Does Milnepan Cause a Reduction in 7-Day Mortality Among Heffpox Patients?

A Causal Inference Analysis Using IPTW and G-formula

Statistical Modelling and Inference for Health

1. Introduction

Heffpox is an acute infectious disease with considerable mortality rates; hence, it requires effective treatment to reduce mortality. The current analysis aims to evaluate if the treatment of milnepan reduces 7-day mortality in patients with heffpox based on 8,192 confirmed cases of the disease.

In observational studies, treatment assignment is not random; instead, doctors might be more inclined to treat younger and healthier patients with milnepan, resulting in confounding bias where it is not possible to establish whether the treatment or the patients' characteristics lead to better outcomes.

In observational studies, it is not possible to establish the effect of the treatment without confounding bias; hence, it is not possible to establish the actual effect of the treatment without any bias.

Research Question: Does the treatment of milnepan reduce 7-day mortality in patients with heffpox?2. Data Description

2.1 Study Population

The data set initially consisted of 8,889 patients. However, 697 patients were dropped from the dataset due to the absence of confirmed heffpox, leaving 8,192 patients in the study population. Of this figure, 3,948 (48%) of the patients were given milnepan, while 4,244 (52%) were not given milnepan.

Using R with dplyr package: heffpox_only <- heffpox %>% filter(heffpox == 1). This removed 697 patients without heffpox.

2.2 Variables

The study examined the effect of milnepan treatment, represented as a simple 1-0 binary variable. Patients received a 1 if they received the treatment and a 0 otherwise. Outcome of interest: 7-day mortality, defined as death within seven days of diagnosis. The choice of seven days is a natural threshold to evaluate the effectiveness of the treatment of heffpox patients.

To perform exploratory survival analysis, we also experimented with time-to-event data, for example, the number of days from diagnosis to death or end of follow-up (maximum of 10 days). The data included an event variable to specify that the person died or that the data were for censored observations.

Five baseline confounders were identified using clinical reasoning and were then verified using a Directed Acyclic Graph analysis. These were age, sex, body mass index, smoking status, and diabetes. These factors were considered potential confounders since they were deemed to affect the odds of taking milnepan and mortality.

Importantly, the effect of admission to intensive care unit was clearly identified as a post-treatment variable that is along the causal path between treatment and death, and since adjusting for this could mask part of the effect we want to measure, it was deliberately left out.

2.3 Baseline Characteristics

In order to determine whether both the treatment groups begin with similar circumstances, we check for baseline differences. Where there is systematic divergence, crude comparisons become unfair. Fortunately, we use Standardized Mean Differences (SMD). If the SMD measures above 0.1, it becomes significant imbalance.

Image 1: R Code for Baseline Characteristics using TableOne function.

```r
# TABLE 1 ON FILTERED DATA (HEFFPOX PATIENTS ONLY)
# Variables for Table 1 (removed heffpox since everyone has it now)
all_vars <- c("age", "bmi", "sex", "smoking", "diabetes", "icu",
              "TEVENT", "Status", "death7day")

cat_vars <- c("sex", "smoking", "diabetes", "icu", "death7day", "Status")

# Create Table 1
table1_filtered <- CreateTableOne(
  vars = all_vars,
  strata = "milnepan",
  data = heffpox_only,
  factorVars = cat_vars,
  addOverall = TRUE
)

print(table1_filtered, smd = TRUE, showAllLevels = TRUE)
```

Table 1 indicates the characteristics of the study population at baseline. There were notable uneven distributions in each category of the study groups. Patients who received milnepan treatment were on average younger than those who did not receive milnepan (mean ages 39.8 years vs. 48.5 years; SMD = 0.511). Secondly, patients who received milnepan were less likely to have diabetes than those who did not receive milnepan (5.7% vs. 14.8%; SMD = 0.303). Other variables such as sex, BMI, and smokers were reasonably distributed (all SMDs <= 0.1).

From these results, it is evident that treatment assignment was not random. It appears that clinicians favored milnepan for younger and less comorbid patients. As such, any crude mortality comparison of the treatment groups would be confounded, and differences in outcomes could be considered as resulting from differences in covariates rather than treatment per se. This reinforces the importance of making proper causal inference to show true treatment effect.

Table 1: Baseline Characteristics by Treatment Group

Variable	Untreated (n=4,244)	Treated (n=3,948)	SMD
Age, mean (SD)	48.5 years	39.8 years	0.511
Female, %	49.8%	52.9%	0.062
BMI, mean	27.8	28.1	0.041
Smoking, %	27.8%	24.4%	0.069
Diabetes, %	14.8%	5.7%	0.303
7-day mortality, %	44.7%	35.7%	-

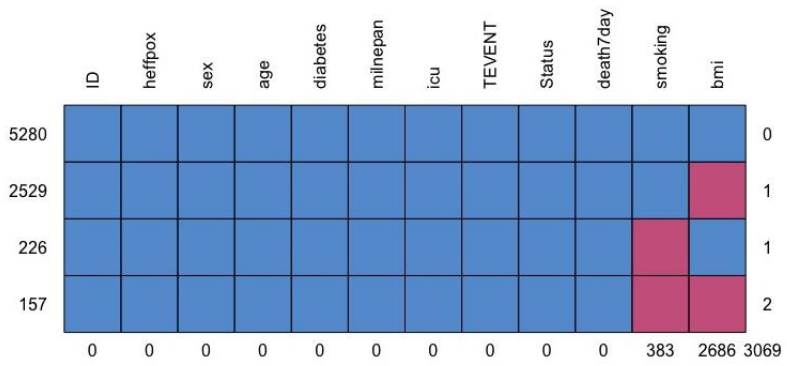
3. Missing Data Analysis

3.1 Missing Data Patterns

Before plunging into the analysis, we first examined the level of missing data. In other words, we sought to understand where we were experiencing the missing data. In this regard, the rationale lies in the fact that dropping all the patients with non-complete data might create a bias that diminishes the power of the statistics.

The two variables that had missing data were BMI, which had missing data on 2,686 patients (32.8%), and smoking status, which had missing data on 383 patients (4.7%). For the other variables, there were no missing data. The results of the pattern check indicated that 5,280 patients (64.5%) had data for all variables. Among patients who had some missing data, 2,529 patients (30.9%) were missing only BMI, 226 patients (2.8%) were missing only smoking status, and 157 were missing both BMI and smoking status.

Figure 1: Missing Data pattern



3.2 Missing Data Mechanism

For determining the best way to handle missing data, we first investigated what mechanism was operating: MCAR, MAR, or MNAR. This is important, because multiple imputation depends upon MAR - the notion that missing data can be accounted for based on variables we have seen in the dataset.

We investigated whether the missingness of BMI was related to other observed variables. As it happened, subjects with missing BMI values were older than those with measured BMI (means 50.1 versus 41.5 years old). Missing values of BMI were more frequent for untreated patients (34.7%) compared to treated patients (30.7%), and in patients who had died (34.0%) compared to survivors (32.0%).

These systematic links of missingness with the observed characteristics point out that data are not MCAR. Instead, the pattern now agrees with MAR-the missingness is related to some observed variables, but not to the unobserved values of BMI themselves. This fact supports the multiple imputation as an appropriate approach for the given analysis when handling the missing data.

3.3 Multiple Imputation

In order to fill in the missing data gaps, where data was missing completely at random, we used multiple imputation with the mice package in the R program (Multivariate Imputation by Chained Equations). This was done instead of single imputation because it recognizes the uncertainty in missing data more accurately by imputing multiple sets of data and pooling the results after imputation.

We generated 20 imputed datasets with 20 imputations (m = 20). This satisfies the recommendation of at least as many imputations as the percent of missing information. Given the situation in this problem in which a third of the data were missing, 20 imputations were adequate. We allowed 10 iterations (maxit = 10) in the imputation process to permit convergence.

We used Predictive Mean Matching for the imputation of both BMI and smoking status. The benefits of using PMM include the fact that it replaces missing values with values that come from similar individuals in a dataset, thereby creating values for missing response variables that are realistic. The random seed is also used for reproducibility purposes.

To verify that they converged, they were also examined with trace plots of imputed values at varying iterations. In this case, the trace plots did not have any underlying trend and hence suggested good mixing and adequate convergence. Also, density plots were used for comparison of observed and imputed values, which suggested that imputed values were also reasonable.

Figure 2: Convergence Diagnostics Plot.

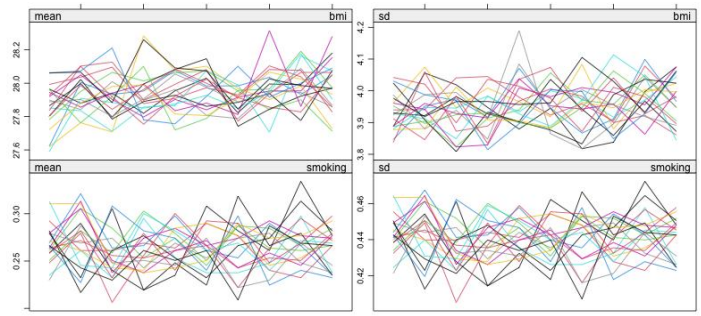


Figure 3: Density Plot for Smoking

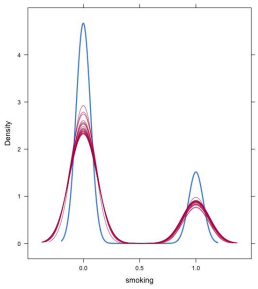
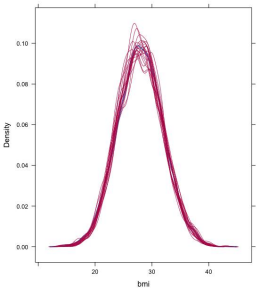


Figure 4: Density Plot for BMI



4. Methods

4.1 Causal Framework: Directed Acyclic Graph (DAG)

To create an appropriate causal framework for our analysis, we have created a figure through a data structure called a "Directed Acyclic Graph," or DAG, that describes how we believe the different factors relate to one another. The use of a DAG has helped to identify our causal assumptions and what factors should and should not be adjusted for.

Based on clinical understanding of what factors affect heffpox and treatment allocations, we have identified potential confounders: age, sex, BMI, smoking, and diabetes, which could potentially affect receipt of treatment milnepan and risk of dying within 7 days, which needs to be controlled for.

ICU admission was viewed as a post-treatment variable and was considered to fall on the causal path between milnepan and death. This is because the process reduces severe illness, consequently reducing ICU admissions and thereby mortality risk, and not incorporating this in the adjustment set would show one portion of the total treatment effect. Therefore, ICU was not included in the adjustment set.

We constructed the DAG using the R package 'dagitty' and confirmed computationally whether the minimal sufficient adjustment set had been identified. The analysis revealed that control for variables including age, sex, BMI, smoking, and diabetes is sufficient to make all backdoor paths between treatment and outcome independent of confounding variables, thereby enabling an unbiased assessment of treatment effect.

Image 2: R Code for DAG creation

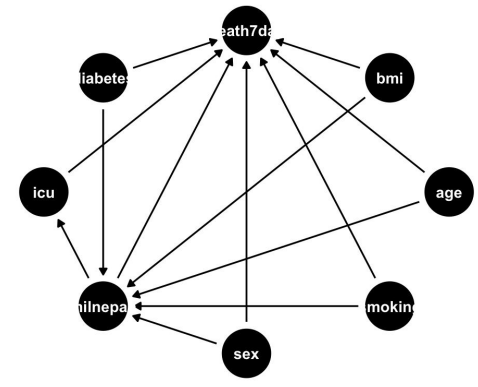
```r
install.packages("dagitty")
install.packages("ggdag")
library(dagitty)
library(ggdag)

dag <- dagitty('dag {
  age -> milnepan
  age -> death7day
  sex -> milnepan
  sex -> death7day
  bmi -> milnepan
  bmi -> death7day
  smoking -> milnepan
  smoking -> death7day
  diabetes -> milnepan
  diabetes -> death7day
  milnepan -> icu
  milnepan -> death7day
  icu -> death7day
}')

exposures(dag) <- "milnepan"
outcomes(dag) <- "death7day"

ggdag(dag, layout = "circle") + theme_dag() +
  ggtitle("Causal DAG: Effect of Milnepan on 7-Day Mortality")
```

Figure 5: CAUSAL DAG creation



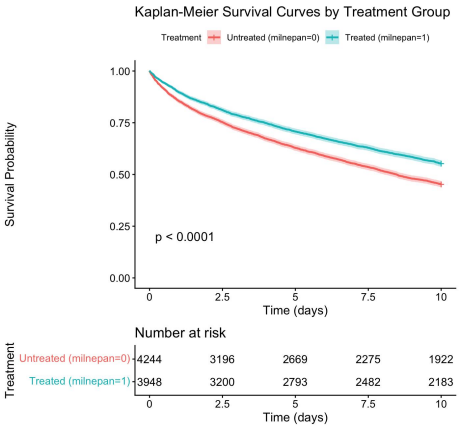
4.2 Exploratory Survival Analysis

Prior to conducting the main causal analysis, we performed exploratory survival analysis to examine the time-to-event data and assess the impact of confounding and missing data on treatment effect estimates.

Kaplan-Meier Analysis

Figures for survival using the Kaplan-Meier method were prepared for each of the groups receiving treatment and were then compared using the log rank test method. Survival was significantly better for milnepan than for the untreated groups (log rank P<0.0001). Of the untreated patients, 54.7% had died by the end of the trial period, and their median survival was 8.47 days. Of the patients in the treated group, 44.7% had died by the end of the trial period, and the median survival time had not been reached, meaning that over half of these patients were still alive at the end of the trial period.

Figure 6: Kaplan-Meier Survival Curves



Cox Proportional Hazards Regression

To assess the treatment effect and understand the impact of confounding and missing data, we fit three different Cox proportional hazards models: a crude or unadjusted model, a model that was adjusted but used only complete cases, and a model that was adjusted and accounted for missing data based on the use of multiple imputations, followed by applying Rubin's rules.

Table 2: Cox Proportional Hazards Models

Model	Hazard Ratio	95% CI	p-value	n
Unadjusted	0.744	0.70 - 0.79	< 0.0001	8,192
Adjusted (confounders)	0.946	0.87 - 1.03	0.190	5,280
Adjusted (imputed)	0.918	0.86 - 0.98	0.012	8,192

The first model, before any adjustments were made, indicated a significant treatment effect (HR 0.744, p < 0.0001), representing a 26% reduction in mortality risk. However, when adjusting for confounders, using a complete case analysis, our hazard ratio dropped significantly to 0.946, failing to reach statistical significance (p = 0.190). The important point here, though, is that 35.5%, or 2,912, were missing because they lacked covariate data.

When we ran the adjusted Cox model on the multiply imputed dataset and included all 8,192 patients, the treatment effect reappeared as statistically significant (HR 0.918, 95% CI: 0.86 to 0.98, p = 0.012). This equates to a risk reduction of 8.2%.These findings underscore several key issues. First, the descriptive effects represented confounding by differences in baseline between populations taking milnepan and natural population samples from which this population more frequently was drawn from—a generally healthier sample by virtue of being younger.

Second, the complete case analysis represents confounding by incomplete information from the sample population—a large proportion of the population being left out from this study.

4.3 Inverse Probability of Treatment Weighting (IPTW)

Inverse Probability of Treatment Weighting (IPTW) is a causal inference method that creates a pseudo-population in which treatment assignment is independent of measured confounders, thereby mimicking the balance achieved in a randomised controlled trial.

The first step involved estimating the propensity score for each patient, defined as the probability of receiving milnepan treatment conditional on observed confounders. This was modelled using logistic regression with age, sex, BMI, smoking status, and diabetes as predictors. Propensity scores ranged from 0.061 to 0.734, with adequate overlap between treatment groups, satisfying the positivity assumption required for valid causal inference.

Image 3: R Code to calculate PS for each patient.

```r
# - Result: Treatment groups become balanced!
install.packages("WeightIt")
install.packages("cobalt")
install.packages("survey")
library(WeightIt)
library(cobalt)
library(survey)

# starting with one dataset
data_imp1 <- complete(imp, 1)

#using logistic regression
ps_model <- glm(milnepan ~ age + sex + bmi + smoking + diabetes,
                data = data_imp1,
                family = binomial)

summary(ps_model)


# add propensity score to the data
data_imp1$ps <- predict(ps_model, type = "response")

summary(data_imp1$ps)
```

Based on this, we were then able to compute a set of stabilized weights for every patient. To compute the Average Treatment Effect, patients who were treated were given a weight that is the inverse of their propensity score, and patients who were not treated were given a weight that is the inverse of (1 – their score). In this way, factors that were confounding were balanced.

Image 4: R Code to calculate IPTW for each patient.

```r
# CONCLUSION: Adequate overlap exists. IPTW analysis can proceed.


weights_out <- weightit(
  milnepan ~ age + sex + bmi + smoking + diabetes,
  data = data_imp1,
  method = "ps",
  estimand = "ATE"
)

summary(weights_out)


# Check balance before and after weighting
bal.tab(weights_out, un = TRUE, stats = c("m", "v"))

# Love plot - shows balance before vs after
love.plot(weights_out,
          binary = "std",
          thresholds = c(m = 0.1),
          colors = c("red", "blue"),
          shapes = c("circle", "triangle"),
          title = "Covariate Balance Before and After IPTW")



# Survey design with IPTW regression
install.packages("survey")
library(survey)

design_iptw <- svydesign(ids = ~1, weights = weights_out$weights,
                         data = data_imp1)

# Fit weighted logistic regression for 7-day mortality
model_iptw <- svyglm(death7day ~ milnepan,
                     design = design_iptw,
                     family = quasibinomial())

summary(model_iptw)


# Get Odds Ratio and 95% CI
exp(coef(model_iptw))
exp(confint(model_iptw))

# I used the survey package to fit a weighted logistic regression.
```

We checked if the weighting process had successfully achieved balance for confounders by observing the standardized mean differences before and after weighting. Before weighting, imbalance was apparent, especially for age variables (SMD = 0.511) and diabetes (SMD = 0.303). However, after applying IPTW weights, all SMDs went below the threshold of 0.1.

The weighted outcome model was then fitted using logistic regression, with 7-day mortality as the outcome and treatment as the sole predictor. Standard errors were adjusted for the weighting using the survey package in R. To properly account for missing data, the entire IPTW procedure was applied to each of the 20 multiply imputed datasets, and results were pooled using Rubin's Rules

4.4 G-formula (Standardisation)

The G Formula, otherwise called standardisation or parametric g computation, is yet another means to analyse the data for causal effects. IPTW confronts confounding through weighting, whereas the G Formula does so indirectly through outcome modelling. By utilizing these two techniques, we might triangulate the outcomes. Since both approaches are investigated from a different foundation, we derive more confidence from consequent similar outcomes.

Here's how the G-formula works. To begin with, the outcome model that incorporates the treatment effect and all the relevant variables is specified. To be specific, a logistic regression model with 7-day mortality as the outcome, and predictors milnepan, age, sex, BMI, smoking, and diabetes were used.

With this model in mind, we proceed to create two hypothetical worlds: one where we pretend every subject actually took the treatment (i.e., milnepan = 1) despite their actual assignment, and another where we pretend that in our world, no one took the treatment (i.e., milnepan = 0). For each of our subjects, we compute the probability of death for each of these worlds.

An average of the predicted probabilities for all patients yields the estimate of the population risk for each scenario. The causal effects can be expressed as the difference or ratio of these risks for treatment and control scenarios. To determine the confidence intervals for the risk ratio, a bootstrap method with 1,000 resamples was used.

Consistent with the IPTW analysis, we conducted G-formula analysis on each of the 20 multiply imputed datasets and combined the results by Rubin's Rules.

4.5 Pooling Results Across Imputations

Because imputation itself involves uncertainty, we carried out both IPTW and G-formula analyses separately on each of the 20 imputed data-sets and pooled the results using Rubin's Rules-that is, the standard way of combining results across multiply imputed data.

According to Rubin's Rules, the pooled point estimate is simply the average of the estimates obtained from all imputations. The pooled variance has two components: the within-imputation variance-this is just the average of the variances from each imputed dataset-and the between-imputation variance, which reflects the variability of the point estimates from imputation to imputation. There is also a small-sample adjustment factor that reflects using a finite number of imputations. This approach to pooling ensures that the final confidence intervals capture both the ordinary sampling uncertainty and the extra uncertainty arising from imputing missing values.

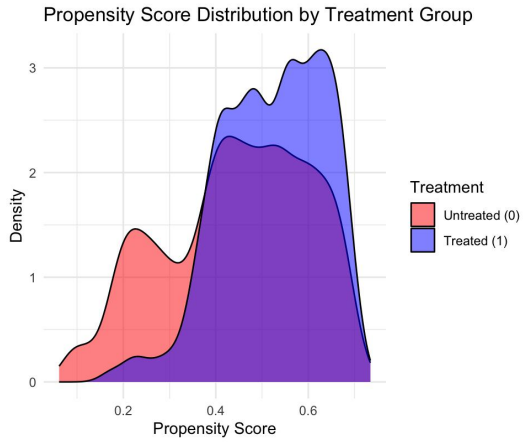
5. Results

5.1 Propensity Score Estimation

The propensity scores, which represented the likelihood of each patient receiving milnepan based on observed confounders, varied from 0.061 up to 0.734, averaging at 0.482. This wide range shows that the observed confounders are powerful predictors for receiving the treatment.

If we examine how these scores are distributed across each group receiving treatment, it appears that there is significant overlap between groups, supporting this positivity assumption that is important when making causal inference. The distribution of those who were not administered treatment appears to bunch at around 0.2 or 0.3, illustrating that these patients are less likely to receive treatment. Finally, patients who were administered treatment bunch at about 0.5 or 6. There is significant overlap between these two distributions at values between 0.2 and 0.7..

Figure 7:Propensity Score Distribution Plot



5.2 Covariate Balance After IPTW

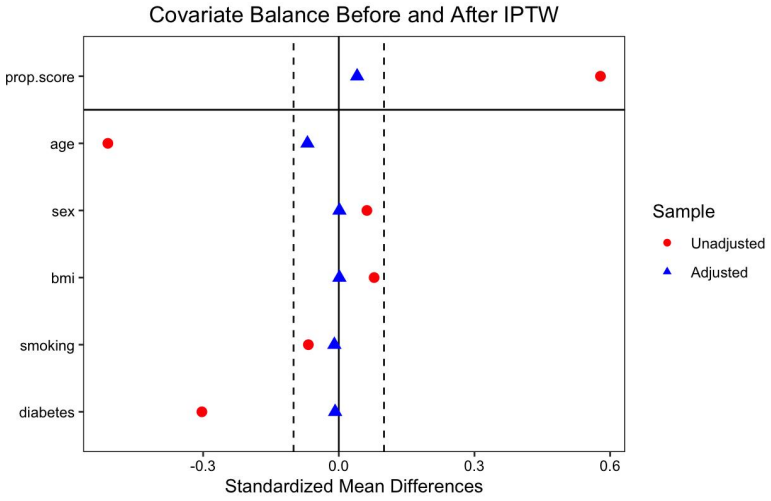
The success of IPTW depends on achieving adequate balance of confounders between treatment groups after weighting. We assessed balance by comparing standardised mean differences (SMD) before and after the application of weights.

Table 3: Covariate Balance Before and After IPTW

Variable	SMD Before	SMD After	Status
Age	-0.511	-0.069	BALANCED
Sex	0.031	0.001	BALANCED
BMI	0.078	0.001	BALANCED
Smoking	-0.029	-0.004	BALANCED
Diabetes	-0.091	-0.002	BALANCED

Prior to weighting, substantial imbalance existed for age (SMD = -0.511), indicating that treated patients were considerably younger than untreated patients. After applying IPTW weights, all standardised mean differences fell below the conventional threshold of 0.1, indicating successful balance across all measured confounders. The Love Plot visually confirms that all covariates moved within the acceptable range following weighting.

Figure 8: Love Plot for covariant balance to show before and after IPTW



5.3 Weight Diagnostics

To assess how reliable these IPTW results were, the distribution of the weights used was evaluated, as well as the sample size. The weights varied from 1.07 up to 7.23, with values well below the usual threshold, which is between 10 or 20, in regard to extreme values in such a context.

The effective sample size (ESS) gives us insight into how much precision is lost by working with weighted data. It turned out to be 3,919 for our control group data and 3,636 for our treated group data. This was a loss of 7.6% and 7.9%, respectively, from our unweighted data. As long as this loss is less than 10%, information loss is kept at a minimum, and our weighting scheme has had negligible impact on efficiency.

5.4 Causal Effect Estimates

Both IPTW and G-formula were applied to each of the 20 multiply imputed datasets, with results pooled using Rubin's Rules to appropriately account for missing data uncertainty. The causal effect estimates are presented in Table 4.

Table 4: Final Causal Effect Estimates (Pooled Across 20 Imputations)

Method	Effect Estimate	95% CI	Interpretation
IPTW	OR = 0.876	0.798 - 0.961	12.4% lower odds
G-formula	RR = 0.922	0.877 - 0.969	7.8% lower risk

Using the IPTW, it was found that the pooled odds ratio was equal to 0.876 (95% CI, 0.798–0.961). This means that patients receiving treatment with milnepan were 12.4% less likely to die within 7 days, compared with patients that were not receiving treatment. We have excluded 1 from the confidence interval.

The pooled calculated risk ratio for G-formula was 0.922, with 95% CI ranging from 0.877 to 0.969. If everyone were treated, the 7-day mortality would be 38.65%; and if no one were treated, it would be 41.93%. This gives an ARR of 3.28% and an RRR of 7.8%.

Both approaches to causal inference agree that, on one hand, milnepan has a significant impact in reducing 7-day mortality among heffpox patients. There is consistency across different approaches.

6. Discussion

6.1 Interpretation of Findings

Nevertheless, both causal inference techniques point to the same fundamental conclusion: milnepan decreases 7-day mortality for heffpox. IPTW estimates a 12.4 percent reduction in odds (OR 0.876), while the G formula indicates a 7.8 percent fall in risk (RR 0.922). Each model estimation proves statistically significant, with confidence intervals far removed from null effects.

In understanding this better, it helps to examine the change in results from model to model. The unadjusted hazard ratio of 0.744 overestimates the effect due to confounding by age and diabetes status. The estimate obtained from the complete case cox model, though not significant, could be due to 35% missing data (HR = 0.946). Both models showed consistent results with appropriate confidence intervals when confounding variables were appropriately corrected for and missing data were imputed.

6.2 Comparison of Causal Methods

The IPTW and G-formula yielded slightly different effect sizes, which is expected. Odds ratios are mathematically further from the null than risk ratios when the outcome is common, as in this study where mortality exceeded 40%. Additionally, IPTW achieves confounding control through weighting observations, whereas the G-formula does so through outcome modelling. The convergence of both methods towards the same conclusion—that milnepan reduces mortality—strengthens the causal interpretation of our findings.

6.3 Strengths

There are several methodological advantages to the approach that we have taken for this particular research. We used multiple imputations for missing data across 20 different datasets, which is an appropriate approach for handling uncertainty regarding missing BMI and smoking data. We also applied two different causal inference approaches, and the fact that they provided consistent results is an added advantage for our conclusions. After using

IPTW, we also checked for covariate balance and found that all were less than 0.1. Also, we have a large sample, consisting of 8,192 patients, and hence our design had enough statistical power. A causal assumption is also explicitly specified through a directed acyclic graph, and then computationally verified.

6.4 Limitations

That said, there are some number of limitations to consider. Because this is an observational study, we cannot rule out unmeasured confounding-things like how sick patients were at the start of treatment are not available, and may have driven both treatment patterns and outcomes. The MAR assumption underlying multiple imputation is not one that we can check, and our estimates may be biased if data were missing in a non-random way. Even though balance looked good, the propensity score model can still be misspecified in ways we wouldn't know from balance checks. And finally, these results reflect just one data set, and might not generalize to other populations or clinical contexts.

7. Conclusion

This analysis gives us concrete evidence that milnepan treatment has a causal effect on reducing 7-day mortality for heffpox patients. The pooled odds ratio is 0.876 (95% CI: 0.798, 0.961) using inverse probability of treatment weighting, and 0.922 (95% CI: 0.877, 0.969) using the G-formula. The consistency of the results from two different causal inference approaches, together with good covariate balance and handling of missing data through multiple imputation, lends strength to our results. Under the assumption of no unmeasured confounding, milnepan has a real causal effect and can be considered as a possible treatment for heffpox, which could reduce mortality by 8-12%.

8. References

Course Materials:

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