Pseudomembranous enterocolitis

Pseudomembranous enterocolitis (also known as CDAD – *clostridium difficile associated diarrhea*) is a serious, life-threatening complication in patients treated with broad-spectrum antibiotics that disrupts the natural microflora and leads to **intestinal dysmicrobia**. Clostridium difficile toxins play the major role in pathogenesis. It originates from endogenous source of infection and is usually transmitted from spores infected objects.

Cause

The causative agent is the anaerobic G+ rod Clostridium difficile, which multiplies extremely, mainly after lincomycin, cephalosporins and aminopenicillins, and begins to produce the toxins **enterotoxin A and cytotoxin B**.

- **Toxin A** – induces fluid accumulation in the intestinal epithelium of the host. It is an accumulation of viscous fluid of blood origin. Toxin A thus causes *dysfunction of the intestinal epithelial cells*, which can no longer optimally control the movement of water due to the large amount of viscous fluid in the cells. Toxin A is a probable **cause of diarrhea**, which is the first symptom of pseudomebranous enterocolitis. It also has a marked polymorphonuclear (*PMN*) chemotaxis and is therefore responsible for the *inflammatory response of intestinal epithelial cells*. Toxin A is toxic to most cells of the immune system because it disrupts cytoskeletal components.
- **Toxin B** – does not show enterotoxic activity, but **kills intestinal epithelial cells**. It is about 100–1000 (tel:100–1000) times more toxic than toxin A.

Image of intestinal mucosa with pseudomembranous enterocolitis

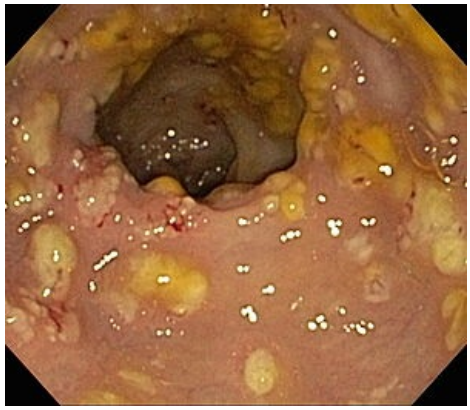
Intestinal mucosa cells are only destroyed if both types of toxins are present at the same time. *Toxins A* destroy the surface structures of the intestinal mucosa cells *and at the same time shield the possible preventive effect of PMNs. This allows toxin B molecules to successfully attack intestinal epithelial cells.* Toxin B *kills intestinal epithelial cells* only if its surface structures are disrupted and if the cells are affected by water transport dysfunction. Extensive ulcerations form on the intestinal mucosa – necrotic formations, pseudomembranes – conglomerates of the destroyed intestinal epithelial cells, dead PMNs, fibrin and mucin in the form of cauliflower swollen, yellow crusts, which can be relatively easily detected via endoscopy.

Any other antibiotic can cause CDAD. It can occur after **5–10 days** of treatment, but also for many days after the end of therapy. Up to **15 %** of patients occur after beta-lactams, and up to **25 %** after clindamycin. As a rule, the disease subsides after the end of therapy.

Clinical picture

Signs of pseudomembranous enterocolitis are:

- severe inflammatory ulcerations of the intestinal mucosa, the mortality of which can often reach up to **45 %**;
- sudden multiple watery diarrheas with dehydration;
- nausea, abdominal pain, often fever and leukocytosis;
- strong-smelling stools, with pieces of mucous membranes and blood;
- in progression – shock and death.



Endoscopic view

Therefore, hospitalization at the ICU is necessary.

Diagnosis

The exact diagnosis is based on two points:

- **Endoscopy** – whitish or yellowish pseudomembranes in the colon with a tendency to merge (ulcers that are typical of bacillary or amoebic dysentery or ulcerative colitis are mostly missing)
- **Detection of clostridial toxin** – performed by ELISA from a stool sample

Therapy

Basic measures include **discontinuation of the current ATB therapy** and **metronidazole** applied orally for 10–14 days. **Vancomycin** is administered **orally** in severe cases due to the risk of spreading the vancomycin-resistant enterococcus. The macrolide antibiotic **fidaxomycin** enables a new, selective therapy, the disadvantage of which is the high cost so far.

Antimotility drugs are not given, diarrhea is an attempt by the body to get rid of the pathogen. The benefit of probiotics has not been proven in treatment. **Intestinal microbiome transplantation** (insertion of homogenized stool from the donor into the patient's intestine using a nasogastric tube or enema) is successfully used in recurrence.

Links

Related articles

- Endotoxin shock
- Clostridium difficile

References

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Source

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