

as volume overload and blood borne infection. We report our experience using Recombinant Factor VIIa (rFVIIa) as a safe adjunct to traditional therapies for coagulopathy due to hepatic synthetic failure. **Methods:** A retrospective review of in-patient pharmacy records from 4/2000 to 12/2001 identified 14 patients who were treated with rFVIIa for coagulopathy due to liver disease. The mean initial dose used was 85.2 ± 13.9 mcg/kg. The medical records of these patients were reviewed to identify patient demographics, indications for therapy, immediate response to rFVIIa infusion, and patient outcome. Mean prothrombin time (PT) results before and after therapy were compared by paired t-test. **Results:** The indication for rFVIIa in all 14 patients was on-going coagulopathy despite treatment with FFP. The mean PT at initial presentation was 37.4 seconds, with a range of 23.9 to > 60 seconds (INR 3.30 to > 15.3). 8 patients had coagulopathy due to acute liver failure and 6 due to chronic end stage liver disease. Administration of the first dose of rFVIIa resulted in correction of the PT from a mean of 29.4 ± 4.3 seconds prior to therapy to 13.6 ± 2.4 seconds ($p < 0.0001$) at 1 hour after infusion and 20.1 ± 5.5 seconds ($p = 0.0011$) at 6 hours after infusion. On-going rFVIIa therapy resulted in sustained improvement in the PT, with mean values for the following 3 days ranging from 18 to 20 seconds. 7/14 patients had clinically significant bleeding despite maximal therapy with FFP and platelets. The remaining 7 were treated empirically for refractory coagulopathy to limit the risk of spontaneous hemorrhage. Of the 7 with bleeding, 6 had subjective improvement, with 2 of these being successfully weaned to < 15% of their original FFP support. Thromboembolic or other adverse events related to rFVIIa were not observed. **Conclusions:** rFVIIa can be a useful adjuvant for coagulopathy and bleeding associated with liver failure. Patients in this series experienced a significant improvement in PT during the first 6 hours after infusion. No thrombotic complications were seen. Prospective studies will be necessary to better define clinical indications, efficacy, and cost-effectiveness of this treatment.

Gastrointestinal biopsy results of 10 patients with chronic abdominal pain, heartburn, mesenteric adenitis, "striae" like skin rash, history of cat scratches and a positive Ig-G titer for *B. henselae*

Age (years)	Gastric Biopsy	Duodenal Biopsy
11	gastritis, <i>B. henselae</i> PCR (+)	duodenitis, <i>B. henselae</i> PCR (+)
16	no pathology	duodenitis, <i>B. henselae</i> PCR (+)
14	gastritis, <i>B. henselae</i> PCR (+)	duodenitis, <i>B. henselae</i> PCR (+)
13	gastritis, <i>B. henselae</i> PCR (+)	duodenitis, <i>B. henselae</i> PCR (+)
7	gastritis, <i>B. henselae</i> PCR (+)	duodenitis, <i>B. henselae</i> PCR (+)
15	gastritis, <i>B. henselae</i> PCR (+)	duodenitis, <i>B. henselae</i> PCR (+)
15	gastritis, <i>B. henselae</i> PCR (+)	no pathology
13	no pathology	duodenitis, <i>B. henselae</i> PCR (+)
7	gastritis, <i>B. henselae</i> PCR (+)	no pathology
9	no pathology	duodenitis, <i>B. henselae</i> PCR (+)

Conclusion- *Bartonella henselae* appears to be associated with a striae-like skin rash, abdominal pain, heartburn, gastritis, duodenitis and mesenteric adenitis in the pediatric population.

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BARTONELLA HENSELAE IS ASSOCIATED WITH HEARTBURN, ABDOMINAL PAIN, SKIN RASH, MESENTERIC ADENITIS, GASTRITIS AND DUODENITIS

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Purpose- To investigate *Bartonella henselae* (*B. henselae*) as a pathogen associated with gastritis and duodenitis in children and adolescents.

Methods- From July 2001 through March 2002, ten patients between the ages of 7 and 16 years presented with a history of cat scratches, chronic abdominal pain, esophageal heartburn for at least two months, a striae-like skin rash, mesenteric adenitis (greater than one cm in diameter) on CT scan of the abdomen and a positive Immunoglobulin G titer (1:64 or higher) for *B. henselae*. Endoscopy assessed the gastrointestinal mucosa for inflammation and biopsies were examined for *H. pylori* by microscopy and for *B. henselae* by polymerase chain reaction (PCR).

Results- Biopsies were PCR positive for *B. henselae* DNA in all with chronic abdominal pain, heartburn, positive Immunoglobulin G titers for *B. henselae*, skin rash and mesenteric adenitis. Chronic gastritis and or chronic duodenitis was found in all and associated with the detection of *B. henselae* DNA in the GI tract at the site of the inflammation.

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FLUCTUATION OF LEVELS OF TRANSGLUTAMINASE AUTOANTIBODIES IN GENETICALLY AT-RISK CHILDREN IN RELATION TO A POSITIVE CELIAC DISEASE BIOPSY

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With the development of quantitative transglutaminase autoantibody assays large scale screening for celiac disease is now practical. Relatively large numbers of asymptomatic children and adults express transglutaminase autoantibodies. It is not uncommon however to identify a child whose intestinal biopsy is negative. This has led to the suggestion that these sensitive autoantibody assays may have a low positive predictive value with the "gold standard" of celiac disease on biopsy. Our analysis of cohorts of genetically at risk children suggests an alternative explanation, namely that the celiac disease process fluctuates as reflected by the levels of transglutaminase autoantibodies and that levels of these autoantibodies are highly correlated with biopsy results at the time of biopsy. We have prospectively followed two genetically at risk cohorts of children, patients with type 1A diabetes and children from the general population HLA typed at birth and expressing DQ2. Once transglutaminase autoantibodies appeared they remained in most individuals above the 100th percentile of normal controls (index >0.05, n=184 samples). Despite remaining positive, the levels fluctuated dramatically (10 to 100 fold) with repeat sampling between 3 months and 1 year. Twenty of 21 children whose transglutaminase autoantibody level exceeded an index of 0.5 at the time of the biopsy had biopsy-confirmed celiac disease. Of the children with a negative biopsy (n=11), only 1 had a transglutaminase autoantibody level greater than 0.5 at the time of biopsy, despite having markedly higher transglutaminase levels greater than 0.5 months prior to the biopsy (the reason for scheduling the biopsy). Those with transglutaminase autoantibody levels greater than 0.5 at the time of biopsy were more likely to have a positive small bowel biopsy than those with levels less than or equal to 0.5 (20/21 or 95% biopsy positive compared to 3/13 or 23% ($p < 0.0001$)). Of those with indeterminate biopsies, 1 of 4 had a transglutaminase auto-