MEDICINE ELSEWHERE

Prepared by

Seda Demiroluk,

M.D., Department of Microbiology, School of Medicine, Marmara University, Istanbul, Turkey.

Eugene Hsin Y, Wen-Chien Ko, Yin-Ching Chuang, Ta-Jen Wu. Suppurative acinetobacter baumanii thyroiditis with bacteremic pneumonia: Case report and review. Clin Infect Dis 1998;27:1286-1290.

Suppurative thyroiditis is rare and the major pathogens are; Staphylococcus and Streptococcus species. This report describes a case caused by Acinetabacter baumanii, which has never before been reported.

In the case; a 70-year-old man who was a heavy smoker with mild obstructive lung disease, had right flank soreness for 2 days followed by severe neck pain, fever and chills. Physical examination revealed a body temperature of 38.5°C and an erythematous nonfluctant mass over the right lobe of the thyroid.

Laboratory data included a WBC conut of 13.900/mm^3 (13%band forms, 83%neutrophils), 6-8 RBCs per high power field (HPF) ad 8-10 WBCs/HPF in urine, a T4 (thyroxine) level of 12.2µg/dL (normal; 4.5-12.5µg/dL) a T3 (triiodothyronine) level of 106.3 ng/dL (80-187ng/dL), a TSH (throid-stimulating hormone) level of 0.24µU/mL (0.5-5.6 U/mL), negative HIV screening and a numerous PMNL in a thyroid aspirate (5mL). The chest radiograph showed hyperinflated lungs, and a radiograph of the abdomen revealed right urether stones with hydronephrosis. Both sonography of the thyroid and CT of the neck showed a hypodense/heterogeneous hypoechoic lesion (3.7cm x 2.7cm) in the right lobe of thyroglossal duct remnants. The iodine 131 (131 I) thyroid scan and the 99mTc sodium pertechnetate neck scan showed no uptake in the thyroid beds. Under the impression of the occurence of suppurative thyroiditis, parenteral ampicillin/sulbactam and gentamicin were administered until both blood and thyroid aspirate cultures revealed A. baumanii growth. At that time the antibiotics were changed to ciproflaxacin and amikacin.

Two weeks later, the chest radiograph showed multiple pneumonic patches with a normal flora in the sputum. The gallium 67 citrate (67Ga) scan showed accumulation of the radiotracer bilaterally in the lower lobes of the lungs (but no uptake in the thyroid). No pathogens were cultured from the initial urine specimen obtained on arrival. By repeated aspiration of the thyroid and 5 weeks' parenteral ciprofloxacin treatment the patient condition improved. The thyroid lesion shrank and the pneumonic patches resolved. Although the origin of A. baumanii was undetermined, authors note a suspicion for an urinary tract origin.

Romanello R, De Santis F, Caione R. A Case of Botulism due to an infected traumatic injury. Eur J Clin Microbiol Infect Dis 1998;17:295-296.

This report is describing a case of botulism after a traumatic injury.

In the case; a 61-year-old farmer was admitted to the emergency department of Trauma Center for treatment of a serious leg injury that occurred while using agricultural equiment. On admission the patient presented with an amputation of the light leg below the knee and the surgery was performed immediately. After surgery antibiotic treatment (Cefizoxime 3g/day) was started and anti-gangrene serum was also administered.

On day 7, the patient temperature was 39°C and a crepitus in the stump appeared, attributed to gas gangrene. Treatment with i.v. imipenem (3g/day), netilmicin (300mg/day) and metronidazole (1.5g/day) was started and followed by a single hyperbaric oxygen treatment. 3 days later, the patient condition worsened; respiratory distress ensued, followed by the appearance of other neurological symptoms; including ptosis, extrinsic and intrinsic bilateral ophthalmoparesis, hyposthenia of the limbs and dysphagia. The neurological diagnosis was myastheniform syndrome of a toxic nature. 2 days later, the patient was transferred to the intensive care unit. He presented with fever, slurred speach, and signs of dehydration as well as previous neurological symptoms. Treatment consisted of reopening the stump, draining the pus, debriding the necrotic skin, cleaning first with hydrogen peroxide and then with antiseptic solutions, plugging the open stump with iodeformic gauze and administering i.v. metronidazole. Hyperbaric oxygen therapy was repeated.

After 24 hours, ventilatory support and nasotracheal intubation were required to relieve increasing respiratory difficulties. Cranial computerized axial tomographic scan and brain nuclear magnetic resonance image were normal.

Electroencephalograms showed irregular peaks. Due to persistance of the neurological symptoms, a test was carried out with prostigmine, with negative results. A presumptive diagnosis of botulism was made. The patient was treated with antibotulinal serum ABE.

A fragment of necrotic materials was taken for culture and serum samples were analysed at the National Centre for Botulism Diagnosis, by standard methods. The cultures of necrotic material were negative for C. botulinum, but serum samples were positive for type B botulinal toxin.

After 20 days, the patient was discharged from hospital without any sign of residual neurogical disability.

The authors mention that; the presence of recent injury site and a history lacking ingestion of spoiled canned foods, as well as development of cranial nerves palsies should alert the physician to the possibility of wound botulism. Suitable treatment which include debridement of the wound, removal of the devascularized tissue that would facilitate anaerobic conditions and ventilatory support care must be implemented promptly to prevent death due to respiratory failure. According to their opinion, botulinal antitoxin should be administered as soon as possible after onset of symptoms.

Prepared by

Berrak Ç. Yeğen,

M.D., Department of Physiology, School of Medicine, Marmara Universiy, İstanbul, Turkey

Scott RB, Kirk D, MacNaughton WK, Meddings JB. GLP-2 augments the adaptive response to massive intestinal resection in rat. Am J Physiol 1998;275 (Gastrointest Liver Physiol 38): G911-G921.

Short bowel syndrome is a clinical condition characterized by rapid intestinal transit and malabsorption in individuals who have had a congenital or, more commonly, an acquired lesion leading to intestinal resection. All layers of the bowel wall have been shown to participate in a hyperplastic adaptive response to intestinal resection. There are structural, functional, and cytokinetic adaptations of the mucosa, with augmentation of mucosal surface area. Identification of factors regulating the adaptive hyperplastic response to resection would improve the clinical care of patients with short bowel syndrome and reduce their utilization of health care resources. A previous report of a patient with a glucagonoma who presented with massive enlargement of the small bowel that abated after surgical removal of the tumor, prompted analysis of the relationship between intestinal proglucagon-derived peptides and intestinal epithelial proliferation.

To determine whether treatment with a potent protease-resistant analog of human glucagon-like peptide 2 (GLP-2) might increase the adaptive response to massive intestinal resection, rats were divided into resected (75 % of the midjejunoileum removed), sham resected, and nonsurgical groups. Within each group, animals were assigned to 21 days of treatment with the GLP-2 analog (0.1 μg/g) or the vehicle alone subcutaneously twice daily. Food intake, weight gain, jejunal and ileal diameters, total and mucosal wet weights per centimeter, crypt depths, and villus heights, mucosal sucrase activity, milligrams of protein per centimeter and micrograms of DNA per centimeter, and D-Xylose absorption were measured.

There was a significant increase in diameter, total and mucosal wet weights per centimeter, crypt-villus height, sucrase activity, milligrams of DNA per centimeter in both the jejunum and ileum in response to resection and a significant additive response to the GLP-2 analog in the jejunum but not in the ileum. The ratio of *mg of protein/cm* to *μg of DNA/cm* of mucosa was not different among groups, consistent with hyperplasia. In response to resection, D-Xylose absorption was significantly reduced; however, the GLP-2 analog enhanced the absorptive capacity in control animals and restored the reduced capacity in resected animals.

Thus the GLP-2 analog induced mucosal hyperplasia and significantly enhanced both the rate and the magnitude of the proximal intestinal adaptive response to massive resection. The lack of benefit of current therapeutic modalities and the incremental trophic effect of the GLP-2 analog compared with the adaptive response to resection alone suggests that a well-designed therapeutical trial of the GLP-2 analog in human short bowel syndrome is warranted. Success in such a human trial would potentially reduce both the morbidity and mortality of individuals with short bowel syndrome.

Prepared by

Hızır Kurtel

M.D., Department of Physiology, School of Medicine, Marmara University, İstanbul, Turkey

Boros M, Massberg S, Branyi L et. al. Endothelin-1 induces leukocyte adhesion in submucosal venules of the rat small intestine. Gastroenterology 1998;114:103-114.

The endothelins (ETs) are a family of 21-aminoacid peptides produced by vascular endothelial cells. Endothelin-1 (ET-1) is the most powerful vasoconstrictor known to date, and it may participate in regulation of the smooth muscle tone in many systems. The plasma levels of ET-1 are enhanced during experimental and clinical ischemia, hypoxia, sepsis, and bacteremia suggesting that it may mediate vasoconstriction of regional vascular beds during these conditions. In addition to this, it has been proposed that, the release of ET-1 and activation of leukocytes are involved in the pathophysiology of gastrointestinal I/R injuries. The aim of his study was to define the in vivo relation between ET-1 and endothelial cell-leukocyte interactions.

Anesthetized rats were studied to characterize the microvascular effects of increasing doses of local and systemic infusions of ET-1 in all layers of an ileal segment. Leukocyte-endothelial interactions were monitored with intravital fluorescence videomicroscopy. The ET-A receptor-selective antagonist BQ610, the novel ET-A receptor antagonist ETR-PL/fl peptide, and the ET-B receptor antagonist IRL1038 were used to investigate the roles of receptor subtypes.

The functional capillary density of the mucosa was decreased by intravenous ET-1. After 30 minutes, the rolling fraction of leukocytes reached 90% in the postcapillary venules, and the number of adherent leukocytes was increased after 90 minutes. ET-A and ET-B receptor antagonists, inhibited the ET-1 induced leukocyte rolling however, ET-A antagonists were more effective than ET-B antagonist. Both ET-A receptor antagonists prevented ET-1 induced firm adhesion.

In conclusion, the result of this study indicates that ET-1 might act as a promoter of leukocyte rolling and adhesion through a predominantly ET-A receptor mediated mechanism in the submucosal venules of the intestinal circulation. These data, together with the observations of elevated plasma levels during certain pathologies, suggest a therapeutic potential for ET-receptor antagonists for the inhibition not only of microvascular dysfunction but also of interactions of PMNs with endothelial cells.

Prepared by

İnci Alican

M.D., Ph.D., Department of Physiology, School of Medicine, Marmara University, İstanbul, Turkey

Wallace JL, Bak A, McKnight W, Asfaha S, Sharkey KA, MacNaughton WK. Cyclooxygenase 1 contributes to inflammatory responses in rats and mice: Implications for gastrointestinal toxicity. Gastroenterology 1998;115:101-109.

The use of nonsteroidal anti-inflammatory drugs (NSAIDs) for treatment of inflammation and pain remains limited by their ability to cause ulceration and bleeding in the gastrointestinal tract. The characterization of two isoforms of the principal enzyme responsible for prostaglandin (PG) synthesis, cyclooxygenase (COX), has led to the proposal that selective inhibitors of the inducible isoform (COX-2) would be effective anti-inflammatory and analgesic agents without causing gastrointestinal injury. This proposal is based on the finding that the major isoform of COX expressed in the normal stomach is COX-1, whereas COX-2 is expressed only at very low levels in most tissues but is rapidly induced at sites of inflammation.

In the present study, the anti-inflammatory effects of a number of agents with varying degrees of in vitro selectivity of COX-2 vs. COX-1 (NS-398, nimesulide, DuP697 and etodolac) were compared and these drugs were examined whether they could reduce inflammation independent of effects on COX-1. Finally, the effects of a standard NSAID (indomethacin) and a selective COX-2 inhibitor (NS-398) on inflammatory responses in mice in which the gene for COX-2 had been disrupted were determined.

Male Wistar rats were used to assess the selectivity of COX-2 inhibitors (NS-398, nimesulide, DuP697 and etodolac) on carrageenan- induced paw inflammation. The role of COX-1 in inflammation was also assessed in COX-2-deficient mice.

Significant anti-inflammatory effects were only observed at doses of the drugs that inihibited COX-1. At these doses, the drugs also significantly

suppressed gastric prostaglandin synthesis and elicited gastric mucosal erosions. The degree of suppression of prostaglandin synthesis at the site of inflammation correlated significantly with inhibition of COX-1 but not COX-2.

The results of this study suggest that although selective blockade of COX-2 may spare gastrointestinal PG synthesis and therefore not cause de novo injury, it may also be less effective in reducing inflammation compared with what can be achieved with combined inhibition of COX-1 and COX-2. At least in some cases, PGs derived from COX-1 contribute significantly to the generation of inflammation. In such cases, significant anti-inflammatory effects are only observed at doses of drugs that produce inhibition of both COX-1 and COX-2. At these doses the drugs are capable of significantly suppressing gastric PG synthesis, causing gastric damage and, as shown previously, exacerbating preexisting gastrointestinal damage. These studies therefore raise questions regarding the premises on which the development of highly selective inhibitors of COX-2 as gastrointestinal-sparing anti-inflammator agents are based.

MEETINGS

22 - 26 September 1999, Snowmass Village Colorado, USA

Biology of Potassium Channels: From Molecules to Disease

Contact: The American Physiological Society 9650 Rockville Pike, Bethesda MD 20814 - 3991
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E-mail: meetings@aps.faseb.org

26 - 29 September 1999, San Francisco, California, USA

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25 - 30 October 1999, Antalya, Turkey

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Contact: Assoc Prof. Dr. Muzaffer Sindel
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Tel.: 90 242 227 44 85 Fax: 90 242 227 44 82
E-mail: anatomi@med.akdeniz.edu.tr

7 - 11 November 1999, Chicago Il, USA

American Public Health Association

127th Annual Meeting and Exposition Continuing Education Program 1999

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15 - 20 July 2000, Florence, Italy

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Tel.: + 39 - 045 - 500408/8074899 Fax: +39 - 045 581111
E-mail: gpvelo@farma.univr.it

ANSWER TO PHOTO QUIZ

Bilateral solid masses which are highlighted by surrounding ascites replace both ovaries. There are segmental wall thickening and a mass projecting into the lumen of the corpus of the stomach. This was confirmed by multiple biopsies via esophagogastroduodenoscopy. Biopsy result revealed gastric adenocarcinoma. At laparotomy, enlarged ovaries containing signet-ring, mucine-secreting cells in an abundant stroma were found. This is Krukenberg tumor which is a metastatic ovarian tumor of the GI tract cancer (colon:stomach=2:1). It may also arise by the spread of other mucinous adenocarcinoma such as those of the breast, pancreas and gallbladder. Krukenberg tumor occurs in 2-7% of females with gastric cancer. These tumors antedate the discovery of the primary lesion in up to 20%. It comprises 6% of malignant ovarian tumors.

ACADEMIC HOSPITAL

Altunizade Nuhkuyusu Cad. No: 88 Üsküdar Tel.: (0.216) 341 28 41

MARMARA ÜNİVERSİTESİ TIP FAKÜLTESİ VAKFI

Academic Hospital

Altunizade Nuhkuyusu Cad. No: 88 Üsküdar
Tel.: (0.216) 492 47 50 Fax: (0.216) 492 47 62

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