Rubber band ligation is one of the most widely used and effective treatments for hemorrhoid disease. Hemorrhoidal band ligation is commonly performed as an outpatient procedure and is usually safe. We describe a patient who developed multiple pyogenic liver abscesses following the procedure.

Patient Description
A 64 year old man was admitted with fever. He was generally healthy except for mild asthma that required no regular therapy, and remote history of rash after penicillin exposure. Two weeks before admission the patient underwent hemorrhoidal band ligation in an outpatient clinic. The following day he developed spiky fever and chills. There was mild nausea but no rectal bleeding or discharge. He consulted his primary physician who prescribed amoxicillin therapy, without benefit. Examination in the emergency department on the 14th day of fever revealed an acutely ill man. His temperature was 38.5°C. There was mild localized tenderness over the right flank and the right upper abdominal quadrant. Examination of the head and neck was normal and chest X-ray was normal. Abnormal laboratory findings on admission included: leukocytosis (white blood cells 19,900 K/µl, normal 4500–11,000), anemia (hemoglobin 10.7 g/dl, normal 12–17.5), elevated C-reactive protein (171 mg/L, normal 0–8), and increased sedimentation rate (86 mm/hour, normal 0–20). Serum bilirubin and liver enzymes were not elevated.

Abdominal ultrasound demonstrated at least three intrahepatic hypoechoic non-homogenous lesions surrounded by a hyperechoic rim. Abdominal computed tomography after radio-contrast injection confirmed the presence of multiple hypo dense lesions with ring enhancement [Figure] compatible with multiple liver abscesses. Other scanned regions including the perianal area and the portal veins looked intact. Needle aspiration of two abscesses performed under ultrasound guidance yielded a small quantity of white purulent and necrotic material. Culture of the sample grew *Fusobacterium necrophorum*. Cefuroxime and metronidazole were prescribed. The patient’s condition gradually improved. Repeated culture from the abscesses after one week of therapy was negative. Abdominal CT showed reduction in the size of the abscesses. The patient was discharged home fully recovered after 2 weeks in the hospital, with oral metronidazole 500 mg three times a day and ciprofloxacin 500 mg twice a day.

Comment
We describe a patient who developed multiple pyogenic liver abscesses caused by *F. necrophorum* following hemorrhoidal band ligation. *F. necrophorum*, known to cause septic thrombophlebitis of the jugular veins (Lemierre syndrome), is a normal inhabitant of the gastrointestinal tract. *F. necrophorum* can gain access to the circulation via local penetration of blood vessels and spread via the portal system to produce multiple liver abscesses, a clinical and radiological picture sometimes reminiscent of diffuse malignancy [1]. It can also produce septic thrombosis of the portal vein (pylephlebitis) [2]. While septic complications occur very infrequently following hemorrhoidectomy or sclerotherapy of hemorrhoids, they are even rarer following band ligation. McCloud et al. [3] recently reviewed all published cases of severe septic complications after surgical treatment of hemorrhoids: 17 of 38 developed following rubber band ligation. Most of these patients developed perineal infection.
Less common presentations included septic shock, retroperitoneal infection and liver abscess. Many patients had mixed infection. The most common pathogens recovered in these cases were E. coli (eight isolates). Other common isolates included streptococci [5], other gram negative rods [4], Bacteroides fragilis [4], Staphylococcus aureus [3] and clostridium species [2].

We could find only two previous case reports of liver abscesses due to hemorrhoidal banding [4,5]. Transient bacteremia can occur following hemorrhoid procedures but usually bears no connection to septic complications. Our patient, like most other published cases with multiple liver abscesses due to F. necrophorum, responded to needle aspiration and medical therapy without the need for catheter drainage or surgical intervention.

References

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Capsule
Suppressing emotional memories
Can people suppress emotional memories and, if so, how do they do it? By examining activity in brain regions that support memory processing, Depue et al. provide evidence that an active memory suppression mechanism really exists. First, one portion of prefrontal cortex suppresses regions involved in the sensory aspects of memory. Second, a different part of prefrontal cortex suppresses brain regions that support memory processes as well as those brain areas that support emotional associations with memory. The results may help to explain the lack of control exhibited in a variety of psychiatric disorders over emotional memories and thoughts, and extend our understanding of brain mechanisms that control their formation.

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Capsule
Person-to-person transmission of Nipah virus
Gurley and team investigated an encephalitis outbreak in Faridpur District, Bangladesh, in April-May 2004 to determine the cause of the outbreak and risk factors for disease. Nipah virus was first identified as the pathogen responsible for outbreaks of encephalitis in Malaysia and Singapore from October 1998 to June 1999. Fever (97%), headache (61%), and reduced consciousness (55%) were the most common symptoms in Malaysia; the case-fatality rate was 40%. Most case-patients lived on pig farms (95% in Malaysia) or worked in abattoirs (100% in Singapore). Serological and reverse transcription polymerase chain eaction (RT-PCR) testing of blood and urine from pteropid fruit bats in Malaysia and Cambodia showed Nipah virus infection, which suggested that these animals were reservoir hosts. During this outbreak, Nipah viruses were also isolated from human respiratory secretions and urine; however, two studies did not find evidence of nosocomial transmission. Biological specimens were tested for Nipah virus and surfaces were evaluated for Nipah virus contamination by using RT-PCR. Thirty-six cases of Nipah virus illness were identified, 75% of case-patients died. Multiple peaks of illness occurred, and 33 case-patients had close contact with another Nipah virus patient before their illness. Results from a case-control study showed that contact with one patient carried the highest risk for infection (odds ratio 6.7, 95% confidence interval 2.9–16.8, P < 0.001). RT-PCR testing of environmental samples confirmed Nipah virus contamination of hospital surfaces. This investigation provides evidence for person-to-person transmission of Nipah virus. Capacity for person-to-person transmission increases the potential for wider spread of this highly lethal pathogen and highlights the need for infection control strategies for resource-poor settings.

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