

## Spontaneous Bacterial Peritonitis Caused by *Haemophilus parainfluenzae*

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Spontaneous bacterial peritonitis is a well-defined clinical entity complicating up to 30% of hospitalized cirrhotic individuals [1] and 3.5% of asymptomatic cirrhotic outpatients [2]. To the best of our knowledge the present report is the first to describe a case of SBP caused by *Haemophilus parainfluenzae*.

### Patient Description

A 44 year old man with a history of intravenous drug abuse and chronic hepatitis C infection complicated with cirrhosis was admitted because of lower abdominal pain. There were no other complaints except for mild watery diarrhea that he attributed to lactulose intake. He was treated on a regular basis with methadone as part of a drug addiction rehabilitation plan, but was not under medical surveillance for his hepatitis C infection and cirrhosis. On admission he had fever of 37.8°C and physical examination revealed moderate splenomegaly, and abdominal distension with mild diffuse tenderness.

Peritoneal fluid was a transudate with 140/mm<sup>3</sup> white blood cells. Carcinoembryonic antigen and alpha-fetoprotein were negative in the fluid. Blood tests revealed 7000 leukocytes/mm<sup>3</sup> with 77% polymorphonuclear cells. Ammonia levels were normal. Abdominal ultrasound revealed peritoneal fluid and splenomegaly. Chest X-ray showed pleural fluid.

The peritoneal fluid samples were inoculated into two blood culture bottles of the Bactec 9240 system (Becton-Dickinson, Bactlab, Israel). Subcultures of peritoneal fluid and blood were performed on blood agar and chocolate agar. Gram-negative coccobacilli grew after 24 hours on choco-

late agar and were identified as *H. parainfluenzae* on the basis of V-factor requirement and absence of hemolysis. Identification of the isolate to species level was confirmed by a rapid biochemical test. The organism was sensitive to ampicillin, amoxicillin/clavulanic acid, cefuroxime, ceftriaxone, chloramphenicol, ciprofloxacin, tetracycline and trimethoprim/sulfamethoxazole.

Initial empiric treatment with intravenous ceftriaxone was replaced by cefuroxime. Transthoracic echocardiogram did not show vegetations or other signs of endocarditis. The patient was treated with intravenous antibiotics for 7 days and continued treatment with oral cefuroxime axetil for another 7 days. A repeat peritoneal fluid sample taken 3 days after the initial one revealed 1000 WBC/mm<sup>3</sup>. Repeated cultures drawn during and at the conclusion of treatment from both peritoneal fluid and peripheral blood were sterile.

### Comment

Spontaneous bacterial peritonitis is commonly caused by enteric Gram-negative bacteria with *Escherichia coli* and *Klebsiella*, responsible for over 50% of cases; however, Gram-positive bacteria including *Enterococcus*, *Streptococcus pneumoniae*, and other *Streptococcus* species are well-documented causative agents. Although cases of primary peritonitis in a healthy child [3] and peritonitis complicating continuous ambulatory peritoneal dialysis [4] caused by *Haemophilus parainfluenzae* have been described, there are no known cases involving this bacterium as the cause of SBP.

At presentation our patient had fewer than 250 polymorphonuclear cells/mm<sup>3</sup> cells in the peritoneal fluid, which after

1 day incubation was positive for *H. parainfluenzae*. At this time the diagnosis of monomicrobial non-neutrocytic bacterascitis was appropriate [5]. Two days later, when the number of polymorphonuclears/mm<sup>3</sup> cells in the fluid rose to more than 250, a diagnosis of SBP was well established. Bacteraemia in this case was probably secondary to SBP. As many as 50% of SBP cases are complicated with bacteraemia [5].

The mechanism by which SBP is caused is thought to involve translocation of intestinal bacteria into the accumulated peritoneal fluid, intestinal bacterial overgrowth and enhanced intestinal permeability augmented by portal hypertension and bowel edema. All these favor the passage of intestinal bacteria into the peritoneal fluid. *H. parainfluenzae* normally colonize the human respiratory tract and are reported to colonize also the gastrointestinal tract. It has a pathogenic role in abdominal infections, particularly in those involving the biliary tract. This may partly explain the role of this bacterium as an atypical SBP causative agent. Our patient had mild diarrhea at presentation and this might have contributed to local bacteria spread.

It is possible that *H. parainfluenzae* SBP may not be so rare. As formerly described regarding CAPD peritonitis cases caused by *H. parainfluenzae* [4], it is possible that some cases of *H. parainfluenzae* SBP are not diagnosed due to non-compatible cultures. This bacterium needs a chocolate agar and factor V (NAD). Although the level of virulence of this organism when causing SBP is unknown it is recommended that the treating physician consider adding

CAPD = continuous ambulatory peritoneal dialysis

SBP = spontaneous bacterial peritonitis

these nutritional requirements to the standard culture, taking into account the high mortality of untreated SBP [5].

## References

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## Capsule

### Immunity and influenza

Seasonal influenza exhibits high morbidity and mortality worldwide, so understanding its phylogenetics and dynamics is important. Despite its high mutation rate, there is limited observed diversity of influenza, perhaps because of generalized strain-transcendent immunity, but there is no evidence for generalized immunity in humans. However, there is evidence of antigenic clusters that sweep through the global human

community between successive seasons. Koelle et al. introduce a phylodynamic model that allows for differences between influenza's genetic and antigenic properties and show that influenza's characteristic phylogeny can arise from cluster-specific immunity alone.

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Eitan Israeli

## Capsule

### Tube feeding in advanced dementia can be harmful

Tube feeding patients with advanced dementia does not prolong life or improve its quality and can actually shorten the life of some patients. In an analysis and comment article, Hoffer reminds us that elderly patients who eat very little are usually not starving but have low energy requirements and exist in a state of meta-

bolic homeostasis. Good practice comprises regularly weighing patients to ensure that weight loss is not life threatening and paying attention to the quality, characteristics and presentation of their food.

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## Capsule

### How cells sort out the trash

When cells accumulate large quantities of proteins that have been damaged (for instance, via modification by reactive oxygen species) or that have not folded properly (e.g., as a result of mutations associated with neurodegenerative diseases), the degradative capacity of the intracellular quality control system can be overwhelmed. Under these conditions, the aberrant proteins collect to form an aggresome, which is an inclusion body situated close to the microtubule-organizing center and just outside of the nucleus. Rujano and co-authors examined the fate of cultured cells containing an aggresome, and of the aggresomes themselves, as the cells divided. Do aggresome-containing cells complete mitosis successfully? Are both daughter cells equally likely to

inherit the parental garbage, or is one daughter preferentially spared? They found that aggresome-containing cells could indeed progress through mitosis productively and that the preexisting aggresome was inherited asymmetrically, yielding daughter cells relatively poor (or rich) in damaged proteins. Furthermore, a survey of cells in the epithelial crypts of the small intestine in two patients with spinocerebellar ataxia (a neurodegenerative disorder) revealed a systematic allocation of the protein inclusions to the short-lived differentiated daughter cells, presumably ensuring the preservation of long-lived stem cells.

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