# Hemodynamic Disorders

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## Edema

- Excess accumulation of fluid in interstitial space or in serous cavity is called edema.
- Types
  - Local edema
  - Generalized edema



- Local edema
  - Acute inflammatory edema
  - Allergic edema
  - Edema due to impaired venous return of leg following
    - Long journey
    - Long period of standing
  - Lymphedema following lymphatic obstruction
    - e.g. Filariasis



- Generalized edema
  - Hepatic edema
  - Cardiac edema
  - Renal edema
  - Nutritional deficiency edema
  - Pregnancy edema
  - Myxedema
  - Unexplained edema



- Clinical Types of Edema
  - Pitting edema
  - Non-pitting edema
- Causes of edema/Pathophysiological Category
  - Increased hydrostatic pressure
  - Decreased colloidal osmotic pressure
  - Retention of salt and water
  - Lymphatic obstruction



## Elephantiasis

- A non-pitting edema following lymphtaic obstruction
- Accumulation of lymph in tissue
- Causes inflammation, fibrosis.
- Fibrosis causes non-pitting edema.

- All causes are found in hepatic edema
- Normally interstitial space and serous cavity contains small amount of fluid. Excess amount of fluid cases edema.



- Serous cavities
  - Peritoneal cavity - Excess accumulation of fluid is called ascites.
  - Pleural cavity - Excess accumulation of fluid is called pleural effusion.
  - Pericardial cavity - Excess accumulation of fluid is called pericardial effusion.
  - Synovial cavity - Excess accumulation of fluid is called synovial effusion.



- Anasarca – Severe generalized edema is called anasarca. It is usually seen in nephrotic syndrome.
- Primary lymphedema – Edema due to malformation of lymphatics.
- Starling postulate- Fluid movement between extracellular space and blood vessels depends on balance of hydrostatic and osmotic pressure.



- Fluid comes out from vessels due to
  - Hydrostatic pressure in blood vessels
  - Colloidal osmotic pressure (COP) in interstitial fluid
- Fluid enters in blood vessels due to
  - Tissue tension
  - Colloidal osmotic pressure of plasma (80% COP is maintained by albumin)



- Tissue where fluid easily accumulates
  - Eyelids
  - Scrotum
  - Ankle etc.
- Tissue where fluid does not easily accumulates
  - Palm
  - Sole
- Water moves
  - From fluid of high hydrostatic pressure to low hydrostatic pressure
  - From low colloidal osmotic pressure to high colloidal osmotic pressure.



## Hepatic Edema

- Edema in liver diseases is called hepatic edema.
  - Cirrhosis
  - Hepatitis
  - Carcinoma of liver etc.

- Pathogenesis of edema in cirrhosis
  - Portal hypertension
    - Contraction of liver due to extensive fibrosis in cirrhosis
    - Portal vein compression at porta hepatis.
    - Increased hydrostatic pressure in portal system.
    - Transudation.
    - Accumulation of fluid in peritoneal cavity and ascites.



- Hypoalbuminemia
  - Reduction of hepatic cell mass in cirrhosis
  - Decreased albumin synthesis
  - Nutritional hypoalbuminemia
  - Transudation and generalized edema
- Lymphatic obstruction at porta hepatis
  - Compression of porta hepatis
  - Impaired lymph drainage
  - Enhancement of ascites



- Secondary hypertension
  - Reduction of hepatic cell mass
  - Delayed aldosterone metabolism. Thus half life of aldosterone is increased.
  - Aldosterone acts upon renal tubules and absorption of sodium and water.
  - Increased volume of blood
  - Increased hydrostatic pressure
  - Transudation and generalized edema.



## Cardiac Edema

- Edema due to heart disease is called cardiac edema.
- Example – Right-heart failure/Congestive cardiac failure (CCF)



- Pathogenesis of cardiac edema
  - Increased hydrostatic pressure
    - Right heart failure causes impaired venous return
    - Increased blood volume in venous system.
    - Increased hydrostatic pressure in blood.
    - Transudation and generalized edema.



  - Salt and water retention
    - Impaired venous return
    - Decreased cardiac output
    - Decreased renal blood flow -> Renin secretion from juxtraglomerular system.
    - Activation of renin angiotensin axis.
    - Secretion of aldosterone -> absorption of sodium and water from renal tubules.
    - Increased blood volume.
    - Increased hydrostatic pressure in blood.
    - Transudation and generalized edema.

## Renal Edema

- Edema following renal diseases is called renal edema.
- Renal diseases associated with edema
  - Nephritic syndrome (Syndrome in AGN)
  - Nephrotic syndrome



- Pathogenesis of edema in nephritic syndrome
  - Proteinuria in nephrotic syndrome
    - Mild to moderate proteinuria
    - Hypoalbuminemia
    - Decreased colloidal osmotic pressure
    - Transudation and edema



- Oligouria in nephritic syndrome
  - Retention of salt and water in blood.
  - Increased volume of blood
  - Increased hydrostatic pressure in blood
  - Transudation and edema



- Pathogenesis of edema in nephrotic syndrome
  - Massive proteinuria or heavy proteinuria
    - Hypoalbuminemia
    - Decreased colloidal osmotic pressure in blood.
    - Transudation and edema
    - Massive or heavy proteinuria
      - » Passage of protein (albumin) > 3.5 gm in 24 hours.



## Clinical Importance of Edema

- Cardiac edema – 1st appear in dependent part, like, ankle.
- Renal edema - 1st appear in face
- Hepatic edema - 1st appear in abdomen [ascites]



## Angioedema

- Edema in Type-I and Type-III hypersensitivity reaction.
- Edema involves in dermis and subcutaneous tissue.

## Pulmonary Edema

- Accumulation of fluid in lung alveoli is called pulmonary edema.
- Causes
  - Left ventricular failure
  - Cerebral damage etc.



## Hypoxia in pulmonary edema

- Fluid in alveolar spaces cause scarcity of gas in alveoli.
- Blood in interalveolar septal capillaries can not obtain sufficient oxygen thus cellular hypoxia throughout the body.



## Cerebral Edema

- Brain is susceptible to edema , because of
  - Little room to expand
  - Absence of lymphatics to carry away any excess fluid that accumulates.
- Types
  - Vesogenic edema – loss of blood brain barrier function results from damage of capillaries or newly formed capillaries that have not yet established barrier causes edema.



- Example of vesogenic edema – edema in
  - Primary and metastatic tumors of brain
  - Brain abscess
  - Infarction of brain etc.
- Cytotoxic edema
  - Accumulation of fluid in cells of gray matter.
  - Example – edema in ischemic brain



- Interstitial edema
  - Example – Edema in noncommunicating type hydrocephalus. Fluid comes out across the ependymal lining of ventricles and accumulates in the periventricular white matter.

*Cerebral edema is more dangerous, because, the cerebrum may herniate through foramen magnum and patient may die.



## Infarct and Infarction

- Localized area of ischemic necrosis either due to sudden arterial occlusion or impaired venous return is called infarct and the phenomenon is called infarction.
- Types
- Based on presence or absence of infarction
  - Septic infarct – presence of infection in infarct.
  - Bland infarct - absence of infection in infarct.

- Based on color
  - Pale or anemic infarct
  - Red or hemorrhagic infarct

  Most common cause of infarct
  - Arterial occlusion, caused by thrombus or embolus



- Effects of abnormal blood flow
  - Arterial dilatation causes increased blood flow. It is called hyperemia.
  - Sudden arterial occlusion causes infarction.
  - Diminised arterial blood flow cases atrophy



- Characteristics of pale infarct
  - Caused by arterial occlusion
  - Occurs in solid organs – heart, kidney, liver, spleen etc.
  - Edema usually absent.
- Atheroma and myocardial infarction
  - Superimposed thrombus formation on coronary artery atheroma -> occlusion of artery and ischemic necrosis.



- Characteristics of red infarcts
  - Caused by impaired venous return
  - Occurs in loose and previously congested tissue, like
    - Intestine (in strangulated inguinal hermia, intestinal obstruction)
    - Lung
    - Testis (in testicular torsion)
    - Overy (in twisting of ovary)
    - Valva etc.
  - Usually, edema present.



- Impaired venous return and infarct
  - Impaired venous return causes stasis of blood in capillaries -> gradual diminishing of arterial blood flow -> ischemia and infarction.
- Factors influencing infarction
  - Oxygen contents of blood.
  - Rate of development of arterial occlusion
  - Nature of vascular supply
  - Tissue vulnerability to hypoxia
  - Cardiorespiratory status of individual



- Oxygen content of blood
  - Diminished blood oxygen enhences infarction. Causes are
    - Hypoxic condition of individual
    - Anemia
    - Carbon monoxide poisioning
    - Chronic obstructive pulmonary diseases (COPD)
    - Heart diseases

- Rate of develpment of arterial occlusion
  - Rapid rate of arterial occlusion enhances infarction.
- Nature of Vuscular supply
  - Tissue with dual blood supply is less sensitive to infarction, exaples
    - Lungs [bronchial and pulmonary artery]
    - Liver [hepatic artery and portal vein]
    - Hand and forearm [radial and ulner artery]



- Tissue vulnerability of hypoxia, examples
  - Brain is more susceptible to hypoxia and can not tolerate hypoxia more than 5 minues.
  - Heart, kidney, liver, adrenal gland can tolerate up to 30 minutes.
  - Skeletal muscle can tolerate for 2 hours.



- Cardio-respiratory status of individual
  - Pre-existing cardiac or pulmonary disease enhences infarction.
- Shape of infarct
  - Wedge shaped – apex towards occlusion and base towards periphery of organ
- Reperfusion injury
  - It means reversibly injured cells of ischemic tissue may undergo irreversible injury following reperfusion.
  - In irreversible cell injury calcium derived from mitochondria activates lysosomal emzymes and cause damage of cells.



- If the ischemic zone – like myocardial infarction – is reperfused
  - Reoxygenation following reperfusion causes increased generation of oxygen derived free redicals from parenchimal cells, endothelium and infiltrating leucocytes, and cause cell damage.
  - Blood calcium enters early in cells due to abnormal permeability of reversible injured cells. Calcium activates lysosomal enzymes and causes irreversible cell injury.
  - Again blood calcium in dying cells causes hypercontruction of myofibrils and leads to contraction band necrosis.



## Hyperemia and Congestion

- Hyperemia
  - Increased volume of blood in an effected tissue or part of tissue due to arteriolar dilatation is called hyperemia.
  - Mechanism: It is an active process. Arterial or arteriolar dilatation by sympathetic neurogenic stimulation or vesoactive amines – like histamine and serotonin –> Increased flow of blood in capillaries -> Tissue become red.



- Example of hyperemia
  - Hyperemia in fever. Skin become red due to hyperemia. Following hyperemia radiation of temperature from blood to atmospherere and temperature falls in fever.
  - Hyperemia in exercise

*Hyperemia is helpful for us.

- Congestion
  - Increased volume of blood in an affected tissue or part of tissue due to impaired venous return is called congestion.
  - Mechanism: It is passive process. Impaired venous return -> stasis of blood in capillaries -> causes decreased arterial flow -> tissue become blue (cyanosis) due to deoxygenated blood.



- Causes of congestion
  - Congestive cardiac failure (CCF) leads to entire body congestion.
  - Impaired venous return in limb leads to localised congestion
    - Examples
      - Following long journey
      - Long period of standing



- Changes in lungs in congestion
  - In acute congestion following myocardial infarction fluid leaks in alveolar spaces, termed wet lungs
  - In chronic congestion
    - Dilatation and rupture of septal capillaries
    - Minutes intra-alveolar hemorrhage
    - Breakdown of red cells.
    - Engulfment of iron of red cells by the alveolar macrophages and formation of hemosiderin laden macrophage, called heart failure cell. Heart failure cell is found in alveolar septum.
    - When alveolar septum become thick due to fibrosis and hemosiderin pigments, is called brown indurations.



- Color of skin in
  - Hyperemia – Red
  - Congestion - Bluish



- Changes in liver in congestion
  - Nutmeg Liver
    - Distended central vein with blood, congested red zone of hepatic lobule around central vein, with uncongested pink rest zone around around red zone is called Nutmeg liver.
  - Central hemorrhagic necrosis
    - Following severe cardiac failure necrosis of central hepatocytes of hepatic lobule with hemorrhage around central vein, called central hemorrhagic necrosis.



- Cardiac cirrosis
  - Following long standing chronic congestion death of central hepatocytes of hepatic lobule and fibrosis called cardiac cirrhosis.
- Changes in spleen in congestion
  - Enlarge spleen in acute congestion
  - Fibrosis and hemosiderin causes siderofibrotic nodule following chronic congestion.

*Hemosiderin – Iron obtained from red cell breakdown form hemosiderin.

## Hemorrhage

Extravasation of blood is called hemorrhage

- Types
  - Petechae : Minute pin point hemorrhage is called petechae or petechial haemorrhage. It occurs in
    - Skin
    - Mucosa
    - Serous surfaces
  - Purpura: hemorrhagic spot larger than petechae [up to 1 cm] is called purpura. Found in ITP.



- Echymoses: Large and blotchy [eruption like] haemorrhagic spot is called echymoses.
- Hematoma: Accumulation of significant amount of blood in tissue is called hematoma.
- Hemothorax: Accumulation of blood in pleural space is called hemothorax.
- Hemoperitoneum: Accumulation of blood in peritoneal space is called hemoperitoneum.
- Hemopericardium: Accumulation of blood in pericardial space is called hemopericardium.



## Thrombus and Thrombosis

- Solid or semisolid mass formed by the constituents of blood is an uninterupted cardiovascular system is called thrombus and the phenomenon or the process of thrombus formation is called thrombosis.
- Types: According to site of formation
  - Cardiac thrombus: Originates in heart chamber
  - Venous thrombus: Originates in vein
  - Arterial thrombus: Originates in artery



- Types: According to nature
  - Mural thrombus
    - Heart chamber
    - Great vessels
  - Occlusive thrombus
  - Propagating thrombus
  - Saddle thrombus
  - Coraline thrombus
  - Consecutive clot thrombus

  *Fragmentation of tail of propagating thrombus usually leads to embulus.



- Thrombus does not occur in blood because
  - Balance prothrombotic and antithrombotic and antithrombotic role of endothelium.
  - Presence of natural anticoagulant – heparin.
  - Continuous blood flow
    - Prevent platelet aggregation
    - Dilute coagulation factors
    - Brings inhibitor of coagulation factor



- Difference between blood clot and thrombus
  - Clot occurs in non-streaming blood.
  - Example: Blood clot in test tube, glass slide and after death.
  - Clot in in streaming blood is thrombus.

- Thrombotic role of endothelium
  - Produces von Willebrand factor - that makes bridges between endothelium and platelets during thrombosis.
  - Synthesizes tissue factor – that activates extrinsic clotting cascade.
  - Secretes inhibitor of plasminogen (fibrinolysin) activator and prevents fibrinolysin activation.



- Antithrombotic role of endothelium
  - Secrets prostacyclin ($PGI_2$) and nitric oxide that inhibits platelet aggregation.
  - Secretes heparin like molecules.
  - Secrets tissue plasminogen activator.



- Virchow's triad of thrombus
  - Changes in endothelium [endothelial injury]
  - Changes in normal blood flow
  - Changes in constituent of blood [hypercoagubility of blood]



- Causes of changes in endothelium / Endothelial injury
  - Direct endothelial injury
    - Traumatic vascular injury
    - Inflammatory vascular injury [vasculitis]
  - Myocardial infarction
  - Bacterial toxin
  - Hemodynamic stress of hypertension
  - Hypercholesterololemia
  - Products absorbed from cigarette smoke – nicotine
  - Snake venom etc.



- Causes of alteration in normal blood flow [turbulence]
  - Atherosclerosis
  - Myocardial infarction
  - Mitral stenosis
  - Aneurysm
  - Hyperviscocity of blood
    - Polycythemia
    - Multiple myeloma



- Mechanism of thrombosis following turbulence and stasis of blood
  - Loss of laminar blood blow – orientation of platelets on endothelial surface.
  - Prevention of dilution of coagulation factors
  - Retardation of inflow of clotting factor inhibitor -> thrombus formation.

- Causes of hypercoagubility of blood
  - Primary hypercoagubility [genetic disorder]
  - Secondary hypercoagubility
    - Prolonged bed rest or immobilization
    - Tissue damage following
      - Big surgery
      - Burn etc.
    - Myocardial infarction
    - Disseminated cancer [cancer cell produces procoagulant]
    - Smoking [unknown cause]
    - Oral contraceptives



- Common sites of thrombus formation
  - Vein [venous or phlebothrombosis]
    - Deep calf vein thrombosis
    - Popliteal vein
    - Internal iliac vein etc.

    *Tend to grow in the direction of blood flow i.e. towards heart.
  - Arteries
    - Coronary artery
    - Cerebral artery
    - Femoral artery etc.

    *Usually superimposed on atherosclerotic plaque or occurs following arteritis and tend to grow in retrograde direction of blood flow



- Thrombogenesis
  - Endothelial injury and exposure of subendothelial collagen.
  - von Willebrand factor secreted by endothelium makes bridge between endothelium and collagen. It has two receptor, one for collagen and another for platelets.
  - After adhession of platelets with collagen dense bodies of plate lets releases
    - Calcium: needed for coagulation cascade and
    - ADP: needed for further platelet aggregation.



- Aggregated platelets form platelet plaque.
- Activation of intrinsic coagulation pathway on platelet plaque and formation of fibrin thread.
- Platelets and fibrin make platelet-fibrin plaque and formation of thrombus.



- Fate of thrombus
  - Either attached at the site of origin
  - Or detached from the site of origin
- Fate of attached thrombus
  - Dissolution by fibrinolysin
  - Propagation: Small thrombus may propagates -> become larger -> occludes vessels.
  - Recanalization
  - Organiztion: Inflammation induced by thrombus causes fibrosis and incorporation of thrombus in vascular wall called organization.



- Fate of detached thrombus
  - Detached from the site of origin and transported by blood as an embolus.
  - It is the most common type of embolus and is called thromboembolus.

## Embolus and Embolism

- Solid, liquid or gaseous mass transported by blood from its point of origin or from its point of entry to a distant place and is impacted. The mass is called embolus and the process of impaction is called embolism.
- Types
  - Solid
  - Liquid
  - Gaseous embolus



- Systemic arterial embolism
  - Embolus travels within arterial circulation and impacts in arteries is systemic embolism.
  - Effects
    - Myocardial infarction following coronary artery occlusion by embolus.



- Paradoxical embolus
  - Embolus originating at venous side, enters arterial side along pressure gradient is called paradoxical embolus and the impaction is called paradoxical embolism.
  - Causes
    - Interatrial septal defect
    - Interventicular septal defects
    - Patent ductus arteriosus [that connect aorta with pulmonary trunk in intrauterine life]-associated with pulmonary hypertension.



- Air/Gas Emboli
  - Gas bubbles may cause vascular occlusion and ischemia in gas embolism.
  - May enter into circulation following
    - Chest wall injury in road traffic accident
    - During obstretric procedure
  - Amount of gas needed for embolism is more than 100 cc.



- Decompression sickness
  - Sickness following a particular form of gas embolism caused by sudden changes in atmospheric pressure is called decompression sickness.
  - Affected individual
    - Deep sea diver
    - Underwater construction worker
    - Mine workers etc.



- Pathogenesis
  - Under compression [high pressure] -> liquefaction of nitrogen of breathed air in blood and tissue of sea diver or other workers.
  - Liquefied nitrogen becomes bubble following decompression [release of pressure] -> when diver ascends rapidly at sea level.
  - Bubbles of gaseous nitrogen forms gas embolism.

- Types of decompression sickness
  - Acute or bends: Characterized by
    - Severe pain due to ischemia due to gas embolism in skeletal muscles and tissue in and around joint.
    - Severe pain causes arching of affected individual. Due arching it is called bend.
  - Treatment
    - Individual is placed in compression chamber to make the bubbles of nitrogen liquid. Subsequently, slow decompression is allowed so that bubbles do not reform to make an embolus.



- Chronic decompression sickness or caisson disease
  - Persistent gas emboli in skeletal system causes ischemic necrosis in head of
    - Humerus
    - Tibia
    - Femur

  *Chokes: Sometimes, gas emboli may cause respiratory distress, called chokes. It results form pulmonary hemorrhage and focal atelactasis of lung following pulmonary gas embolism.



## Shock

- Widespread hypoperfusion of tissue due to disparity between cardiovascular system and blood volume that may threatened life is called shock.
- End result of shock
  - Hypotension, followed by impaired tissue perfusion and cellular hypoxia.



- Types of shock
  - Hypovolumic shock
  - Cardiogenic shock
  - Septic shock
  - Neurogenic shock
  - Anaphylactic shock



- Hypovolumic shock [Hypovolumia of blood]
  - Causes of hypovolumia
    - Acute blood loss following road traffic accident, during delivery etc.
    - Diarrhea
    - Vomiting
    - Extensive burn
    - Extensive trauma



- Cardiogenic shock: due to cardiac diseases
  - Myocardial infarction
  - Endocarditis
  - Ventricular arythmia etc.
- Septic shock
  - Most common causes: Gram-negative organism
    - E. coli
    - Klebsiaella
    - Enterobacter
    - Pseudomonus
    - Bactaroids etc.

- Less common causes: Gram-positive organisms
  - Streptococci
  - Pneumococci etc.
- Endotoxic shock: Lipopolysaccharide (LPS) is an endotoxin derived from gram-negative organism is associated with shock. Thus septic shock is called endotoxic shock.
- Peptidoglycan present in cell wall of Gram-positive organism is also associated with septic shock.



- Pathogenesis of septic shock: LPS or peptidoglycan causes
  - Injury to endothelium -> increased vascular permeability -> Reduction of effective circulatory blood volume.
  - Injury to leucocytes -> Release of toxins -> further endothelial injury and reduction of blood volume.
  - Injury to platelets -> release of toxin -> further endothelial injury and reduction of blood volume.
  - Activation of complement system by the alternate pathway -> production of vasoactive C3a and C5a -> vasodilatation and increased vascular permeability -. Reduction of effective circulating blood volume.



- Vasoactive amines, histamine and serotonin released from the injured endothelium, leucocytes and platelets and vasoactive complement products, C3a and c5a causes
  - Vasodilatation and increased vascular permeability. Peripheral pooling of blood following vasodilatation and hypovolumia following increased vascular permeability, decreased effective circulating blood volume -> shock.



- Consumption coagulopathy
  - DIC (disseminated intravasular coagulation) is characterized by numerous thrombi formation throghout the vascular system followed by bleeding. As bleeding occurs following consumption of platelets and coagulation factors thus called consumption coagulopathy.



- Neurogenic shock
  - Spinal cord injury
  - Anesthetic accident etc.

  Loss of vascular tone->peripheral polling of blood -> reduction of effective circulatory blood volume -> disparity between cardiovascular system and blood volume -> shock.



- Anaphylactic shock: IgE mediated hypersensitivity reaction
  - Systemic vasodilatation and
  - Increased vascular permeability

  Thus reduction of effective circulatory blood volume -> disparity between cardiovascular system and blood volume -> shock.

- Stages of shock
  - Early compensated stage
  - Progressive decompensated stage
  - Irreversible stage



- Early compensated stage
  - Body can compensate and recover from shock without supplement by infusion or transfusion.
  - Mechanism of compensation and recovery
    - Following blood loss
      - Peripheral vasoconstriction
      - Decreased cardiac output
      - Decreased renal blood flow
      - Renin secretion from juxtaglomerular apparatus and activation of renin angiotensin axis.
      - Secretion of aldosterone and absorption of sodium and water from renal tubules.



- Activation of renin-angiotensin-aldosterone axis and increased ADH secretion following hypovolumia leads to increased blood volume and recovery from shock.



- Progressive decompensated stage
  - Body tries to compensate and recovered from shock but can not recover without supplement by infusion and transfusion.
  - Mechanism
    - Following blood loss
      - Peripheral vasoconstriction
      - Decreased cardiac output
      - Decreased renal blood flow
      - Renin secretion from juxtaglomerular apparatus and activation of renin angiotensin axis.
      - Secretion of aldosterone and absorption of sodium and water from renal tubules.



- Activation of renin-angiotensin-aldosterone axis and increased ADH secretion following hypovolumia leads to increased blood volume and recovery from shock. Patient only recovers when supplemented by infusion or transfusion.



- Irreversible stage
  - Body tries to compensate. Infusion or transfusion given but no recovery.

- Morphological changes in tissue in shock
  - Hypoxia following shock causes injury in all tissue, but main threats to life from in juries are
    - Brain
    - Heart
    - Lungs
    - Kidney
  - Brain: Ischemic liquefaction necrosis
  - Heart: Ischemic coagulation necrosis
  - Lungs: Diffuse alveolar damage and adult respiratory distress syndrome (ARDS)
  - Kidney: Acute tubular necrosis (ATN).



Thanks