

Nonspecific Epididymitis and Orchitis

Etiology · spread · morphology · functional consequences

Pathology teaching lecture

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Learning objectives

- Map the routes by which urinary tract infection reaches the epididymis and testis.
- Relate age-associated pathogens to nonspecific epididymitis.
- Recognize interstitial inflammation, tubular extension, abscess and suppurative necrosis.
- Explain scarring-associated fertility loss with relative Leydig-cell preservation.
- Distinguish nonspecific inflammation from selected infectious, granulomatous and vascular lesions.

Epididymis is the common initial site

Epididymis

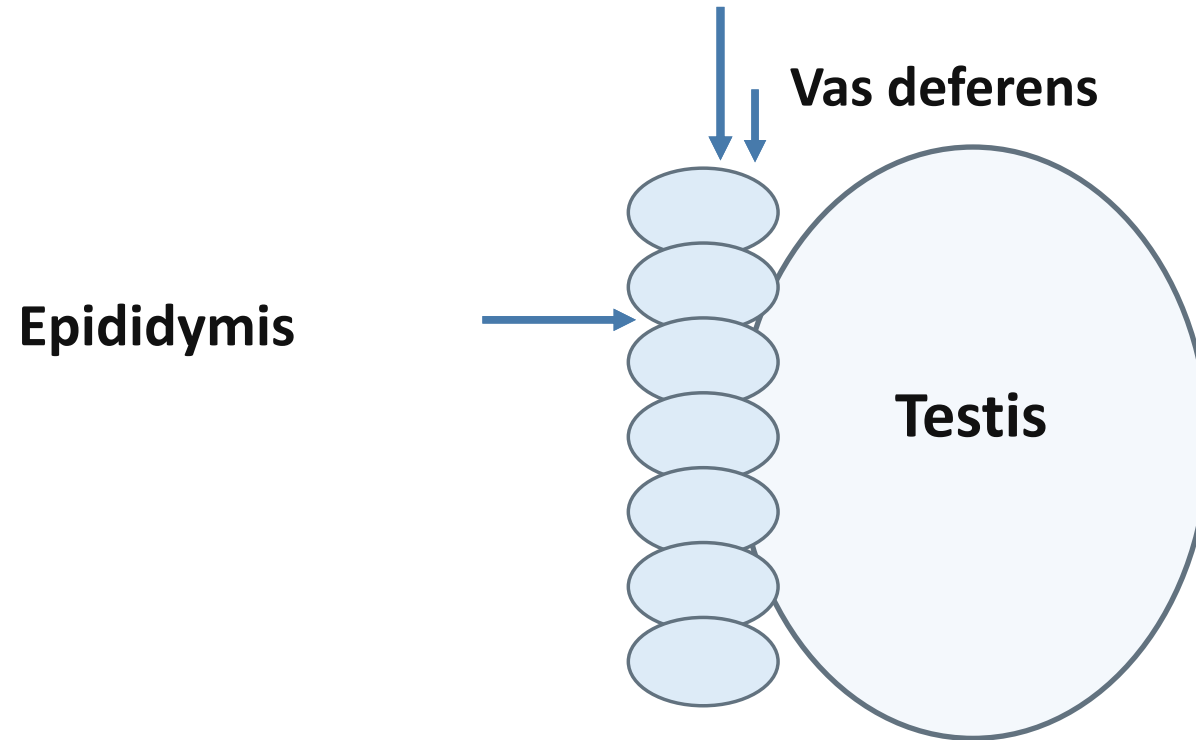
- Inflammatory lesions occur more frequently here.
- Several infections originate in, or first reach, this structure.
- Acute infection can later extend into testis.

Testis

- Testicular inflammation may be secondary to epididymitis.
- Some etiologies—mumps and syphilis—primarily involve testis.
- The first affected site helps frame the differential.

Context from the parent section: Inflammation and Infections.

Route-relevant anatomy of the male genital tract



Simplified topology; Infection may travel through the vas deferens or lymphatics of the spermatic cord.

Three common upstream infection sites

Urethritis

An upstream genital-tract infection that may extend toward the epididymis.

Cystitis

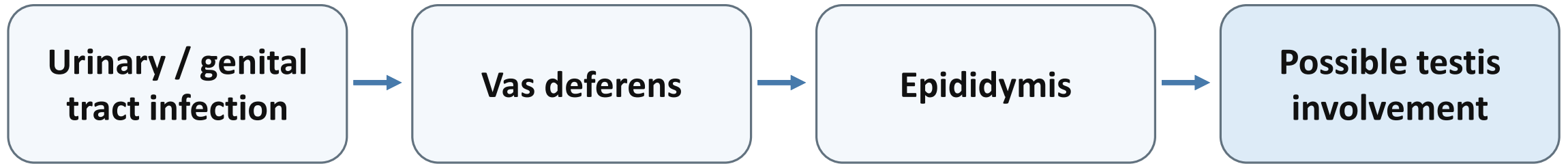
A urinary bladder infection that can be associated with epididymal infection.

Prostatitis

A prostatic infection included among the recognized sources.

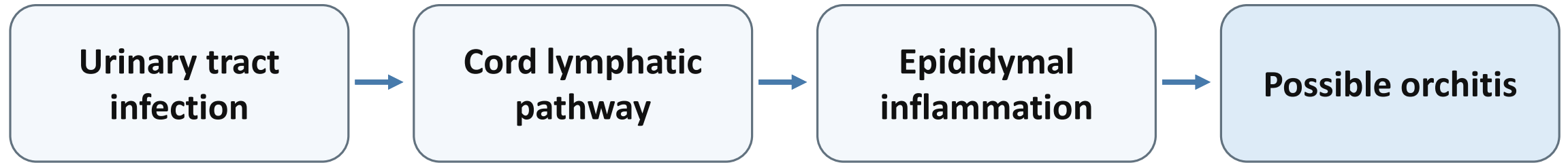
Source identifies each as a common associated urinary tract infection.

Ascending infection through the vas deferens



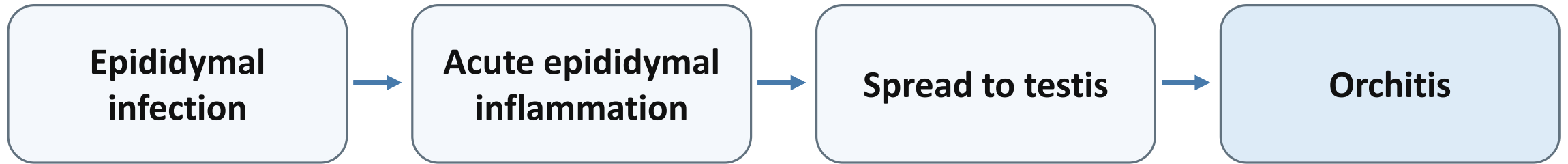
This route helps explain why epididymitis often precedes orchitis.

Spread through spermatic-cord lymphatics



Lymphatic spread is a second route.

Epididymitis may be followed by orchitis



The testis may show an inflammatory reaction similar to that in the epididymis.

Age-associated microbiologic pattern

Population	Clinical context	Frequent organisms
Children	Uncommon; congenital genitourinary abnormality often present	Gram-negative rods
Sexually active males <35 years	Sexually transmitted infection	Chlamydia trachomatis; Neisseria gonorrhoeae
Males >35 years	Common urinary tract organisms	Escherichia coli; Pseudomonas

Childhood epididymitis: Look for congenital context

- Nonspecific epididymitis is uncommon in children.
- The textbook associates pediatric cases with a congenital genitourinary abnormality.
- The commonly described infecting organisms are gram-negative rods.
- Age and anatomic context must be interpreted together.

Sexually active males younger than 35 years

Chlamydia trachomatis

- Recognized sexually transmitted pathogen.
- One of the most frequent agents in this age group.
- Belongs in age-pattern differential.

Neisseria gonorrhoeae

- Recognized sexually transmitted pathogen.
- One of the most frequent agents in this age group.
- May produce severe suppurative disease.

After age 35: Ordinary urinary pathogens

Escherichia coli

- Common urinary tract pathogen.
- One of the characteristic organisms in older males.
- Supports urinary-source model.

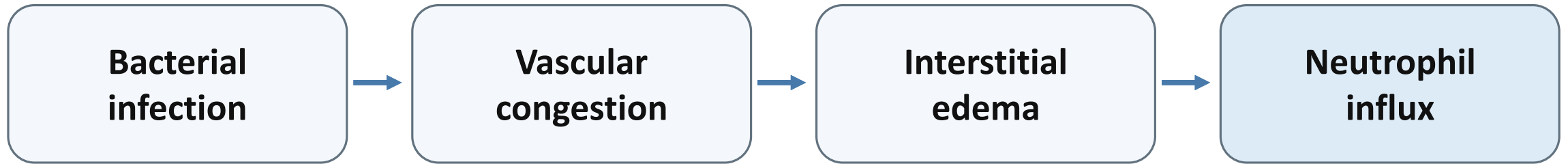
Pseudomonas

- Another pathogen named for older males.
- Associated with urinary tract infection.
- May participate in nonspecific inflammation.

What age stratification does—and does not—show

- Predominant associations rather than mutually exclusive pathogen lists.
- Childhood, young adulthood and later adulthood represent differing etiologic contexts.
- Age is therefore an organizing clue; The tissue reaction remains an acute inflammatory pattern.
- Avoid equating a pathogen prediction with confirmed microbiologic identification.

Core tissue response to bacterial infection



The same basic acute inflammatory components occur at other infected sites.

Congestion: An early vascular component

- Congestion is a defining morphologic feature of acute bacterial inflammation in this setting.
- Vascular engorgement accompanies the inflammatory response.
- The process initially involves epididymal interstitial connective tissue.
- Congestion should be read in combination with edema and neutrophils.

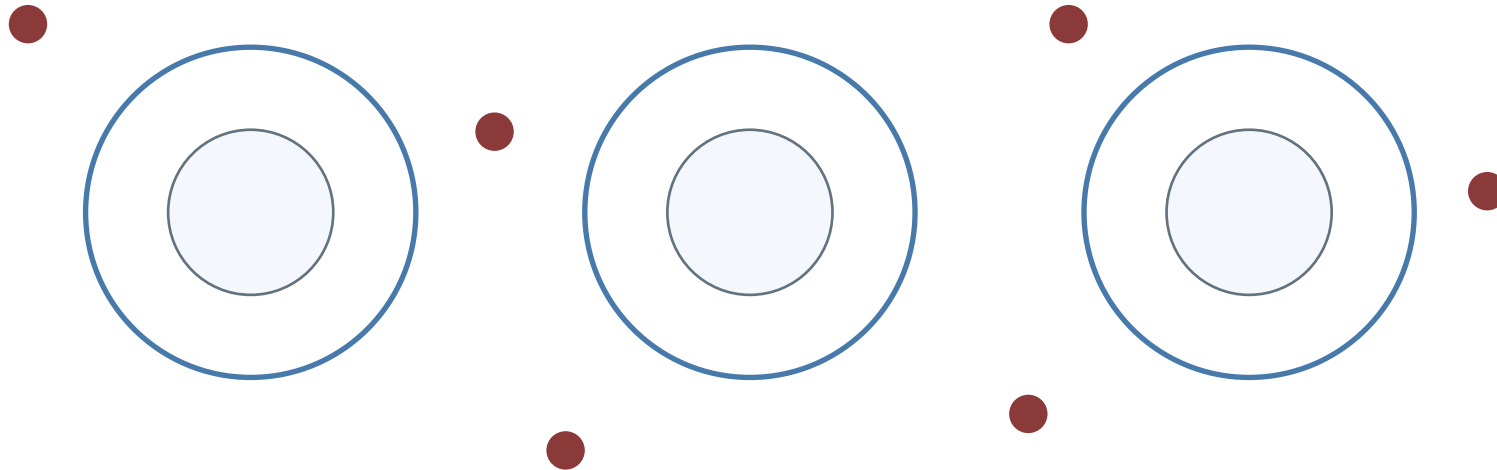
Edema expands the inflammatory interstitium

- Edema is part of the acute bacterial inflammatory pattern.
- It occurs in the same interstitial compartment as initial inflammation.
- Edema contributes to expansion of inflamed tissue before and during tubular extension.
- Edema alone is less specific than the full acute inflammatory pattern.

Neutrophils identify suppurative inflammation

- Neutrophilic infiltration is characteristic of bacterial epididymitis.
- Neutrophils are present with congestion and edema.
- Marked neutrophilic accumulation may culminate in an abscess.
- The dominant inflammatory-cell type helps distinguish bacterial and viral patterns.

Acute inflammation begins in interstitial connective tissue

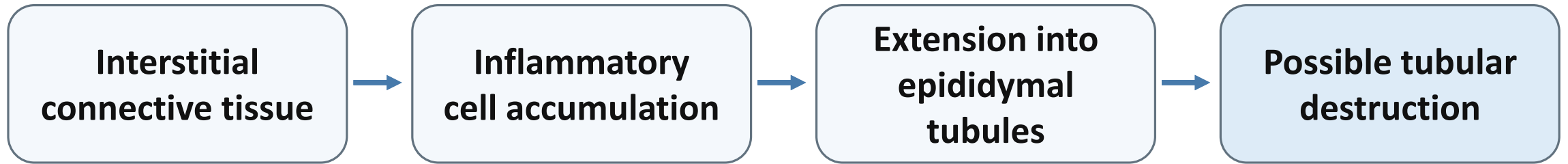


Schematic · not microscopy

Tubular lumina

Acute inflammatory cells occupy interstitial spaces before rapidly extending into tubules.

Tubular involvement follows interstitial inflammation

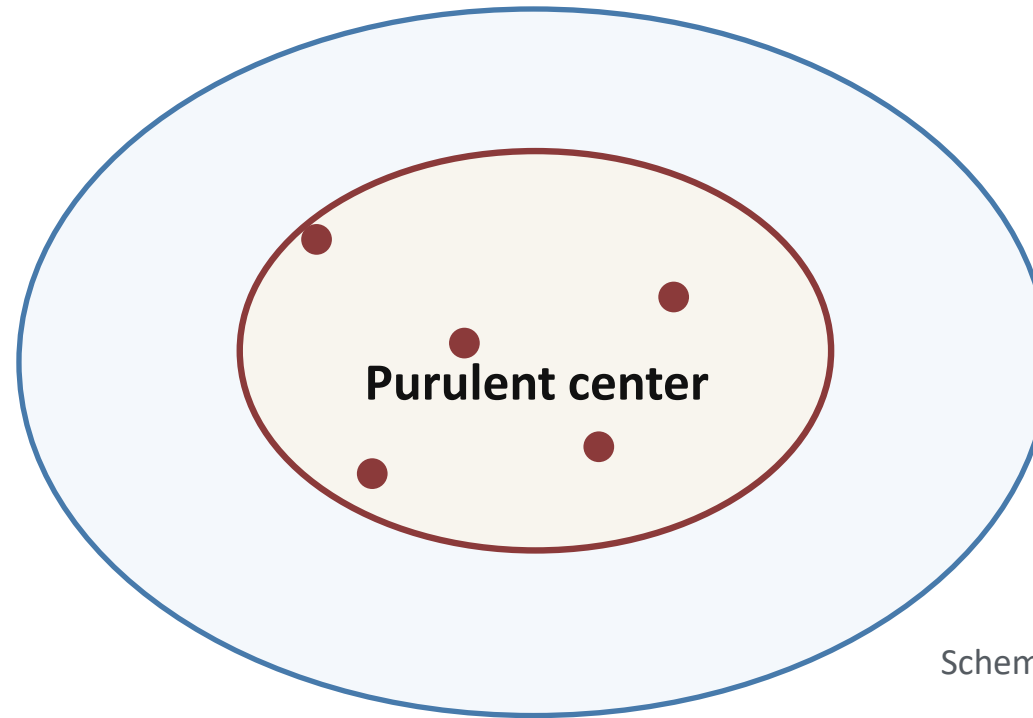


Robbins specifically describes rapid extension from interstitium to tubules.

Tubular extension marks advancing disease

- The inflammation starts interstitially but can involve tubular structures rapidly.
- An inflammatory infiltrate limited to stroma and one involving tubules represent different extents of injury.
- The tubules lie directly in the tissue compartment threatened by suppuration.
- Severe extension can progress to destructive abscess formation.

An abscess is a focal outcome of severe suppuration



Schematic · not a gross specimen

Surrounding inflamed epididymal tissue

Suppurative necrosis: The destructive extreme

- Acute inflammation can produce suppurative necrosis of the entire epididymis.
- The term denotes more extensive destruction than a localized abscess.
- A spectrum: Interstitial inflammation → tubular involvement → suppurative destruction.
- Severe tissue loss helps explain the possibility of subsequent scarring.

Gonococcal epididymitis may replace tissue with abscess

- Figure 21.16 is described as acute gonococcal epididymitis.
- The figure caption reports replacement of epididymal tissue by an abscess.
- The adjacent testis is described as normal in that particular specimen.
- This example demonstrates that severe epididymitis need not imply simultaneous orchitis.

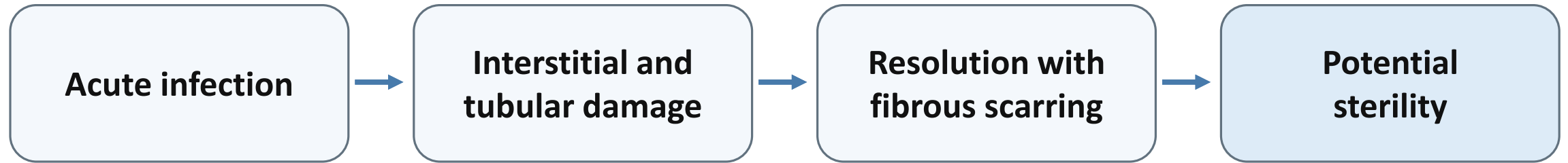
Secondary orchitis follows local spread

- Infection can extend from the epididymis into the testis.
- Once involved, the testis exhibits a similar acute inflammatory response.
- Presence of inflammation in both organs is more extensive than epididymitis alone.
- The expected morphologic vocabulary remains congestion, edema and inflammatory infiltration.

Epididymitis versus epididymo-orchitis

Feature	Epididymitis	Epididymo-orchitis
Initial involvement	Epididymal tissue	Epididymis with secondary testicular inflammation
Inflammatory reaction	Interstitial neutrophils; may extend into tubules	Similar acute inflammation also involves testis
Severe morphologic pattern	Abscess / suppurative necrosis possible	Testicular damage may accompany epididymal injury
Late consequence	Fibrous scarring may impair fertility	Scarring and fertility effects may involve both sites

Sequence from inflammation to fibrosis

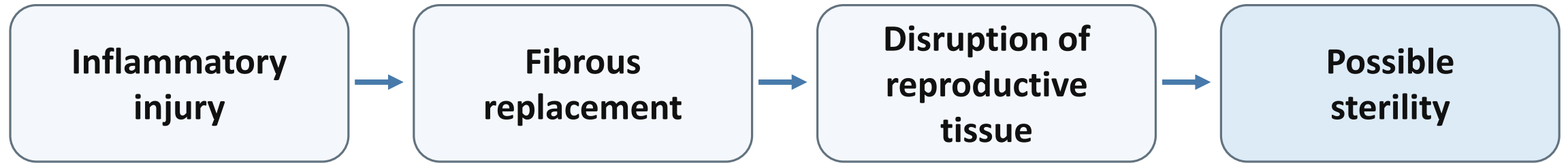


Fibrosis is a recognized late outcome of inflammation involving epididymis and testis.

Fibrous scarring can follow acute inflammation

- Inflammation of epididymis and testis is often followed by fibrosis.
- The late scar may reflect healing after destructive inflammatory tissue injury.
- Tissue scarring is distinct from the congestion, edema and neutrophils of the acute phase.
- Recognizing the phase of injury is important when interpreting pathology.

Why fibrosis may cause infertility



The text explicitly links postinflammatory fibrosis with sterility; Not every infection causes it.

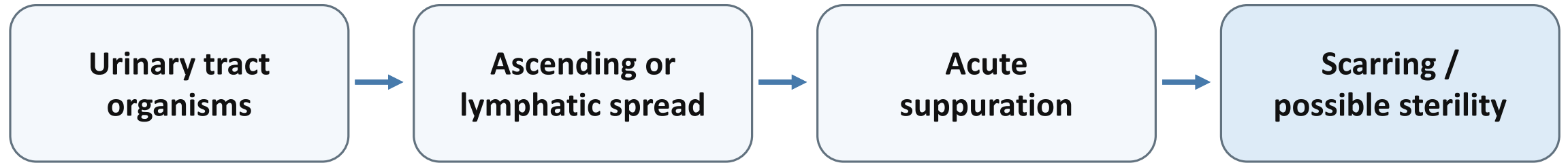
Leydig cells are usually preserved

- Leydig cells usually are not destroyed by this inflammation.
- Preservation provides a morphologic explanation for maintained androgen production.
- Relative endocrine preservation can coexist with damage to sperm-bearing structures.
- The qualifier “usually” is important: Preservation is not presented as universal.

Reproductive and endocrine consequences can differ

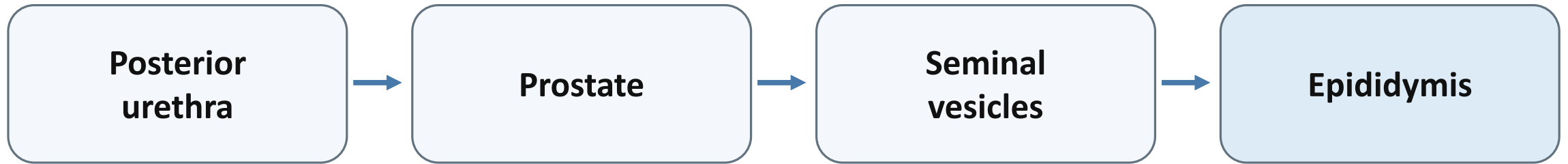
Aspect	Pathologic basis	Typical implication
Fertility	Inflammation and fibrous scarring of epididymis/testis	Sterility may occur
Androgen production	Leydig cells usually not destroyed	Testicular androgen output relatively unaffected
Interpretation	Different structures mediate different functions	Do not equate fertility loss with obligatory androgen failure

Mechanism-to-outcome synthesis



Leydig-cell preservation helps explain relatively retained androgen synthesis.

Gonococcal infection can extend sequentially

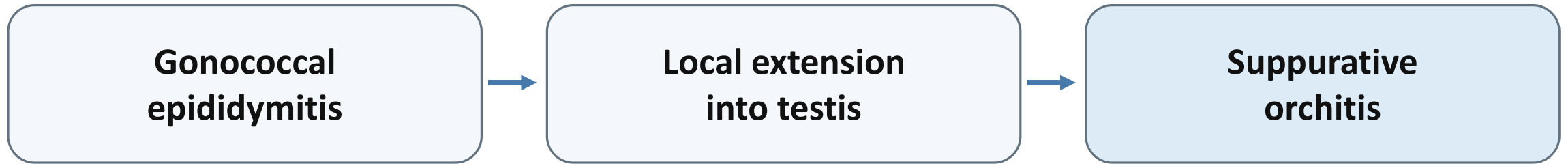


This sequence as a complication of neglected gonococcal infection.

Severe gonorrhea may destroy epididymal tissue

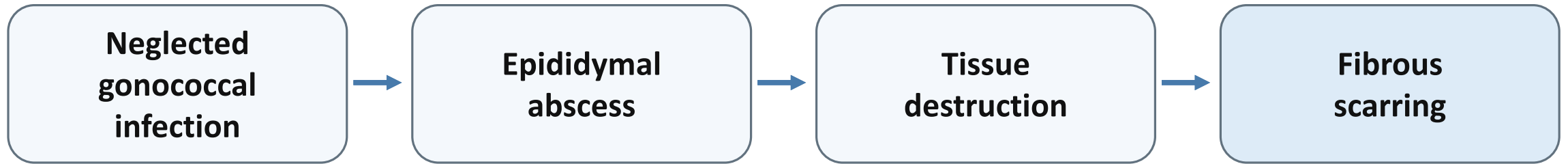
- Gonococcal infection is among the major sexually transmitted causes in younger males.
- Severe involvement can produce epididymal abscesses.
- Abscess formation may be accompanied by extensive destruction.
- The healing process can leave fibrous scarring.

Gonococcal infection may extend into the testis



This represents a route from lower genitourinary infection to testicular inflammation.

Possible consequences of gonococcal epididymitis



The listed sequence is an illustrated severe pathway, not the inevitable course of every case.

Suppurative and granulomatous patterns differ

Nonspecific bacterial epididymitis

- Neutrophil-rich acute response.
- Initial interstitial inflammation.
- Abscess / suppurative necrosis can occur.

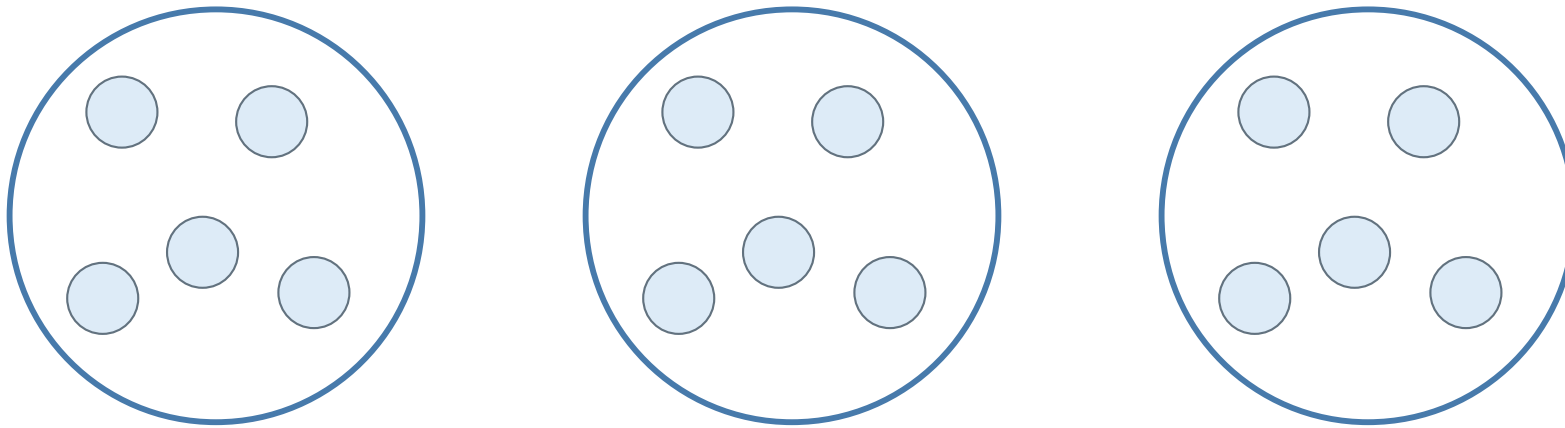
Idiopathic granulomatous orchitis

- Rare inflammatory testicular disorder.
- Granulomas restricted to spermatic tubules.
- May resemble testicular neoplasm clinically.

Granulomatous orchitis may simulate a tumor

- Idiopathic granulomatous orchitis may present as a moderately tender mass of sudden onset.
- Fever sometimes accompanies the presentation.
- An insidious painless testicular mass also occurs.
- This tumor-like presentation makes histologic distinction important.

Granulomatous orchitis: Tubule-centered lesions



Schematic · not microscopy

Granulomatous reaction confined to spermatid tubules

Granulomatous orchitis: Proposed etiologies

Trauma	Infection	Extravasated sperm	Autoimmunity
One postulated mechanism, not a proven universal cause.	A proposed possible trigger in this idiopathic disorder.	Proposed inflammatory stimulus from sperm outside its usual compartment.	Another proposed explanation; The name does not establish pathogenesis.

Robbins explicitly presents these as hypotheses, not demonstrated causes.

Idiopathic granulomatous orchitis versus tuberculosis

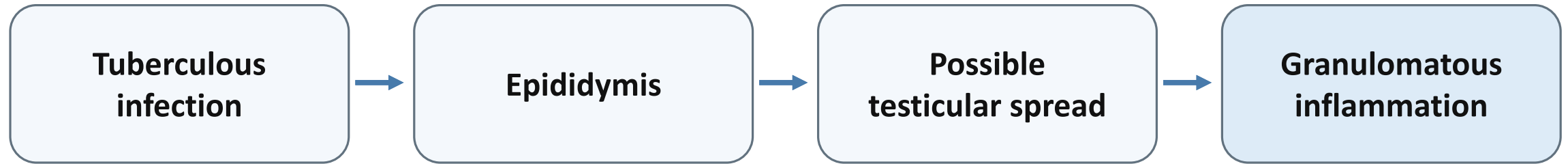
Idiopathic granulomatous orchitis

- Granulomas restricted to spermatic tubules.
- May clinically resemble a tumor.
- No caseous necrosis specified as defining feature.

Genital tuberculosis

- Often begins in epididymis.
- May spread to testis.
- Typically shows caseating granulomatous inflammation.

Tuberculosis commonly begins in the epididymis



Unlike nonspecific acute bacterial disease, its characteristic response is caseating granulomas.

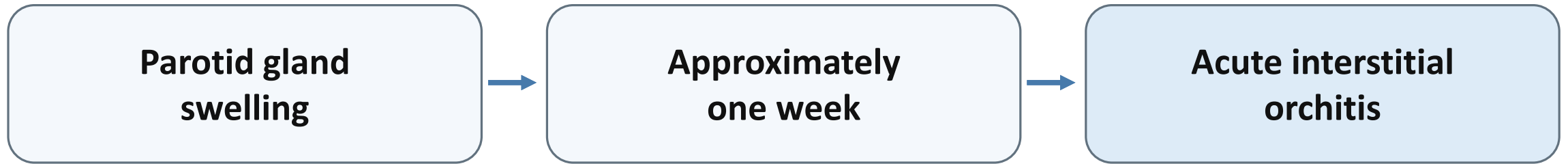
Caseating granulomas favor tuberculous infection

- A classic caseating granulomatous reaction in genital tuberculosis.
- Genital infection almost invariably begins in epididymis before testicular spread.
- Tuberculosis is an important differential for idiopathic granulomatous orchitis.
- Distribution and caseous necrosis distinguish it from tubule-restricted idiopathic disease.

Mumps orchitis mainly concerns postpubertal males

- Mumps is described as a systemic viral disease often affecting school-aged children.
- Testicular involvement is extremely uncommon at school age.
- Orchitis in 20%–30% of postpubertal males with mumps.
- The age contrast differs from the bacterial epididymitis pathogen patterns.

Timing of mumps-associated orchitis



The testicular phase often follows the systemic parotid presentation.

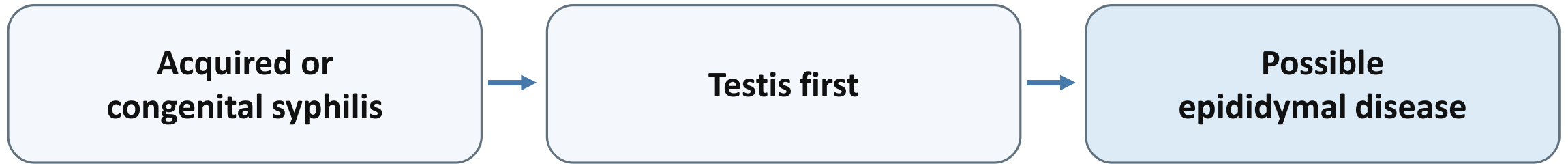
Inflammatory cells help distinguish mumps

Pattern	Predominant cells	Distribution
Acute bacterial epididymitis	Neutrophils	Interstitial initially; may extend into tubules
Mumps orchitis	Lymphocytes and plasma cells	Acute interstitial orchitis

Bilateral mumps orchitis may reduce fertility

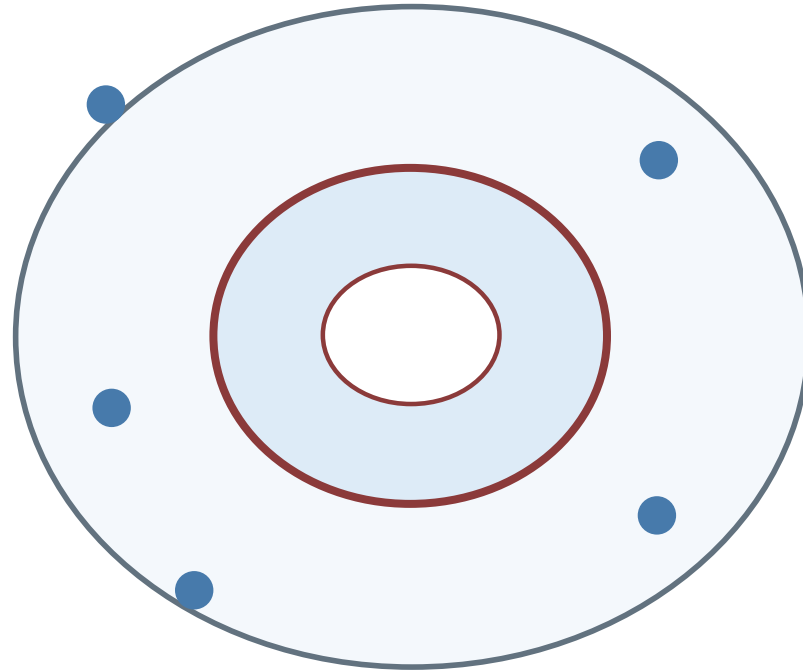
- Mumps-associated orchitis usually develops after parotid gland swelling.
- The testicular inflammation is lymphoplasmacytic rather than neutrophil-dominant.
- Bilateral testicular involvement may reduce fertility.
- This effect is separate from the fibrosis-associated sterility described in bacterial infection.

Syphilis often starts in the testis



This direction contrasts with epididymis-first gonorrhea and tuberculosis.

Syphilis: Obliterative endarteritis and perivascular infiltrate



Schematic · not histology

Concentric vascular-wall injury and perivascular lymphoplasmacytic cells

Gumma: The second syphilitic reaction pattern

- Syphilis can cause granulomatous inflammation known as a gumma.
- Gumma as an alternative to the vascular-centered reaction.
- These patterns are not the neutrophilic abscess described in nonspecific bacterial epididymitis.
- Syphilitic pathology therefore requires a separate morphologic category.

Two morphologic patterns in syphilis

Pattern	Hallmark	Distinguishing point
Obliterative endarteritis	Vascular lesion with perivascular lymphocytes and plasma cells	Prominent vessel-centered process
Gumma	Granulomatous inflammation	Granulomatous rather than acute suppurative reaction

Inflammatory patterns by selected cause

Etiology	First common site	Principal morphology
Nonspecific bacterial	Epididymis	Congestion, edema, neutrophils; possible abscess
Mumps	Testis	Acute interstitial lymphocytes and plasma cells
Tuberculosis	Epididymis	Caseating granulomas
Syphilis	Testis	Endarteritis / plasma cells or gumma

Anatomic origin is a differential clue

Epididymis-first patterns

Nonspecific bacterial epididymitis

Gonorrhea

Genital tuberculosis

Testis-first patterns

Mumps orchitis

Syphilis

Some idiopathic granulomatous lesions are primarily testicular

Other rare disorders listed in the source

Infectious

Leprosy; Toxoplasmosis; Fungi; Viruses;
Parasites; Brucellosis.

Inflammatory / systemic

Sarcoidosis; Crohn disease; Malakoplakia.

These are listed as rare causes; The source does not provide detailed individual criteria.

Abscess versus granulomatous lesion

Abscess / suppuration

- Predominantly neutrophilic inflammatory process.
- Can destroy epididymal tubules.
- Described in severe gonococcal disease.

Granulomatous lesion

- Granulomas are the organizing feature.
- Tuberculosis can show caseation.
- Idiopathic orchitis can be tubule restricted.

Neutrophils versus lymphoplasmacytic inflammation

Bacterial epididymitis

- Neutrophils are prominent.
- Congestion and edema accompany cells.
- Suppurative necrosis may occur.

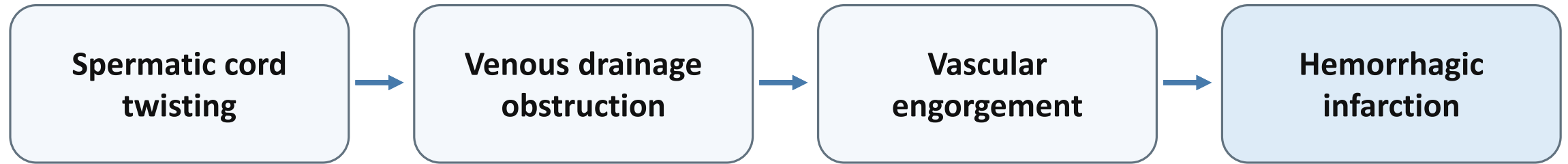
Mumps orchitis

- Lymphocytes and plasma cells.
- Acute interstitial testicular pattern.
- Often follows parotid swelling.

Torsion belongs in the acute scrotal differential

- Testicular torsion is a vascular lesion, not an infectious orchitis.
- Twisting of the spermatic cord compromises testicular venous drainage.
- If untreated, testicular infarction frequently follows.
- Torsion as a true urologic emergency.

Torsion: Venous outflow interruption



Thick-walled arteries may remain patent while venous outflow is impeded.

Torsion and infection cause different tissue injury

Finding	Acute bacterial inflammation	Torsion
Primary mechanism	Infection with inflammatory response	Mechanical interruption of venous drainage
Major acute lesion	Neutrophilic inflammation / suppuration	Engorgement and hemorrhagic infarction
Principal hazard	Scarring / possible sterility	Rapid testicular infarction
Source urgency	Source does not prescribe a timing threshold	Urologic emergency

Recognizing torsion changes urgency

- “Adult” torsion typically presents in adolescence with sudden testicular pain.
- The bell-clapper abnormality increases mobility and predisposes to twisting.
- Manual untwisting within approximately six hours may spare orchiectomy.
- The stated interval is from textbook context, not a guarantee of testicular salvage.

Case: Epididymitis in a child

Clinical-pathologic scenario

A boy has documented epididymal infection and a known congenital genitourinary abnormality.

Which pathogen class best fits the Robbins age pattern?

Gram-negative rods are the characteristic reported group.

Case: Infection in a young sexually active adult

Clinical-pathologic scenario

A 27-year-old sexually active man develops a documented epididymal infection.

Which two organisms are emphasized by the textbook for this setting?

Chlamydia trachomatis and Neisseria gonorrhoeae.

Case: Epididymitis in later adulthood

Clinical-pathologic scenario

An older man develops epididymitis in association with a urinary tract infection.

Which pathogen examples does Robbins highlight?

Escherichia coli and Pseudomonas.

Case: Severe suppurative damage

Clinical-pathologic scenario

A resected epididymis shows abscess formation, broad tissue loss and subsequent scarring.

What major long-term function can be affected?

Fertility may be impaired; Leydig-cell-mediated androgen synthesis may remain relatively unaffected.

Case: Tumor-like testicular inflammatory mass

Clinical-pathologic scenario

A middle-aged patient has a testicular mass; Microscopy shows granulomas limited to spermatogenic tubules.

Which non-neoplastic diagnosis is most consistent?

Idiopathic granulomatous (autoimmune) orchitis; Distinguish tuberculosis.

Case: Orchitis after parotid swelling

Clinical-pathologic scenario

A postpubertal male develops orchitis about one week after parotid enlargement.

What inflammatory cell pattern is expected?

An interstitial lymphocytic and plasma-cell infiltrate is characteristic of mumps orchitis.

Case: Sudden testicular pain in an adolescent

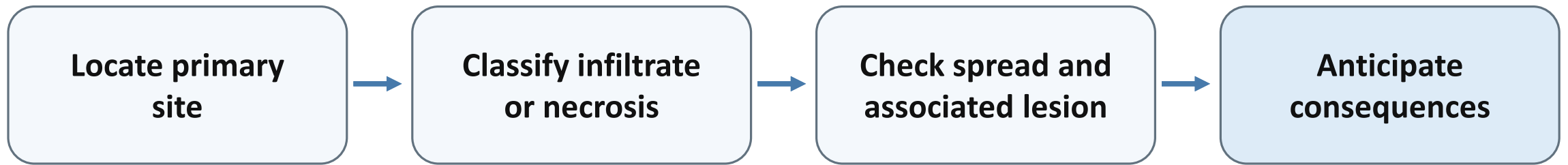
Clinical-pathologic scenario

An adolescent has sudden testicular pain, with a spermatic-cord vascular event suspected.

What pathologic process must be urgently excluded?

Torsion: Impaired venous drainage can produce hemorrhagic testicular infarction.

A structured pathology reading strategy



Integrate distribution, inflammatory phenotype, and potential functional sequelae.

Three high-yield cause–morphology links

Bacterial disease

Epididymis-first;
Neutrophils, edema and
congestion; Possible
abscess.

Selected nonbacterial infections

Mumps:
Lymphoplasmacytic
orchitis. Tuberculosis:
Caseating granulomas.

Postinflammatory sequelae

Fibrous scarring may
produce sterility while
Leydig cells are usually
preserved.

Concise differential of the testis and epididymis

Disease	Morphologic clue	Functional or clinical implication
Nonspecific epididymitis	Interstitial → tubular neutrophilic injury	Fibrosis may impair fertility
Idiopathic granulomatous orchitis	Spermatic-tubule granulomas	Can mimic a testicular tumor
Mumps orchitis	Lymphoplasmacytic interstitial reaction	Bilateral disease may reduce fertility
Tuberculosis	Caseating granulomas; epididymis first	Can extend to testis
Torsion	Hemorrhagic infarction	Urgent, potentially organ-threatening

Key conclusions

- Epididymitis is more common than primary orchitis; Ascending or lymphatic spread is typical.
- Probable organisms vary with age and context.
- Bacterial injury evolves from interstitial neutrophils toward possible abscess and suppurative necrosis.
- Scarring may impair fertility, while Leydig-cell function is commonly relatively preserved.
- Granulomatous, viral and vascular lesions have distinct morphologic and clinical patterns.

Source and scope

Primary textbook

Robbins, Cotran & Kumar: Pathologic Basis of Disease. 11th edition (2026). Chapter 21, “The Lower Urinary Tract and Male Genital System,” pp. 880–881.

Sections used

Nonspecific Epididymitis and Orchitis; Inflammation and Infections; Granulomatous (Autoimmune) Orchitis; Infections; Torsion.

Illustrations are original editable schematics; they do not substitute for original H&E or gross photographs.