker, Barbara, Reid-Searl, Kerry, and Stanley, David (2015) *Kozier and Erb's Fundamentals of Nursing [3rd Australian edition].* Pearson Australia, Melbourne, VIC, Australia.

Berman, A., Snyder, S., Levett-Jones, T., Dwyer, T., Hales, M., Harvey, N., … Stanley, D. (2015) *Kozier and Erb's Fundamentals of Nursing (3ed).* Pearson Australia, Melbourne, VIC, Australia.