Understanding the dynamics of gastro-intestinal syndrome: general practioner and hospital data vs laboratory surveillance

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OBJECTIVE

Understanding the baseline dynamics of syndrome counts is essential for use in prospective syndromic surveillance. Therefore we studied to what extent the known seasonal dynamics of gastro-intestinal (GI) pathogens explain the dynamics in GI syndrome in general practitioner (GP) and hospital data.

METHODS

Retrospective, weekly GI syndrome counts from two medical registration systems in the Netherlands were assessed: 1) Signs and symptoms of patients visiting GP's, coverage: 1-2% (2001-2003). 2) Hospital main discharge diagnoses, coverage: 100% (1999-2004). A GI syndrome was defined for each registration using the CDC syndrome grouping of ICD9 codes as a guidance. Trends in positive diagnoses of known GI pathogens were abstracted from national laboratory surveillance which includes the following 5 GI pathogens: rotavirus, *Campylobacter* (coverage 50%), *Shigella* (coverage 100%), and *Salmonella* (typhi and non-typhi; coverage 64%).

We used linear regression models to characterize the relationship between the GI syndrome trends and the known GI laboratory surveillance trends, also stratified by age. Besides the current pathogen counts we also evaluated lagged values (up to 5 weeks backwards and forwards in time) of all GI pathogens as explanatory variables. For multivariate models a forward stepwise regression approach was used, building each increment in the model by adding all possible lags of all pathogens and selecting the pathogen with the best fit (assessed with akaike's information criterion). Each pathogen was included in the model maximally once. Negative associations were not included.

RESULTS

Visually, seasonal variation in GI syndrome is seen in both GP (broadly spread elevations in winter and smaller ones in summer/autumn) and hospital diagnoses (peaks in winter). Due to very high colinearity between several of the GI pathogens (R's: below -0.68 and above 0.70) and the resulting modeling problems we eventually assessed the effect of 3 pathogens: rotavirus, *Shigella*, and *Salmonella* Typhi. The explained variance was not very high in either model: R-square GP data: 0.29 (all 3 pathogens included in final model, hospital data: 0.40 (only rotavirus included in the final model (fig.1), but greatly increased to 0.51 and 0.85 respectively when limiting analysis to children younger than 5 years of age. In this age group rotavirus was the sole significant predictor of GI syndrome, and the best fit was achieved with the rotavirus laboratory counts set 2 weeks later than syndrome counts in GP data and 1 week later in hospital data.

CONCLUSIONS

In the general population, laboratory counts of GI pathogens account for a relatively low level of explained variance in GI syndrome, while in young children only rotavirus was associated with GI syndrome. Thus, the syndrome group as it is currently defined, may be of limited value for aberration detection, but, we should bear in mind that the traditional pathogen surveillance is itself not complete. The unavailability of counts of norovirus and pathogens causing travellers diarrhea could underlie the low level of explained variance. In that case, it may be the traditional pathogen surveillance (based on the available pathogens) that might be of limited additional value in understanding and verifying aberration signals in prospective GI syndromic surveillance. The current GI syndrome does precede laboratory rotavirus counts by 1 to 2 weeks in both the younger and general population, indicating a potential for earlier detection. Furthermore, the actual performance of GI syndromes in detecting outbreaks needs to be ascertained. This should be done at the regional level by using known and documented point source or regional outbreaks of GI pathogens.

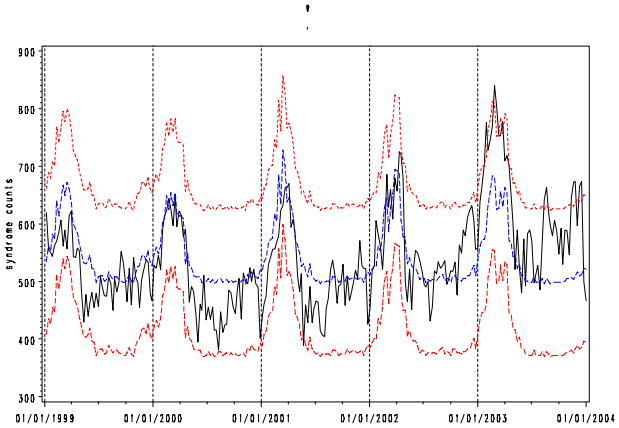

fig 1. Gastro-intestinal syndrome (black line) & predicted GI syndrome (blue line) in hospital data (confidence limits in red), in the general population.