Inflammation, Acute Phase Response, Fever, Hyperthermia.

Case 1.

Patient C., 30 years old, is delivered to a medical aid post in a poor condition. His skin and mucosa are cyanotic, the pulse is weak,146/min; blood pressure is 90/60 mm Hg. The breathing is rapid and shallow, the temperature - 40.6˚C. The man's colleagues say that he has been working for 40 minutes at air temperature 70˚C and high humidity, liquidating an industrial accident.

1. What pathological process caused the temperature increase?
2. What is the pathogenesis of the symptoms?
3. What stage (phase) of the pathological process does the patient have?
4. Is it advisable to give the patient antipyretics? Why?

Case 2.

Patient B., 47 years old, after an operation for a thyroid gland tumor removal developed symptoms of hypothyroidism (thyroid gland insufficiency). She was prescribed thyroxin. Her condition improved, and she increased the dose of the medication without consulting her doctor. Some time later, she began to complain of insomnia and palpitations, her body temperature increased to 37.5 – 37.7˚C.

1. Can we regard the body temperature increase in this patient as a fever?
2. How can we explain an increase in body temperature in excessive intake of thyroxin?
3. Is it advisable to administer aspirin to this patient? Why?
4. What is the difference between fever and hyperthermia?

Case 3.

Patient N., 27 years old, was delivered to hospital in a condition of psychomotor excitation after an accident at the building grounds where he had fallen down from a considerable height. On examination: the patient is pale, nystagmus is present, body temperature is 37.7˚C; there are contusions of the soft parts of the body, but no fractures. A diagnosis: brain concussion.

1. Explain a possible pathogenesis of the temperature rise in this patient.
2. Is it reasonable to give aspirin to this patient? Why?
3. List the negative consequences of fever.
4. What is lytic and critical decrease of body temperature? Why is critical decrease of body temperature in the case of fever dangerous?

Case 4.

Natasha K., 6 years old, was admitted to hospital with a diagnosis "infectious inflammation of the parotid glands (mumps)". The disease began with general malaise and gradual increase in the body temperature which reached 39˚C. The high temperature persisted for ten days. The difference between the temperature in the morning and in the evening did not exceed 1˚C. The patient developed weakness, drowsiness and poor appetite. 10 days later the temperature began to decrease gradually which was accompanied by intensive sweating.

1. What typical pathological processes were observed in this patient?
2. Explain their interrelation.
3. What type of temperature curve was found in this patient?
4. What degree of temperature rise was observed in this patient?
5. Explain the mechanism of the development of weakness, drowsiness and poor appetite.

Case 5.

Patient K., 18 years old, is admitted to an internal disease department with croupous pneumonia. His temperature is 40.5˚C, he is pale, the skin is dry. The tongue is white and coated. The patient complains of headache, complete loss of appetite, drowsiness, severe cough with sputum, dyspnea, soreness of muscles and joints. His blood pressure is 130/90 mm Hg, pulse - 98/min. The heart boundaries are normal, heart sounds are muffled. The breathing is rapid and shallow. Crepitation is heard in the lower part of the left lung. The liver is slightly enlarged.
Blood test: leukocytes – 18 x 10^9/l, neutrophil leukocytosis; erythrocyte sedimentation rate (ESR) – 22 mm/h. Blood sugar is 7mmol/l, albumin/globulin (A/G) coefficient is decreased.

1. Make a pathogenetic chain characterizing the mechanism of temperature rise in this patient.
2. What degree of temperature rise is observed in this patient?
3. Explain the connection between the inflammatory process in the lungs and general reactions of the organism.
4. What inflammatory phenomenon can crepitation be associated with?
5. Explain the mechanism of tachycardia, neutrophilia, hyperglycemia and a decrease in A/G coefficient.

Case 6.

A patient with a confirmed diagnosis of cancer disease had been running a subfebrile temperature for a year. After administration of medications suppressing protein synthesis his temperature returned to normal.

1. What pathological process caused the rise of temperature in this patient (infectious fever, noninfectious fever, endogenous hyperthermia)?
2. How can you justify your conclusion?
3. What is the mechanism of antipyretic action of medications suppressing protein synthesis? Can they normalize temperature in the case of endogenous hyperthermia?
4. Point out possible mechanisms of weight loss in this patient.

Case 7.

An ambulance was called to patient T., 43 years old, because of a renal colic. The patient complains of colicky pains in the right lumbar region radiating to the right testicle. The pain is so severe that the patient nearly faints. He has frequent micturition. In the urine newly formed erythrocytes are present; body temperature is 37.7˚C. The patient has a history of renal calculi.

The pains disappeared after an injection of morphine with atropine and a hot-water bottle on the loin. Two hours after the colic, the body temperature returned to normal.

1. Explain a possible mechanism of the temperature rise in this patient.
2. What degree of temperature rise was observed in this case?
3. What is fever?
4. What changes in the cell and protein composition o f the blood are observed in the case of acute phase response?

Case 8.

Physical examination of the patient revealed a temperature increase to 37.8 ˚C. She has a history of persistent subfebrile temperature over the last year. Taking antipyretic medications (such as aspirin) does not lead to temperature normalization.

1. What pathological process (fever or hyperthermia) takes place in this case?
2. What causes can lead to this process?
3. What additional data should be obtained to make an accurate diagnosis?
4. Name the endogenous pyrogens (3) and list their main properties.
5. Give a brief characteristic of the stages of hyperthermia.

Case 9.

A child of 8 months of age was taken to hospital with a confirmed diagnosis of an infectious disease. Physical examination revealed a temperature rise up to 39.7˚C.

1. Explain the pathogenesis of the child's temperature rise.
2. What complications associated with high temperature can develop in this case?
3. Is it advisable to use methods of physical cooling in such cases? Why?
4. What is the difference between fever and hyperthermia?
5. What changes in the protein composition of blood plasma will be observed in this child?

Case 10.

A patient, aged 20, with a confirmed diagnosis of an infectious disease at the moment of examination has a temperature 38.7 ˚C.

1. Is infectious fever an apparent indication for administration of antipyretics such as aspirin?
2. How can you justify your opinion?
3. What additional data should be obtained to decide whether administration of antipyretics is advisable in this case?
4. Is it advisable to use physical methods of cooling for treatment of infectious fever and if yes, in what cases?
5. Explain the mechanism of temperature rise in the case of fever.

Case 11.

A 42-year-old man with thyrotoxicosis has a history of constant subfebrile temperature over the last year. 5 days ago a diagnosis of pneumonia was made. The patient's temperature at the examination is 38.77 ˚C; he has neutrophil leukocytosis and increased erythrocyte sedimentation rate (ESR).

1. Can we regard a temperature increase in this case as a manifestation of infectious fever?
2. Can administration of antipyretics (aspirin) normalize temperature in cases like this?
3. Is administration of antipyretics advisable?
4. Explain the difference in the mechanisms of development of fever and endogenous hyperthermia.
5. Explain the mechanism of the development of neutrophil leukocytosis and ESR increase in this patient.

Case 12.

An experiment was done on two narcotized rats. The first rat was subcutaneously injected 0.1 ml of ***histamine*** into the left hind leg and 0.1 ml of ***histamine*** into the right hind leg on the background of previously injected ***dimedrol*** (0.1ml). The second rat was injected 0.1 ml of ***turpentine*** into the left hind leg, and 0.1 ml of ***turpentine*** into the right hind leg on the background of previously injected ***dimedrol*** (0.1 ml).

Results: The first rat: 30 minutes after the injections the left leg was 1.5 times enlarged in volume, hyperemic and warm. The right leg was insignificantly enlarged, cold and pink in color. 2 hours later all visible changes in both legs disappeared. The second rat: 30 minutes after the injections the left leg was 1.5 times enlarged in volume with accompanying redness and rise of temperature. Similar changes in the right leg were less pronounced. 2 hours later both legs enlarged in volume twice, both were hyperemic, hot and jerked periodically.

1. What pathological process developed in the first and in the second rat?
2. Explain the difference in the dynamics of the edema development in these two rats.
3. What general changes can develop in the organism of a rat on the background of turpentine injection?
4. List the mechanisms of edema development in the case of inflammation.

Case 13.

Patient B., aged 22, and patient K., aged 43, were found to have fluid accumulation in the pleural cavity. Both patients underwent a pleural puncture.

Patient B.: the punctate was cloudy, of light-yellow color with a relative density 1.029, protein content 39 g/l and high activity of lactatedehydrogenase (LDG). In the sediment there were numerous formed elements, predominantly neutrophils of degenerative forms. Microbe flora was present inside and outside the cells.

Patient K.: The punctate was transparent, of light-yellow color with a relative density 1.014, protein content 16 g/l and low activity of LDG. In the sediment there was insignificant amount of cells, mostly lymphocytes.

1. What is the character of the fluid in patient B. and in patient K.? Justify your answer.
2. Describe the main differences in the fluid composition of patient B. and patient K.
3. What are possible mechanisms of fluid accumulation in the pleural cavity of these patients?
4. Describe the mechanism of leukocytes appearance at the site of inflammation.

Case 14.

An experiment was carried out on three narcotized rats: in the hind leg of each animal 0.1 ml of turpentine (a powerful phlogogenic agent) was injected. The first rat was intact, the second was previously injected 0.1 ml of dimedrol into the same leg, the third was previously injected 0.1 ml of hydrocortisone into the same leg. The volume increase of the legs was assessed 30 minutes, 1 hour and 2 hours after the injection.

Results (volume increase)

	30 minutes	1 hour	2 hours
Rat 1 (intact)	10%	40%	90%
Rat 2 (dimedrol)	2%	35%	90%
Rat 3 (hydrocortisone)	5%	15%	40%

1. Explain the mechanism of an increase in the volume of the rats' legs.
2. Explain the difference in the dynamics of volume growth in these three rats.
3. Explain the mechanism of anti-edematous action of dimedrol.
4. Explain the mechanism of anti-edematous action of hydrocortisone.

Case 15*.

In the alteration phase in the focus of inflammation there is a marked increase in highly active enzymes: elastase, collagenase, hyaluronidase, phospholipase A_2, myeloperoxidase and others.

1. Which of these enzymes induces increased formation of prostaglandins?
2. Describe the role of prostaglandins in the focus of inflammation.
3. What other inflammation mediators are formed after activation of this enzyme? List their main properties.
4. How can increased production of this enzyme be blocked?

Case 16.

Two patients were delivered to the admitting office of a hospital with acute pain in the ileac area of the abdomen. Their blood tests were made with diagnostic purposes.

Patient A.: no deviations from the norm were found in the patient's blood. The body temperature was 36.8 ˚C. After administration of spasmolytics the pain disappeared and the patient went home.

Patient B.: neutrophil leukocytosis with the left shift and an increased erythrocyte sedimentation rate (ESR) were detected, body temperature was 38.2 ˚C. The patient was taken to the operating block for further evaluation and possible surgical intervention.

1. What pathological processes did patients A. and B. most likely have?
2. What is a possible pathogenesis of pain syndromes in patients A. and B.?
3. Explain the mechanism of leukocytosis and ESR increase in patient B.
4. Explain the mechanism of body temperature rise.

Case 17.

Two patients were delivered to the admitting office of a hospital with complaints of severe constricting pains in the chest radiating under the left scapula and to the left arm. Taking of validol was not effective. Both patients were administered analgesic and spasmolytic medications; blood tests and ECG were performed.

Patient K.: The pain disappeared within 30 minutes. His blood test was normal. ECG showed elevated symmetrical T-wave in the chest leads. The patient was allowed to go home and given a recommendation to visit a cardiologist.

Patient M.: The pain syndrome increased, body temperature rose to 37.7˚C. His blood test revealed neutrophil leukocytosis with the left shift, elevated ESR, presence of troponin in the blood. ECG showed the signs of acute myocardial infarction. The patient was hospitalized.

1. What pathological processes are most likely to be found in patients K. and M.?
2. What is the connection between patient M.'s infarction and changes in his blood?
3. Make a pathogenetic scheme of the mechanism of body temperature rise in patient M.
4. Explain the mechanism of pain syndrome in these patients.

Case 18.

Patient addressed to doctor with complaints on impairment of left eye's vision, swelling of eyelid and redness of left eye mucous, tearing, cramping of left eye. Two days ago during working in the country, foreign body got into his eye. In the morning, his eye was swollen, watery, and unable to open. During examination, eyelids are swollen, their edges were hyperemic, having purulent discharge.

1. Which typical pathological process is developed in the patient? Justify your opinion.

2. Explain the pathogenesis of mucosal redness and pus formation in the left eye.

Case 19.

A patient went to the doctor and complained that she had severe and occasionally "twitch" pain on the second finger of the right hand. Symptoms appeared on the second day after the patient did manicure and scratched her skin around the nail meanwhile she was doing manicure.

During the examination, doctor found out that her finger was swollen, especially around the nail, it appeared to be red, and hot was felt when touching. Patient complained that it is hard to bend her finger.

1. Which typical pathological process is developed in the patient?
2. What are the classical signs for this process?
3. Specify the local pain factors and explain their appearance.

Case 20.

A child accidentally touched a hot iron at forearm and cried when he felt the pain. On the skin at the site of contact occurred rapidly reddening and bubble filled with clear liquid.

1. What is the mechanism of pain in this case?
2. What are the mechanism of occurrence of mediators in the inflammation area?
3. What are the pathological reactions which can cause release of inflammatory mediators?

Case 21.

A patient was admitted into the surgical department of hospital, with the diagnosis acute abdomen (abdominal pain). Based on the local symptoms like high temperature and neutrophilic leukocytosis, this confirms the diagnosis of acute appendicitis. The patient was sent to the operation room. As a result of appendectomy, the appendix was removed, which was then developed into purulent inflammation.

1. What is the mechanism of formation of purulent exudate?

2. How does purulent exudate fundamentally differ from serous exudate?

3. What complications can arise during the formation of purulent focus in the tissue?

Case 22.

Patient B, is diagnosed with "tuberculosis of lungs" in the lung tissue, found foci of caseous necrosis surrounded by macrophages, lymphocytes, epithelioid cells, and multinucleated cells of Pirogov-Langerhans.

Patient D, is diagnosed with "lobar pneumonia", caused by streptococcus pneumonia, exudates are detected in alveoli of the infected lung, which contain neutrophils, isolated erythrocytes and fibrin.

1. Name the type of inflammation of lungs developed in patients B and D. Justify your answer.

2. What are the causes of this type of inflammation suffered by patient B? What are the other reasons of the inflammation that you know?

3. What kind of exudates are formed in the lungs of patient D, what is the mechanism of its formation?

Case 23.

A child accidentally touched a nettle (plants with stinging hairs that irritate the skin on contact) with his forearm and cried out in pain. On the skin at the site of contact quickly developed redness and blisters that disappeared in 25-30 mins. It is known that in the stinging hairs of the nettle contains histamine, choline, formic acid, etc. They get into the skin through the wound, which is formed by pricking the skin with sharp hairs.

1. Can the observed changes be considered as manifestations of inflammation? Why?

2. Is it possible to stop inflammation in the early stages of development?

3. What is the role of histamine in the mechanisms of cutaneous manifestations?