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Department of Pathological Anatomy

Chronic inflammation. Granulamatosis.

1. **Purpose**: To study the causes, pathogenesis and significance for the body of the main types of chronic inflammation.
2. **The student needs to know**:
1. Terms - interstitial inflammation, granulomatous, with the formation of polyps and genital warts, monocytic phagocytes, epithelioid cells, giant Pirogov-Langhans cells, caseous necrosis, granuloma, productive reaction.
2. Etiology, pathogenesis, morphogenesis, the main types of granulomatous inflammation.
3. Principles of classification of chronic inflammation.
4. Fundamentals of clinical and anatomical analysis and principles of constructing a diagnosis in identifying signs of chronic inflammation.
The student must be able to
1. The concept of inflammation. The biological meaning of inflammation.
2. The etiology of inflammation. Inflammatory mediators.
3. Classification of chronic inflammation. Morphogenesis of productive inflammation.
4. Characteristics of interstitial inflammation, granulomatous inflammation, inflammation with the formation of polyps and genital warts.
5. Characteristics, outcomes of chronic inflammation.

Block of information on the topic:
Overview of Chronic Inflammation
Chronic inflammation can be defined as a long-term (over weeks and months) inflammatory process, in which there are simultaneously signs of active inflammation, tissue damage, and damage repair.
In contrast to acute inflammation, which manifests itself in noticeable alteration, vascular reactions, exudation, edema and severe neutrophil infiltration, in chronic inflammation, alteration is less pronounced. The process is characterized by a productive tissue reaction with infiltration of mononuclear cells (macrophages, lymphocytes and plasma cells), with foci of necrosis formed as a result of the activity of cells of the inflammatory infiltrate, as well as inadequate repair, antiogenesis and tissue sclerosis.
Chronic inflammation can develop as a result of acute inflammation. However, it is often chronic from the very beginning and a latent asymptomatic course for a long time.
The chronization of inflammation depends both on the characteristics of the cause that caused it, and on the individual characteristics of the response of the organism in which it develops.
Chronic inflammation can be caused by a variety of agents. It occurs in bacterial, viral, fungal infectious diseases (tuberculosis, syphilis, sepsis, diphtheria, typhus, chronic viral hepatitis, etc.), as well as in chronic exogenous and endogenous intoxications (dust and interstitial lung diseases, interstitial nephritis). However, in many chronic diseases with immune pathogenesis (idiopathic fibrosing alveolitis, Abramov-Fiedler myocarditis), the etiology of chronic inflammation remains unclear.
The morphology of chronic inflammation. Morphological changes in chronic inflammation reflect its main feature - a long course with varying degrees of activity. In this regard, there are 5 main signs of chronic inflammation:
• the predominance of a productive tissue reaction;
• the presence of secondary tissue damage caused by the cells of the very focus of chronic inflammation;
• features and weak severity of exudative tissue reaction, incomplete phagocytosis;
• mononuclear tissue infiltration, diffuse or focal, up to the formation of granulomas;
• sclerosis and persistent destruction of connective tissue.

Productive tissue reaction. Its predominance is not accidental, since inflammation becomes chronic when, for various reasons, there is no effective cell proliferation in the focus of inflammation, resulting in tissue repair.

Secondary tissue damage (alteration) caused by the cells of the very focus of chronic inflammation. Alteration in the form of necrosis and histolysis in chronic inflammation is induced by inflammatory mediators of plasma and cellular origin, accumulating in the inflammation focus.

Features and low severity of exudative tissue reaction. In chronic inflammation, exudative changes are, as a rule, insignificant and are represented mainly by the emigration of cellular elements and phagocytosis, which is often incomplete (for example, in granulomatous inflammation). Without the process of exudation, it is impossible to imagine the formation of cellular inflammatory infiltrates in foci of chronic inflammation and granulomas, since most of the cells of these infiltrates are of hematogenous origin. In certain types of chronic inflammation, exudation is pronounced, as, for example, in chronic catarrhal inflammation, chronic abscess, as well as in the infiltrative form of secondary pulmonary tuberculosis.

Mononuclear tissue infiltration, diffuse or focal up to the formation of granulomas. The main cells of mononuclear infiltration are cells of monocytic origin and lymphocytes. The traditional histological concept of "mononuclear cells, mononuclear cells" covers all mononuclear cells with non-segmented nuclei originating from a hematopoietic stem cell, except for erythrocytes and segmented leukocytes.

Sclerosis and persistent destruction of connective tissue. The most important sign of chronic inflammation is also the development of sclerotic changes with persistent destruction of connective tissue, which is a manifestation of a violation of the repair processes. Therefore, the restoration of destroyed tissues occurs by replacing damaged parenchymal cells with connective tissue, as a result of which fibrosis or scarring develops. This process is similar to wound healing, but due to the fact that the damage continues and the inflammatory response dies down and then resumes, events become less predictable.

In addition, in a number of cases with chronic inflammation, large thick-bodied blood vessels are detected, angiogenesis is noted. This symptom is especially clearly observed in productive inflammation with the formation of polyps and genital warts.

The outcomes of chronic inflammation are rarely favorable and are associated with the replacement of the inflammation focus with connective tissue. In most cases, there are various complications (secondary amyloidosis, cachexia, arrosive bleeding), sclerotic changes leading to organ failure, as well as malignant tumors.

Interstitial (interstitial) inflammation

Interstitial inflammation develops in the stroma of the parenchymal organs - the myocardium, liver, kidneys and lungs. It can be not only chronic, but also acute. Interstitial inflammation is characterized by a combination of productive and exudative tissue reactions with sclerotic changes.

The etiology of interstitial inflammation is diverse. It occurs in bacterial and viral infections with a severe course (sepsis, diphtheria, typhus, acute and chronic viral hepatitis, etc.), chronic exogenous and endogenous intoxications (interstitial lung disease, interstitial nephritis) and diseases of unknown etiology with immune pathogenesis (idiopathic fibrosing alveolitis, Abramov-Fiedler myocarditis).

At a macroscopic examination, the organs are slightly changed. It is possible to note a slight increase in their size, uneven vascular blood filling and a flabby consistency. Microscopically, focal or diffuse inflammatory cellular infiltration in the stroma of the myocardium, liver, kidneys and lungs can be detected. In the parenchyma of organs, pronounced dystrophic and sometimes necrobiotic changes are found. Similar changes are observed, for example, in pneumocytes of the 1st and 2nd orders in interstitial lung diseases, cardiomyocytes in Abramov-Fiedler myocarditis, in hepatocytes in viral hepatitis.

Sclerosis as a result of interstitial inflammation is caused by the activation of fibroblasts, which occurs as a result of initial damage due to inflammatory mediators, the production of growth factors by infiltrate cells, primarily macrophages, endothelial cells.

• **Outcomes of chronic interstitial inflammation**. In the end, connective tissue grows diffusely, which can lead to dysfunction of the organ. So, at the end of interstitial myocarditis, diffuse small-focal cardiosclerosis often develops, which underlies chronic heart failure. Interstitial lung diseases often lead to interstitial fibrosis with the formation of the so-called cellular lung and progressive pulmonary heart disease.

Granulomatous inflammation

Granulomatous inflammation is a form of chronic inflammatory reaction in which nodules and diffuse tissue infiltrates are formed; the predominant type of cells in infiltrates are cells of monocytic-macrophage origin: macrophages, epithelioid cells, giant multinucleated cells of foreign bodies and Pirogov-Langhans cells.

The etiology of granulomatous inflammation is diverse. Granulomatous inflammation of the established etiology is caused by both endogenous and exogenous factors, which in turn can be infectious and non-infectious in nature. Exogenous factors that cause the formation of granulomas include biological (bacteria, fungi, protozoa, helminths), organic and inorganic substances (dust, smoke, etc.), drugs (granulomatous hepatitis). Endogenous factors include sparingly soluble products of damaged tissues, especially adipose tissue (soap), as well as products of impaired metabolism, such as urates.

Among the infectious agents that cause granulomatous inflammation, banal and specific are distinguished (tuberculous mycobacterium, treponema pallidum, mycobacterium leprosy, rhinoscleroma bacillus).

However, in a number of cases, the etiology of gramulomatous inflammation remains unclear. Granulomas of unknown etiology include granulomas in sarcoidosis, Crohn's disease, primary biliary cirrhosis, etc.

Granuloma morphogenesis consists of 4 stages:

1) accumulation of monocytes in the focus of tissue damage;

the maturation of these cells into macrophages and the formation of macrophage granulomas;

2) transformation of macrophages into epithelioid cells and the formation of epithelioid cell granulomas;

3) transformation of epithelioid cells into giant cells (Pirogov-Langhansa and or foreign bodies) and the formation of giant cell granulomas.

Thus, given the composition of the granuloma, morphological characteristics distinguish 3 types of granulomas: macrophage (simple or phagocytoma); epithelioid and giant cell.

The classification of granulomatous inflammation takes into account its etiology, pathogenesis, course and morphological features. The following types of granulomatous inflammation are distinguished:

By etiology:

• unknown etiology;

• established etiology;

1) non-infectious,

2) infectious (commonplace and specific).

By pathogenesis:

• immune;

• non-immune.

By morphology:

• tuberculoid type (with the formation of granulomas, classified by cell composition and type of necrosis);

• diffuse type (with the formation of infiltrate).

Some granulomas of infectious etiology have a relative morphological specificity. Identification of the pathogen is required to confirm the diagnosis. Specific are those granulomas, the formation of which is associated with specific pathogens:

• mycobacterium tuberculosis;
• mycobacterium leprosy;
• pale treponema;
• a rhinoscleroma stick.
Specific granulomas are characterized by relatively specific morphological manifestations (only for these pathogens and not for any others), and the cellular composition, and sometimes the location of cells inside the granulomas (for example, in tuberculosis) is also quite specific.

Outcomes of granulomatous inflammation. The following outcomes are possible:
• resorption of cellular infiltrate;
• development of sclerosis;
• necrosis of granulomas;
• suppuration of granulomas.

Granulomatous diseases
This is a heterogeneous group of diseases (nosological forms) of various etiologies, the structural basis of which is granulomatous inflammation. These diseases are united by a number of symptoms:
• presence of granulomatous inflammation;
• violation of immunological homeostasis;
• polymorphism of tissue reactions;
• tendency to chronic course with frequent relapses;
• frequent vascular lesions in the form of vasculitis.

Classification of granulomatous diseases. It is based on the etiology of the disease.
Granulomatous diseases of established etiology.
1. Granulomatous diseases of infectious etiology: rabies, viral encephalitis, cat scratch disease, typhus, typhoid fever, paratyphoid fever, yersiniosis, brucellosis, tularemia, glanders, rheumatism, rhinosclerosis, tuberculosis, syphilis, leprosy, malaria, toxoplasmosis candidiasis, schistosomiasis, trichinosis, alveo coccosis.
2. Granulomatous diseases of non-infectious etiology: silicosis, asbestosis, talcosis, anthracosis, aluminosis, berylliosis, zirconia, bogassosis, byssinosis, amylosis.
3. Granulomatous drug diseases: granulomatous drug hepatitis, oleogranulomatous disease, gluteal granuloma of infants.
Granulomatous diseases of unknown etiology. These include sarcoidosis, Crohn's disease, Horton's disease, rheumatoid arthritis, primary biliary cirrhosis, Wegener's granulomatosis, Weber-Christian panniculitis, xanthogranulomatous pyelonephritis.

Lesson plan.

Explore and describe:

1. To study the interstitial (interstitial) inflammation by the microscopic picture. Describe the micropreparation "Autoimmune thyroiditis" (staining with hematoxylin and eosin). Pay attention to the cellular composition and localization of the inflammatory infiltrate, the amount of connective tissue elements in the myocardium / or thyroid tissue; assess the state of parenchymal cells in the area of inflammation, note the formation of lymphoid follicles with germinal centers in the thyroid gland.

1. To study the interstitial (interstitial) inflammation by the microscopic picture. Describe the micropreparation "Interstitial (interstitial) myocarditis" or "autoimmune thyroiditis" (staining with hematoxylin and eosin). Pay attention to the cellular composition and localization of the inflammatory infiltrate, the amount of connective tissue elements in the myocardium / or thyroid

tissue; assess the state of parenchymal cells in the area of inflammation, note the formation of lymphoid follicles with germinal centers in the thyroid gland.
2. To study the structure of the macrophage and the giant cell of Pirogov-Langhans using . Describe the patterns electronogramme "Macrophage of tissue infiltrate" and "Giant cell of Pirogov-Langhans". Pay attention to the state of ultrastructures: the number of lysosomes and phagolysosomes, the presence and nature of inclusions in the tubules of the cytoplasmic reticulum in both cells.
3. To study the productive inflammation around the animal parasite by the macroscopic picture. Describe the macropreparation "Echinococcosis (or alveococcosis) of the liver". Pay attention to the size and consistency of the liver, especially its structure in the section.
4. To study the productive inflammation around the animal parasite on the microscopic picture. Describe the micropreparation "Echinococcal cyst of the liver" (staining with hematoxylin and eosin). Pay attention to the localization of the parasite, the structure of its shell, the localization and cellular composition of the inflammatory infiltrate, the state of the liver tissue.
5. To study miliary pulmonary tuberculosis by macroscopic picture. Describe the macropreparation "Miliary pulmonary tuberculosis". Pay attention to the size and consistency of the lung, the number, localization, size and color of tuberculous tubercles.
6. Examine the tuberculous granuloma on the microscopic picture. Describe the micropreparation "Tuberculous granuloma in the lungs (miliary tuberculosis)" (stained with hematoxylin and eosin). Pay attention to the localization, cellular composition, the state of the central part of the tuberculosis granuloma, changes in the surrounding lung tissue.
7. To study caseous pneumonia according to the macroscopic picture. To describe the macro-preparation "Caseous pneumonia". Pay attention to the area of the lesion, texture, color of the lung tissue in these areas.
8. To study syphilitic mesaortitis on a macroscopic picture. To describe the macropreparation "Syphilitic mesaortitis (syphilitic aortic aneurysm)". Pay attention to the localization of the lesion in the aorta, the perimeter and thickness of the aortic wall in the damaged section, the nature of the intimal lesion.
9. To study syphilitic mesaortitis by microscopic picture. Describe the micropreparation "Syphilitic mesaortitis" (stains with hematoxylin and eosin, fuchselin). Pay attention to the localization and cellular composition of the infiltrate, the state of elastic fibers in the aortic wall and lesions.
10. Examine the endometrial polyp in the macroscopic picture. Describe the macro-preparation "Endometrial polyp". Pay attention to the localization of the lesion in the uterus, the size and color of the formation.

Questions

1. CHRONIC INFLAMMATION SHALL BE A SIMULTANEOUS COMBINATION
1) untenable reparation
2) angiogenesis, scarring
3) reactive changes
4) tissue damage
5) embolism

2. CAUSES OF CHRONIC INFLAMMATION
1) acute infection
2) persistent infection
3) long-term exposure to toxic substances

3. CHRONIC INFLAMMATION IS CHARACTERIZED
1) deposition of amyloid
2) mononuclear infiltration
3) fibrosis of damaged tissues
4) persistent destruction of connective tissue

4. MACROPHAG BY THE CELL OF CHRONIC BASIC INFLAMMATION
1) is
2) is not

Add:

5. The main cell of chronic inflammation is _____.

6. CELLS OF INFILTRATE IN THE FOCUS OF CHRONIC INFLAMMATION
1) macrophages
2) lymphocytes
3) neutrophils
4) eosinophils

7. TISSUE REACTION IN GRANULOMATIC INFLAMMATION
1) exudative
2) proliferative

8. CELLS PARTICIPATING IN PHAGOCYTOSIS
1) macrophages
2) lymphocytes
3) neutrophils
4) erythrocytes
5) plasma cells
6) epithelioid

9. IN THE FOCUS OF PRODUCTIVE INFLAMMATION PHAGOCYTOSIS DEVELOPS
1) pinocytosis
2) completed
3) endocytobiosis
4) unfinished

10. CELLS OF HEMATOGENIC ORIGIN IN THE FOCUS OF CHRONIC INFLAMMATION
1) monocytes
2) labroites
3) lymphocytes
4) fibroblasts
5) endothelial

11. CELLS OF HYSTIOGENIC ORIGIN IN THE FOCUS OF CHRONIC INFLAMMATION
1) monocytes
2) lymphocytes
3) fibroblasts
4) endothelial
5) myofibroblasts

12. CELL OF INFILTRATE
Set correspondence:
1) macrophage
2) lymphocyte
3) fibroblast
4) epithelioid
ORIGIN
A) hematogenous
B) histiogenic

13. CHARACTERISTIC OUTCOME OF PRODUCTIVE INFLAMMATION
1) necrosis
2) sclerosis
3) dystrophy
4) tissue melting

14. TYPES OF PRODUCTIVE INFLAMMATION
1) interstitial (interstitial)
2) granulomatous
3) serous
4) purulent

15. WITH RHEUMATISM IN ADULTS, INFLAMMATION DEVELOPS IN THE MYOCARDIUM
1) granulomatous chronic
2) acute granulomatous
3) interstitial
4) exudative

16. WITH TUBERCULOSIS IN THE LUNGS INFLAMMATION DEVELOPS
1) granulomatous chronic
2) acute granulomatous
3) interstitial
4) exudative

17. WITH ALVEOCOCCOSIS IN THE LIVER INFLAMMATION DEVELOPS
1) interstitial
2) granulomatous
3) exudative
4) polyposis

18. INFLAMMATION DEVELOPS AROUND ANIMAL PARASITES
1) interstitial
2) granulomatous
3) exudative
4) intermediate
5) purulent

19. MICROSCOPIC INTERMEDIATE MYOCARDITIS CHARACTERIZED
1) inflammatory infiltrate in the myocardial stroma
2) dystrophic changes in cardiomyocytes
3) diffuse small focal cardiosclerosis
4) giant cell granulomas
5) stromal hyalinosis

20. CELL COMPOSITION OF INFILTRATE IN INTERCURRENT MYOCARDITIS
1) macrophages
2) lymphocytes
3) neutrophils
4) fibroblasts
5) plasma cells
6) multinucleated giant cells of foreign bodies

21. CHARACTERISTIC OUTCOMES OF INTERSTITIAL (INTERSTITIAL) INFLAMMATION
1) edema
2) necrosis
3) sclerosis
4) calcification

22. CHARACTERISTIC OUTCOME OF INTERSTITIAL (INTERSTITIAL) MYOCARDITIS
1) cardiomyopathy
2) myocardial infarction
3) large focal cardiosclerosis
4) small focal diffuse cardiosclerosis

23. INTERSTITIAL INFLAMMATION MOST OFTEN DEVELOPS IN
1) the brain
2) liver and kidney
3) myocardium and lungs
4) spleen and lymph nodes

24. TYPES OF GRANULOMATOUS INFLAMMATION DEPENDING ON PATHOGENESIS
1) spicy
2) immune
3) subacute
4) chronic
5) non-immune

25. GRANULE IS A MANIFESTATION OF TISSUE REACTION
1) exudation
2) proliferation

26. CHARACTERISTIC CELLS FOR NON-IMMUNE GRANULA
1) lymphocytes
2) eosinophils
3) neutrophils
4) plasma
5) multicore giant foreign bodies

27. IMMUNE GRANULOMATOUS INFLAMMATION DEVELOPS WHEN
1) silicosis
2) syphilis
3) rabies
4) tuberculosis
5) alveococcosis

28. NON-IMMUNE GRANULOMATOUS INFLAMMATION DEVELOPS WHEN
1) rabies
2) syphilis
3) tuberculosis
4) schistosomiasis
5) rhinoscleroma

29. CLINICAL COURSE OF DISEASES WHICH DEVELOP GRANULOMATOUS IMMUNE INFLAMMATION
1) acute
2) subacute
3) the sharpest
4) chronic

30. CLINICAL COURSE OF DISEASES WHICH DEVELOP GRANULOMATOUS NON-IMMUNE INFLAMMATION
1) spicy
2) subacute
3) the sharpest
4) chronic

31. SYNONYM OF ENDOCYTOBIOSIS
1) pinocytosis
2) granulomatosis
3) completed phagocytosis
4) incomplete phagocytosis

32. IN MULTI-CHAMBER ECHINOCOCCOSIS OF THE LIVER ORGAN SIZES
1) reduced
2) not changed
3) increased

33. OUTCOMES OF INFLAMMATION AROUND ANIMAL PARASITES
1) petrification
2) encapsulation
3) hyalinosis of arterioles
4) atrophy of organ tissue
5) secondary amyloidosis

34. MICROSCOPIC TUBERCULOSIS GRANULE IS CHARACTERIZED
1) neutrophils
2) lack of blood vessels
3) many small vessels
4) caseous necrosis in the center
5) fibrinoid necrosis in the center
6) giant cells of Pirogov-Langhans

35. STROMA COMPONENT IN TUBERCULOSIS GRANULE
1) collagen fibers
2) reticular fibers
3) elastic fibers
4) fibrin filaments
5) vessels

36. CELLS OF TUBERCULOSIS GRANULUM
1) MONOCYTES
2) lymphocytes
3) fibroblasts
4) epithelioid
5) plasma
6) giant Pirogov-Langhansa

37. TUBERCULOSIS GRANULE IS A MANIFESTATION OF A HYPERSENSITIVITY REACTION
1) immediate type
2) delayed type

38. VESSELS IN TUBERCULOSIS GRANULE
1) are present in small quantities
2) are determined in large quantities
3) absent

39. THE CENTRAL PART OF THE TUBERCULOSIS GRANULE IS PRESENTED BY NECROSIS
1) fatty
2) caseous
3) waxy
4) fibrinoid
5) colliquation

Add:
40. The central part of the tuberculous granuloma is represented by _____ necrosis.

41. CHARACTERISTIC TISSUE REACTION IN THE PRESENCE OF IMMUNITY IN A PATIENT WITH TUBERCULOSIS
1) productive
2) alternative
3) exudative

42. TUBERCULOSIS BUILDINGS DEPENDING ON CELL COMPOSITION
1) lymphoid
2) necrotic
3) plasma cell
4) giant cell
5) epithelioid cell

43. SYNONYM OF SYPHILYTIC GRANULEMA
1) petrification
2) tubercle
3) knot
4) gumma

44. GUMMA IS A MORPHOLOGICAL MANIFESTATION OF SYPHILIS
1) early congenital
2) primary
3) secondary
4) tertiary

45. BASIC CELLS OF GUMMA
1) lymphocytes
2) neutrophils
3) fibroblasts
4) epithelioid
5) plasma

46. FIBER OF THE STROMA OF A SYPHILYTIC GRANULE
1) collagen
2) reticular
3) elastic
4) fibrin

47. VESSELS IN A SYPHILYTIC GRANULE
1) are present in small quantities
2) are determined in large quantities
3) absent

48. VESSELS IN GUMMA
1) little
2) a lot

49. A MICROSCOPIC SYPHILYTIC GRANULE IS CHARACTERIZED
1) hyalinosis in the center
2) lack of blood vessels
3) productive vasculitis
4) caseous necrosis in the center
5) collagen fibers in the stroma

50. MICROSCOPIC SYPHILYTIC GRANULE IS CHARACTERIZED
1) lack of necrosis
2) caseous necrosis in the center
3) reticular fibers in the stroma
4) an abundance of epithelioid cells
5) the predominance of lymphocytes and plasma cells

51. MOST OF ALL GUMMA IS LOCALIZED IN
1) stomach and intestines
2) the brain
3) spleen
4) liver
5) aorta

Add:
52. Gummas and syphilitic mesaortitis are manifestations of the _____ period of syphilis.

53. MORPHOLOGICAL MANIFESTATIONS OF THE TERTIARY PERIOD OF SYPHILIS
1) hard chancre
2) gum in the internal organs
3) fibrinous inflammation of the mucous membranes
4) gummy infiltration of the vessel walls and organ stroma

54. COMPONENTS OF THE GRANULE
Set correspondence:
1) plasmocytes and lymphocytes
2) collagen fibers
3) reticular fibers
4) epithelioid cells
5) caseous necrosis
6) fibroblasts
DISEASE
A) syphilis
B) tuberculosis
B) tuberculosis and syphilis

55. MEZAORTITIS DEVELOPS IN SYPHILIS
1) primary
2) secondary
3) tertiary

56. LOCALIZATION OF CHANGES IN THE AORTE IN SYPHILITIC MESORTITIS
1) arc
2) thoracic region
3) abdominal

57. LOCALIZATION OF INFILTRATE IN THE AORTIC WALL IN SYPHILITIC MESAORTITIS
1) only media
2) only intimacy
3) only adventitia
4) media and intimacy
5) media and adventure
6) intimacy and adventitia

58. PRIMARY SYPHILIS IS CHARACTERIZED BY EDUCATION
1) gum
2) syphilis
3) hard chancre
4) gummy infiltrate

List of recommended literature:
Basic literature:

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Additional literature:

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