Causal Impact of the Hospital Readmissions Reduction Program on Hospital Readmissions and Mortality

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Abstract

Estimating causal effects of the Hospital Readmissions Reduction Program (HRRP), part of the Affordable Care Act, has been very controversial. Associational studies have demonstrated decreases in hospital readmissions, consistent with the intent of the program, although analyses with different data sources and methods have differed in estimating effects on patient mortality. To address these issues, we define the estimands of interest in the context of potential outcomes, we formalize a Bayesian structural time-series model for causal inference, and discuss the necessary assumptions for estimation of effects using observed data. The method is used to estimate the effect of the passage of HRRP on both the 30-day readmissions and 30-day mortality. We show that for acute myocardial infarction and congestive heart failure, HRRP caused reduction in readmissions while it had no statistically significant effect on mortality. However, for pneumonia, HRRP had no statistically significant effect on readmissions but caused an increase in mortality.

keywords: causal inference, time-series, policy evaluation, hospital readmissions reduction program

1 Introduction

The passage of the health care reform in 2010 included the establishment of the Medicare Hospital Readmissions Reduction Program (HRRP). Although Medicare had been publicly reporting hospital risk-standardized readmission rates since 2009, passage of the HRRP in 2010 created the prospect of financial penalties based on risk-standardized readmission rates that would start being applied to payments in 2012. In the initial phase, the HRRP included readmissions for Medicare fee-for-service patients within 30 days of discharge after index hospitalization for acute myocardial infarction (AMI), congestive heart failure (CHF), or pneumonia. As part of a movement from volume to value, the HRRP reflected intent to incentivize providers and hospitals to improve the coordination and quality of care for patients.

Despite that conceptual promise, much debate exists about the efficacy and potential adverse consequences of the HRRP [Joynt et al., 2011, Joynt and Jha, 2012]. Previously, it was shown that passage of the law in 2010 was associated with substantial declines in readmissions [Zuckerman et al., 2016, Desai et al., 2016, Wasfy et al., 2017, MedPAC, 2018]. Whether the law was associated with increased mortality is very controversial [Fonarow and Yancy, 2017, Fonarow et al., 2017]. An analysis of Medicare claims did not find evidence of a statistically significant association between hospital-level
reduced readmissions and increased mortality \cite{Dharmarajan2017}, in contrast to a study of CHF patients from a clinical registry which found evidence of an increase in mortality after the passage of the law \cite{Gupta2017}.

While interesting, the data and analysis techniques used to produce these results limit the ability to interpret these associations as explicit causal effects of the HRRP. In fact, previous studies have been restricted to pre-post analyses of (risk-standardized or not) readmission rates \cite{Wasfy2017} or interrupted time-series analysis \cite{Desai2016}. Improving our understanding of the causal effects of the HRRP on patient outcomes is critical, because this type of information will inform whether the policy should be continued, expanded, or stopped \cite{Shakir2018}. From a health policy perspective, the effectiveness of the HRRP lies in how successful the law was at reducing readmissions, while, at the same time, not having increased mortality, which would be a price too significant to bear. Therefore, two key questions persist in the evaluation of the HRRP: What was the causal effect of the passage of the law (the intervention) on the number of 30-day hospital readmissions and 30-day mortality, for the reporting quarters following the intervention in 2010, and until 2014, the last available year of data? In other words, how did the observed readmissions and mortality compare to what would have happened absent the passage of the law?

In this paper, we formulate these questions (i.e., specify the causal estimands of interest) within the potential outcomes approach to causal inference using time-series potential outcomes representing the readmissions and mortality in the presence of the intervention, which is observed, and in the absence of the intervention, which is not observed and has to be estimated. The data we use and the potential outcomes we consider are constructed to represent all hospitals eligible for penalty (and therefore the vast majority of the hospital system), and they are each in the form of one time-series representing the total number of readmissions (or deaths) across all hospitals (see discussion in Section 4.2). Doing so allows us to estimate the effect of the passage of the law on the whole hospital system rather than at the individual hospitals. We formalize and discuss assumptions necessary for identification of the causal estimands using observed data, in general, but also in the context of our study. Then, we present the previously-developed Bayesian structural time-series model of \cite{Brodersen2015}. The model is used to predict the missing potential time-series (the time-series that would have been observed absent the intervention) using local and seasonal trends, and covariate time-series as predictors of the outcome. Causal estimates are obtained comparing the imputed time-series under no intervention to the observed time-series.

Our approach has important commonalities and differences to previously developed causal inference methods in time-series analysis. The difference in differences approach (e.g. \cite{Athey2006}) assumes that time-series data are available on observations some of which are treated, and linear trend is assumed pre- and post-intervention. Availability of observations with both treatment levels is also assumed in synthetic control approaches \cite{Doudchenko2017}, where a weighted average of the time-series from other (untreated) individuals is used to impute the missing potential outcome of a treated unit in the absence of the treatment. In our case, all the available hospitals are “treated” since all of them experienced the passage of the law and were eligible for penalization under the HRRP, and the available data are in the form of a single treated time-series. For the analysis of data including solely one time-series, interrupted (or quasi-experimental) time-series analysis is often used, allowing for and incorporating more complex trends, such as seasonal variations. Causal inference with one time-series was recently formalized, for which an interrupted time-series model was suggested \cite{Bojinov2017}. \cite{Bojinov2017} assume that the treatment can be applied and taken away at any time point, whereas here we consider a treatment (HRRP passage) that once initiated it remains active throughout. Even though our approach, proposed by \cite{Brodersen2015}, has many similarities to the interrupted time-series model for causal inference, there are important differences with respect to the causal quantities of interest, and the use of covariates. In the approach presented here, covariate time-series which are not affected by the intervention and are predictors of the outcome time-series are used to improve efficiency in the prediction of the missing potential time-series in the absence of the intervention.
2 Data sources and construction of the data set

The study population and variables used in this analysis are the same as in previously published analyses [Wasfy et al., 2017], but now they have been updated with data for the year 2014. We initially queried the Medicare Provider and Analysis Review (MedPAR) files to identify all Medicare fee-for-service beneficiaries aged 65 years or older hospitalized in an acute care hospital in the United States for AMI, CHF, or pneumonia from January 1, 2000 through November 30, 2014. Hospitalizations for AMI, CHF, or pneumonia were defined as hospitalization with a principal discharge diagnosis of an International Classification of Diseases, Ninth Revision, Clinical Modification (ICD-9-CM) code for those conditions. These codes are used for both public reporting and financial penalties [Krumholz et al., 2006, Bratzler et al., 2011]. We restricted the sample to patients enrolled in the fee-for-service plan at least 12 months prior to an index hospitalization and continued in the fee-for-service plan for at least 1 month following the index discharge.

Patient demographic information included age, sex, and major comorbidities, including 11 cardiovascular history variables and 26 other variables that represent other comorbidities. Clinical variables were obtained from secondary diagnosis codes in the index condition-specific hospitalization as well as from principal and secondary diagnosis codes from all hospitalizations for 12 months before the index hospitalization. These comorbidities were classified using the Hierarchical Condition Categories method [Pope et al., 2000]. Inpatient claims from 1999 were used to obtain comorbidities for patients hospitalized in 2000. These variables include the variables used by CMS to risk-standardize readmission rates.

We obtained publicly-available hospital penalty information [CMS, 2018]. CMS combined the hospital-specific diagnosis-related group (DRG) payments and hospital-specific excess readmission ratios for AMI, CHF, and pneumonia to determine penalties [CMS, 2018]. We excluded hospitals not in the CMS HRRP penalty list; many of these hospitals were small and acquired by other hospitals during our study period. Only hospitals that existed during the entire time period were included in this analysis.

The primary outcomes of this analysis were total numbers of quarterly 30-day all-cause readmissions for patients discharged with AMI, CHF, or pneumonia, as well as the total number of 30-day all-cause mortality for AMI, CHF, or pneumonia hospitalized patients. We also created a combined-condition total readmissions (deaths) outcome as the sum of all-cause readmissions (deaths) for patients initially admitted with one of the three conditions. To permit complete 30-day follow-up, we used December 2014 inpatient data to obtain readmission information for patients discharged in November 2014. As in previous analyses [Zuckerman et al., 2016, Wasfy et al., 2017], we defined the intervention as passage of the health reform law on March 23, 2010, also discussed in Section 4.1. Given that readmission rates are estimated quarterly, we approximate the law passage as the end of the first calendar quarter (March 31, 2010).

3 Bayesian time-series for evaluation of causal effects

Our analytic goal is to estimate the causal impact of the HRRP on the total number of readmissions and deaths across all hospitals eligible for penalty. For that purpose, we formalize the Bayesian methodology introduced by Brodersen et al. [2015] within a potential outcome framework similar to the one in Bojinov and Shephard [2017], and discuss the necessary assumptions for identification of the causal estimands based on observed data.

3.1 Notation, potential time-series and estimands of interest

Since the total number of readmissions/deaths across all hospitals that were eligible for financial penalty is of interest, our data are in the form of one time-series describing the total number of readmissions/deaths of Medicare patients for all hospitals combined (remember that the hospitals excluded from the CMS HRRP penalty list are mostly small and are excluded from this analysis). Consider $t \in \mathcal{T} = \{1, 2, \ldots, T\}$ be the 3-month intervals that correspond to calendar year quarters for which data are available (from 1999 Q1 to 2014 Q4). Let $t^* \in \mathcal{T}$ denote the first interval following the announcement of the HRRP in March 2010 (2010 Q2).

In order to formally define potential outcomes and causal estimands in this framework, let $W_t$
denote the treatment status at time point \( t \in \mathcal{T} \), and \( \overline{W}_t = (W_1, W_2, \ldots, W_t) \) denote the treatment history up to time point \( t \). Further, denote \( w_t, \overline{w}_t \) as a realization of \( W_t, \overline{W}_t \). Then, we postulate the existence of potential outcomes of the form

\[
Y^t(\overline{w}_t), t \geq t^*
\]

for two sequences of treatments; one corresponding to the outcome that would have been observed at time point \( t \) had the treatment not been initiated,

\[
\overline{w}_t, \text{ such that } w_j = 0, \forall j \in \{1, 2, \ldots, t\},
\]

and one corresponding to the outcome that would have been observed at time point \( t \) had the treatment been initiated at time point \( t^* \),

\[
w_t, \text{ such that } w_t = \begin{cases} 0 & \text{if } t < t^* \\ 1 & \text{if } t \geq t^* \end{cases}.
\]

The potential outcomes \( \{Y^t(\overline{w}_t), t \geq t^*\} \) can be thought of as potential time-series for a vector of treatments \( \overline{w}_T \). Note that the context of our data analysis requires the postulation of potential time-series for treatment vectors of the form \([1],[2]\) only, indicating that if the intervention is initiated, the time-series remains treated for all future time points. Evaluating the effect of the HRRP passage on the outcome of interest at all time points following program announcement corresponds to the comparison of the potential time-series for the two treatment levels. Causal estimands can target the outcome change at the \( k^{th} \) time point after the intervention (lagged effect)

\[
\Delta_k = Y^{t^*-1+k}(W_{t^*-1}, 0, 0, 0, \ldots, 0, 1, 1, \ldots, 1) - Y^{t^*-1+k}(W_{t^*-1}, 0, 0, 0, \ldots, 0, 0, 0, \ldots, 0),
\]

or the cumulative effect over \( K \) time-points, defined as

\[
C\Delta_K = \sum_{k=1}^{K} Y^{t^*-1+k}(W_{t^*-1}, 0, 0, 0, \ldots, 0, 1, 1, \ldots, 1) - Y^{t^*-1+k}(W_{t^*-1}, 0, 0, 0, \ldots, 0, 0, 0, \ldots, 0) = \sum_{k=1}^{K} \Delta_k.
\]

In our data application, causal effects of the type \([3]\) correspond to the change in the number of readmissions (deaths) caused by the passage of HRRP at \( k \) quarters after the program passage, whereas causal effects of the type \([4]\) correspond to the change in the number of readmissions (deaths) caused by the passage of HRRP cumulatively for \( k = 1, \ldots, K \) quarters after the program passage.

Let \( Y_t \) denote the observed outcome at time point \( t \in \mathcal{T} \), representing the total number of readmissions or deaths observed across all hospitals included in the CMS HRRP penalty list. Assuming consistency of the potential outcomes \([\text{Robins et al., 2000}]\), we have that \( Y_t = Y^t(\overline{W}_t) \), indicating that the observed outcome is exactly the potential outcome for the observed treatment path \( \overline{W}_t \). Since treatment in our data was initiated at time point \( t^* \), all potential outcomes of the form \( Y^{t^*-1+k}(W_{t^*-1}, 0, 0, 0, \ldots, 0, 1, 1, \ldots, 1), k \in \{1, 2, \ldots, T-t^*+1\} \) are indeed observed. Therefore, estimation of the causal quantities in \([3],[4]\) can proceed by estimation of the potential outcomes \( Y^t(0, 0, \ldots, 0), t \geq t^* \).

### 3.2 Bayesian time-series analysis for estimation of missing potential outcomes

Since estimation of the causal quantities can be based solely on the estimation of \( Y^t(0, 0, \ldots, 0), t \geq t^* \), a fully Bayesian structural time-series model is built for prediction of the unobserved potential outcomes: the outcome time-series that would have been observed in the absence of the intervention for the time period after the intervention initiation at time point \( t^* \). Prediction of potential outcomes is based on two sources of information:

1. The observed outcomes pre-intervention \( Y_t(0, 0, \ldots, 0), t < t^* \) capturing temporal and seasonal trends in the absence of the intervention, and
2. Covariate time-series that are unaffected by the intervention but are potentially correlated with the outcome time-series pre-intervention, and they will be used to improve precision of the potential outcome time-series imputation post-intervention.

The model formulation (Brodersen et al. [2015] for static covariate coefficients, see therein for more details) consists of the state-space model where the outcome \( y_t \) depends on covariate time-series \( x_t \) and latent variables capturing various trends:

\[
y_t = x_T^T \beta + \mu_t + \gamma_t + \epsilon_t
\]

where \( \epsilon_t \sim N(0, \sigma^2) \) independent. The latent states \( \mu_t, \gamma_t \) are introduced to represent local and seasonal trends of the outcome time-series accordingly. The locally linear \( \mu_t \) is specified as

\[
\mu_{t+1} = \mu_t + \delta_t + \eta_{\mu,t}
\]

\[
\delta_{t+1} = \delta_t + \eta_{\delta,t},
\]

where \( \eta_{\mu,t} \sim N(0, \sigma^2_{\mu}) \), \( \eta_{\delta,t} \sim N(0, \sigma^2_{\delta}) \) independent. In this model, \( \delta_t \) represents the overall trend of the outcome time-series with variations from the overall mean allowed through the introduction of \( \eta_{\mu,t} \), incorporating overall trend information in the potential outcome prediction. This specification of a mean trend represents a highly flexible model adaptive to local variations that does not assume much smoothness on how the time series varies over time.

Seasonal trends are also incorporated in the model representing seasonal variation in the outcome time-series. If \( S \) seasons are assumed, seasonal trends are incorporated by considering the latent variable \( \gamma_t \) in the following form

\[
\gamma_{t+1} = -\sum_{s=0}^{S-2} \gamma_{t-s} + \eta_{\gamma,t},
\]

summing over the last \( S - 1 \) seasons, ensuring that over \( S \) seasons the aggregate contribution of \( \gamma \) is centered at zero.

Here, it is worth noting that estimation of the local and seasonal trends is informed by the (untreated, observed) outcome time-series over the time period before the intervention. The approach assumes that the local and seasonal trends in \([3]\) and \([5]\) would have persisted had the intervention not occurred, and they are therefore used in predicting the missing potential time-series. It is therefore necessary to specify the intervention time \( t^* \) such that the intervention and potential outcomes are well-defined, and the outcome time-series has not already “reacted” to the anticipation of the intervention at \( t < t^* \). The plausibility of this assumption when evaluating the HRPP is discussed in Section 4.1.

The model as described up to now (without the use of covariates \( x_T^T \beta \)) uses the outcome time-series pre-intervention to identify local linear and seasonal trends in order to predict the potential time-series post-intervention, and in the absence of intervention. A novelty of the approach by Brodersen et al. [2015] is the use of covariate time-series as predictors of the outcome time-series, which can improve efficiency of predictions. For example, the mean age of the population might be a useful predictor of the number of readmissions, and by incorporating such information might lead to more efficient estimators of the missing potential outcomes.

Incorporation of covariate time-series with time-constant association is performed by including the term \( x_T^T \beta \) in the mean structure of the observation model \( y_t \). If covariate time-series are unaffected by the intervention and they are predictive of the outcome time-series in the absence of the intervention, their use in the observation model will improve efficiency in the estimation of the missing potential outcomes. However, if such covariate time-series are, in reality, affected by the intervention, they should not be included in the prediction model, since prediction of the missing potential outcomes based on such covariate time-series will include part of the intervention effect (the effect that is mediated through change in the covariates). In terms of potential outcome notation, the necessary assumption for the inclusion of covariates in the model is that such covariate time-series are not
affected by the intervention, and therefore
\[ X_t^{w_t} = X_t^{w_t} \]
where \( X_t^{w_t} \) represents the value that would have been observed for covariates \( X \) at time point \( t \) under the treatment vector \( w_t \). Section 4.2 discusses the importance of using the total number of readmissions across all hospitals as the outcome time-series in ensuring that the above assumption holds for all covariates included in the analysis.

4 Evaluation of the Hospital Readmissions Reduction Program

4.1 The HRRP passage as the intervention time point

The model for the prediction of the missing potential time-series is formulated including local and seasonal trends which are estimated based on the observed outcome time-series at \( t < t^* \). If the observational unit has “anticipated” the intervention and reacted to it before time point \( t^* \), the observed potential outcomes used in the estimation of these trends will not correspond to potential outcomes in the absence of the intervention.

Since a lot of hospitals responded to the program announcement before program initiation, it is important that we consider that the intervention was initiated at the time point of the HRRP passage (or before it) in order to represent the totality of the effect of HRRP. We assume that hospitals did not react to the HRRP before the law passed.

4.2 Plausibility of the unaffected covariate assumption in the HRRP evaluation

If covariates are used in the potential outcome prediction model, these covariate time-series should be unaffected by the intervention. Covariates which can be used as predictors in the observation model include variables such as patients’ age, race, and sex. Previous studies have argued that penalization of hospitals might have led penalized hospitals to avoid patients with certain characteristics, which are afterwards accepted for care in a different (non-penalized) hospital. Therefore, if the analysis included the number of readmissions among only penalized hospitals as the outcome time-series, the inclusion of covariates such as age in the model would not be appropriate, since the age of patients initially admitted in the penalized hospitals might have been affected by the intervention and does not hold.

However, in our study the outcome of interest is the total number of readmissions across all hospitals that are eligible for penalty. By considering such an aggregate measure, instead of number of readmissions within penalized and non-penalized hospitals separately, the assumption that covariates such as demographics are unaffected by the intervention becomes plausible, since a patient’s visit and admission to any hospital can be assumed to not be affected by the announcement of the HRRP.

4.3 HRRP evaluation in terms of number of readmissions and patient mortality

We estimated the effect of the passage of the HRRP on the total number of readmissions and deaths for patients initially admitted for any of the three conditions (AMI, CHF, pneumonia), and for each condition separately.

For each of the four conditions (combined conditions, AMI, CHF, pneumonia) and each outcome time-series (number of deaths, number of readmissions) we fit the model presented in Section 6 to estimate the counterfactual time-series in the absence of the HRRP passage, representing predictions for the potential outcomes \( Y^t(0,0,\ldots,0), t \geq t^* \). We include yearly seasonal trends in number of deaths and hospitalizations by considering 4 seasons within the year.

Evaluation of the model fit is performed based on the predictions of the potential outcomes for \( t < t^* \) corresponding to observed potential outcomes in the absence of the intervention. Based on the counterfactual predictions, pointwise effect estimates of \( \Delta_k \) in (3) can be acquired by contrasting the observed post-intervention time-series in the presence of the intervention to the estimated post-intervention time-series in the absence of the intervention. Pointwise contrasts represent the effect of HRRP on the outcome time-series of interest for each time point \( t \geq t^* \). These estimates are aggregated over the whole post-intervention period providing an estimate of \( C \Delta_k \) in (4), the cumulative effect of the intervention over the time-period \([t^*, t^* + K], K = 1,2,\ldots,T - t^* \).

Figure 1 depicts all model estimates and corresponding 95% credible intervals. First, we examine the concordance of predictions with the observed outcome time-series in the pre-intervention period.
Figure 1: Bayesian time-series results for the evaluation of the HRRP on number of deaths and readmissions for patients originally admitted for any condition (combined conditions - CC), acute myocardial infarction (AMI), congestive heart failure (CHF), or pneumonia (PNE). For each combination of condition and outcome, results include (a) ‘original’: observed time-series (solid line) and counterfactual predictions of the number of deaths/readmissions in the absence of the interventions (dashed) with 95% credible intervals (shaded area), (b) pointwise predictions of the effect of HRRP at every time point pre- and post-intervention, and (c) the cumulative effect of HRRP on deaths/readmissions.
Table 1: Posterior means and 95% posterior credible intervals of the number of readmissions and deaths caused (if estimate is positive) or avoided (if estimate is negative) for each condition, during the whole study period and across all hospitals eligible for penalty.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Combined conditions</th>
<th>AMI</th>
<th>CHF</th>
<th>Pneumonia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Readmissions</td>
<td>-113,070 (-185,834 to -34,648)</td>
<td>-25,071 (-38,372 to -11,005)</td>
<td>-82,596 (-159,969 to -39,904)</td>
<td>-4,675 (-38,969 to 29,848)</td>
</tr>
<tr>
<td>Mortality</td>
<td>15,925 (-6,520 to 76,300)</td>
<td>5,829 (-5,024 to 12,680)</td>
<td>3,222 (-7,077 to 16,481)</td>
<td>15,604 (2,145 to 29,404)</td>
</tr>
</tbody>
</table>

Table 2: Posterior probability of the presence of a causal effect of the HRRP on number of readmissions and deaths for combined conditions and each condition separately.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Combined conditions</th>
<th>AMI</th>
<th>CHF</th>
<th>Pneumonia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Readmissions</td>
<td>99.3%</td>
<td>99.4%</td>
<td>≈ 100%</td>
<td>62%</td>
</tr>
<tr>
<td>Mortality</td>
<td>91%</td>
<td>90%</td>
<td>91%</td>
<td>98.7%</td>
</tr>
</tbody>
</table>

The predicted time-series (dashed line in the “original” panel) consistently overlaps with the observed time-series (solid line) during the pre-intervention time period. As a result, we can confidently claim that the model performs well in fitting the local and seasonal trends. From the same panel, we also see that the posterior credible intervals for the counterfactual predictions in the post-intervention period are getting wider as we look further into the future, depicting the increasing uncertainty for predictions at time points further from the observed data.

Pointwise estimates for the effect of HRRP on the combined conditions, AMI and CHF readmissions indicate that the program led to a significant decrease in the number of readmitted patients for at least one time point after program announcement. The effect is larger for later time points indicating that the hospitals progressively adjusted to the HRRP.

Table 1 presents the cumulative effect estimates of the HRRP on the number of readmissions and deaths for each condition. For the combined conditions, from the second quarter of 2010 to the last quarter of 2014, a total of 113,070 readmissions were avoided thanks to the program, without any significant change in the number of readmissions for patients initially admitted for pneumonia. These results are in line with previous associational work. On the other hand, the mortality effect estimates for the combined conditions, AMI and CHF indicate an increase in the number of deaths due to the HRRP, with 15,925 more deaths (95% credible interval: 6,520 less deaths to 76,300 more deaths) for patients initially admitted for AMI, CHF or pneumonia than what would have been observed in the absence of the program. However, these effects are not statistically significant. The HRRP led to a significant increase in the number of deaths for patients initially admitted for pneumonia, with 15,604 (95% posterior credible interval 2,145 – 29,404) additional deaths attributable to the HRRP.

Significance results are also reflected in the posterior probabilities of the presence of a causal effect in Table 2. In this table, the entries reflect the posterior probability that the causal effect is larger than zero (for positive estimates) or smaller than zero (for negative estimates), corresponding to posterior probabilities of one-sided hypothesis tests. In fact, the posterior probability of an effect of the HRRP on the number of readmissions was > 95% for all conditions but pneumonia, while the reverse is true for the effect of the HRRP on the number of deaths.

5 Discussion

In this paper, we formalized the causal estimands and identifiability assumptions on which the previously-developed Bayesian structural time-series model of Brodersen et al. [2015] was based. Before the model was used, we provided a comprehensive interrogation of the assumptions underlying the estimation of such causal estimands in the context of our study. Then, we evaluated the effect of the HRRP passage on the number of readmissions and deaths of patients initially admitted for AMI, CHF, or pneumonia. Therefore, this paper provides formalization of the assumptions that need to be met in the estimation of causal effects in a time-series setting, and guidance on how to properly verify them, laying the foundation for the use of this method in a wide range of applications assessing clinical outcomes for health policy in non-randomized settings.

To our knowledge, this is the first paper evaluating the effect of the HRRP in a principled causal
inference framework. As has been similarly seen in previous associational studies, the HRRP was successful in reducing the number of readmissions compared to what would have been observed in the absence of the program, with a total of 113,070 readmissions avoided across all conditions. Even though the HRRP led to reductions in the number of readmissions, the analysis presented here raises concerns about both increased mortality and ineffective readmission reduction for pneumonia patients. This indicates that the program evaluation and potential future amendments need to take into consideration the differential regulations’ impact on various outcomes among patients with different conditions.

These results are critically important for several reasons. Previously published analyses have suggested that after enactment of the HRRP, readmissions have decreased for all conditions and for each of the 3 initial penalty conditions considered separately [Desai et al., 2016; Zuckerman et al., 2016; Wasfy et al., 2017]. In this analysis using causal inference methods, we demonstrate that these conclusions are robust for AMI and CHF but not for pneumonia. From a perspective of evaluation of the actual policy, this suggests a need to better understand why the HRRP was more effective at reducing readmissions for AMI and CHF than for pneumonia patients. Furthermore, these discrepant results illustrate how challenging policy evaluation can be in non-randomized populations, since results can be sensitive to different assumptions and statistical techniques.

In the setting of these heterogeneous results, we suggest a few potential steps forward. Further research could seek to better understand the causal pathways from HRRP to increased mortality for pneumonia patients, with the goal to assess a plausible causal mechanism for increased mortality. Alternatively, time-series analysis of the semi-competing risks of mortality and readmission could provide further intuition on the relative risks and benefits of HRRP while treating the two outcomes of interest simultaneously.

References


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