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Seroprevalence and Infection Attack Rate of COVID-19 in Indian Cities

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Abstract

Objectives. Serological surveys were used to infer the infection attack rate in different populations. The sensitivity of the testing assay, Abbott, drops fast over time since infection which make the serological data difficult to interpret. In this work, we aim to solve this issue.

Methods. We collect longitudinal serological data of Abbott to construct a sensitive decay function. We use the reported COVID-10 deaths to infer the infections, and use the decay function to simulate the seroprevalence and match to the reported seroprevalence in 12 Indian cities.

Results. Our model simulated seroprevalence match the reported seroprevalence in most (but not all) of the 12 Indian cities we considered. We obtain reasonable infection attack rate and infection fatality rate for most of the 12 Indian cities.

Conclusions. Using both reported COVID-19 deaths data and serological survey data, we infer the infection attack rate and infection fatality rate with increased confidence.

Keywords: COVID-19; pandemic; seroprevalence; attack rate; mathematical modelling.

Introduction

The COVID-19 pandemic has caused tremendous impact globally. The situation in terms of reported cases and deaths were low in the year 2020 in India. It was once claimed

that the control in India was a success until a major second wave hit the country and causing large number of mortalities. But the reported deaths per capita is still relatively low compared to other Western countries. Velumani et al. (Velumani et al. 2021) studied seroprevalence in 12 Indian cities from July to December 2020. Their result showed two distinct patterns: a wave pattern, e.g., Mumbai and Pune, and a monotone increasing pattern, e.g. Delhi and others. These patterns were driven by the underlying COVID-19 pandemic in each city. However, the other key drivers are the use of multiple brands of antibody testing machines (or assays): in particular Abbott and non-Abbott, e.g., Roche. Abbott and Roche are widely used serological testing devices in the market. The method of Abbott SARS-CoV-2 IgG assay is acridinium-labelled anti-human IgG. Roche used ruthenium-labelled and biotin-conjugated N protein to perform the Anti-SARS-CoV total antibody assay. Velumani et al. (Velumani et al. 2021) highlighted that two machines were used in this study, without specifying which city used which one. Presumably, the Abbott was used in these wave-pattern cities while Roche was in others (we explain the reason later). The two machines have very different ‘decay rates’ as found in (Muecksch et al. 2021). With data from (Muecksch et al. 2021), Figure 1 shows a comparison of the sensitivity of Abbott vs Roche.

We observed that Abbott showed a very rapid drop in sensitivity over time for RT-PCR confirmed infections after RT-PCR confirmation (or onset of symptom for symptomatic cases after RT-PCR confirmed). While the sensitivity of Roche is stable. Moreover, as explained in the method (later section). The seroprevalence obtained with Roche would be much closer to the infection attack rate (IAR, defined as the cumulative proportion of the infected population), which shows a monotone increasing pattern. Since the antibody level of infected individual will gradually wane over time which causing the sensitivity of the testing drop (an infected person became seronegative), the seroprevalence will be lower than the attack rate (AR), and this is very evident with Abbott than Roche. We first estimate the sensitivity drop

function of Abbott based on publicly available data of Abbott. Then we collect reported COVID-19 deaths and serological studies from 12 Indian cities. To connect the two sources of data, we use the independently estimate sensitivity decay function of Abbott to reconstruct the IAR and the seroprevalence simultaneously in 12 Indian cities. We found that our approach works well in most cities where Abbott was likely and failed in a few cities where non-Abbott was likely used. Since we combine two sources of data, our inference on the IAR should be of higher confidence than using a single source of data.

Material and Methods

Data

COVID-19 deaths data were obtained from <https://api.covid19india.org>. Serological survey data for 12 Indian cities were obtained from (Velumani et al. 2021). A serological survey is usually carried out to investigate the prevalence of the size of an epidemic (population infected) in a given population by identifying the presence of specific antibodies used against the virus (Anand et al. 2021; George et al. 2021; Inbaraj, George, and Chandrasingh 2021; Murhekar et al. 2020; Murhekar, Bhatnagar, Selvaraju, et al. 2021; Murhekar, Bhatnagar, Thangaraj, et al. 2021; Velumani et al. 2021).

Method

Sensitivity decay of Abbott

To quantify the drop in Abbott, we collected data from several independent studies (Maine et al. 2020; Muecksch et al. 2021; Eberhardt et al. 2021; Kahre et al. 2021; Harris et al. 2021), pooled data together (see Figure 2a), and fitted a modified Gamma distribution function (which has a max value of 1, thus not a distribution function anymore) to the data to quantify the drop rate (see Figure 2b). Thus, $f(t) = \text{Gamma}(t, \alpha, \beta)$. Hence, we find the best estimate $\alpha = 0.165$, $\beta = 0.012$.

100 Following (He et al. 2021), we denote the daily COVID-19 seroprevalence as p
 101 (proportion of population showing seropositive / antibody positive at a given day), daily sero-
 102 conversion as p^+ (sero-negative individual turns sero-positive), daily sero-reversion as p^-
 103 (sero-positive individual turns sero-negative due to antibody waning), then we have the
 104 following equation:

$$105 \quad p[T] = p[T - 1] + p^+ [\text{in the last 24 hours}] - p^- [\text{in the last 24 hours}]$$

106 Assuming initially all these are zero, this can be written as

$$107 \quad p(T) = \sum_0^T p^+ - \sum_0^T p^- .$$

108 Denote $f(s)$ as the sensitivity decay function which is given in Figure 2b.

$$109 \quad p(T) = \int_0^T c(t)f(T - t)/Ndt$$

110 i.e., the convolution (here we rewrite the summation in the above equation into integral
 111 fashion) between $c(t)$ and $f(s)$, where $c(t)$ is the time series of daily new cases, N is the
 112 size of the population. But, typically $c(t)$ is not very reliable due to time-varying testing
 113 policy and testing effort and a significant amount of asymptomatic cases. Nonetheless, the
 114 daily COVID-19 deaths are available ((by law in many regions) and relatively reliable than
 115 reported cases. We denote r as the infection fatality ratio (IFR). Thus, the daily new cases
 116 are roughly given as $c(t) = d(t + 14)/r$. We assume a delay between deaths and case
 117 confirmation (ie, symptom onset for symptomatic cases) of 14 days. The seroprevalence on
 118 day T is given:

$$119 \quad p(T) = \int_0^T r^{-1}d(t + 14)f(T - t)/Ndt$$

120 We use the black curve in Figure 2b as $f(t)$ for Abbott. When $f(t) \equiv 1$, $p(T) = AR(T)$, the
 121 infection attack rate is:

$$IAR(T) = \int_0^T r^{-1} d(t + 14) / N dt.$$

Thus given $f(s)$ and death data, we can get $p(t)$ and $IAR(t)$ easily if r is known. However, r estimates vary in previous studies, across locations and rely on the efforts of COVID-19 cases and deaths reporting. We argue that we may estimate r by matching $p(t)$ to reported seroprevalences at different time points. If the matching is acceptable, then we may accept the reported seroprevalences (given that it was Abbott based), and we immediately obtain the IAR and r .

Results

Figure 3 and Table 1 shows the main results, where we applied our method to 12 large Indian cities. Since we do not know the infection fatality ratio (IFR), we align the reconstructed seroprevalence (blue dashed curve) and serological survey (black curve with circles) to be equal on Oct 15, 2020; this alignment will yield an IFR r , which is applied to the whole reconstructed seroprevalence and the IAR curve (red curve). The reconstructed and observed seroprevalence matched reasonably well for most cities, where Abbott is likely used for most of the period. This works fine for most cities except for a few cities, e.g., Ahmedabad. In Ahmedabad, the serological survey went up too fast than the increase in IAR. Thus, some mechanism unknown might occur (e.g. non-Abbott or inconsistency assay or inconsistency in death reports) which affected the serological survey or dramatic changing in COVID-19 death reporting process.

Table 1. Summary of estimates of infection attack rate and seroprevalence for 12 Indian cities.

In our method, we assumed a reported COVID-19 accurately reflect the attack rate subject to a constant IFR. This could fail if the death reporting effort dramatically changes or

the intrinsic IFR changes (likely due to the emergence of new variants). The reconstructed seroprevalence successfully matched with the reported serological survey in Mumbai, Delhi, Chennai, and Coimbatore. The reconstructed seroprevalence also successfully matched with the *trend* of serological surveys in Kolkata, Nagpur, and Visakhapatnam. The method failed in the rest, where a non-Abbott (or change of testing assay) was likely used. We do not have sufficient data to reconstruct the sensitivity decay curve of Roche. Once it is available, a similar approach can be made readily (we leave for further study). In Figures 1&2, we assume a peak sensitivity of 100% for Abbott, which could be too ideal (since the data collected was mainly symptomatic cases). For asymptomatic cases, the peak sensitivity could be as low as 80%. However, as long as the shape of the decay curve is kept, a factor (80%) on the whole decay curve can be translated into a factor in the IFR. The reciprocal of the IFR and the sensitivity decay rate appear in a product form in our IAR and seroprevalence equation. Therefore, it would not change our final estimate of IAR and seroprevalence when the factor is in IFR or in the decay rate.

Moreover, we formulated a simple method to reconstruct the IAR and estimate the seroprevalence of COVID-19 deaths in India. Our simple approach works well in most of 12 Indian cities where serological survey data are available. We achieved this without knowing which cities used Abbott (we know Abbott was used in Mumbai). In some cities where our method did not yield a reasonable fit were likely due to the use of a non-Abbott (e.g. Roche) or mixing of different testing assays. According to the available information, we observed that the serological testing equipment used in Mumbai is Abbott and Delhi used Mylab, German Altona Diagnostics, Roche, and ELISA. The devices use in Chennai include Abbott, Roche, and ELISA. The instruments used in Kolkata include Vitros ECi, Ortho Clinical Diagnostics, Roche, and ELISA. The device Bangalore used was the Elecsys Anti-SARS-CoV-2 assay. The equipment used in Ahmedabad was Zydus Diagnostics. The devices Pune

used include ELISA and Roche. The device used in Surat was the ICMR National Institute of Virology. The devices Nagpur used include Roche and ELISA. The devices used in Coimbatore include Siemens and Abbott. The devices used in Jaipur include ICMR and IgM /IgG Duo Test Kit (SD, Biosensor, Republic of Korea). Nevertheless, the device used in Visakhapatnam is unknown. An alternative explanation could be that the serological survey or death reporting is inconsistent in some cities where our approach fails. Our method provided a reasonable and likely more reliable estimate of IAR by March 2021 in 12 Indian cities since it used both serological data and reported COVID-19 deaths.

Discussion

Sero-epidemiological studies are crucial in understanding the current and future scenario of the COVID-19 epidemics. Based on some recent reports, the average seroprevalence rate of COVID-19 in India has been meagre, which indicates that a significant proportion of the overall population is still susceptible to COVID-19 infection (Murhekar, Bhatnagar, Selvaraju, et al. 2021). This further highlighted that the virus's transmission could likely continue in most cities in India until the herd immunity threshold is reached. The herd immunity can be achieved either by previous exposure to the virus or through vaccination (Britton, Ball, and Trapman 2020; Murhekar, Bhatnagar, Selvaraju, et al. 2021). However, the successful control depends on the individual susceptibility to infection, pre-existing immunity, and compliance of non-pharmaceutical interventions (NPIs) measures, which could most likely affect the required number of infected individuals for the herd immunity threshold to be reached at a time of point (Aguas et al. 2020; Lourenço et al. 2020; Simoneaux and Shafer 2020; Mendez-Brito, El Bcheraoui, and Pozo-Martin 2021).

The IFR estimates in Table 1 are low. On one hand, many serological studies found that a very large proportion of the population has been infected. On the other hand, the

reported deaths is low (may or may not be due to under reporting). These two factors led to a seemingly low IFR in India. Banaji (Banaji 2021) found a lower bound IFR in Mumbai at 0.1% with consideration of excess deaths (additional to reported COVID-deaths), which is not very far from our estimates here. Here we used reported COVID-19 deaths only, which could lead to a low IFR.

We estimated the seroprevalence rate, and reconstructed an IAR in 12 Indian cities and found a reasonable seropositivity rate to SARS-CoV-2 in most cities. We showed that the seroprevalence rate was lower than the reconstructed IAR, which is a monotone-increasing function due to antibody waning. Since we do not know the infection fatality ratio, we aligned the reconstructed seroprevalence and serological survey to be equal in the middle point of the study period, e.g., Oct 15, 2020, to yield an IFR, which was applied to both the reconstructed seroprevalence and the IAR curve. These results are in line with previous estimates, which highlighted the seroprevalence of COVID-19 deaths to be high in large cities; as well as with a limited number of serological surveys in more randomly selected populations (George et al. 2021; Velumani et al. 2021).

A significant amount of the people infected with SARS-CoV-2 do not show symptoms, yet they transmit the disease to other susceptible populations (Long et al. 2020; Oran and Topol 2021; Tu et al. 2020). It has been reported that asymptomatic cases of COVID-19 could reach from 5% up to about 80% of all infections (Inbaraj, George, and Chandrasingh 2021; Yanes-Lane et al. 2020), especially for young and healthy individuals. In slums settings like India, these estimates could reach up to more than 90% (George et al. 2021; Velumani et al. 2021). Therefore, it is imperative to identify an infected individual at the early stage of the infection and trace all possible contacts in order to break the transmission paths, and also to prevent more rapid spread of the pandemic. However, recognizing an asymptotically (and

pre-symptomatic) infected individual is practically difficult (and sometimes impossible) since they don't seek medical attention, which helps to identify their exposure to the disease (Gao et al. 2021; Inbaraj, George, and Chandrasingh 2021). As a result, relevant authorities, including the WHO, did not recommend absolute reliance on the morbidity and mortality cases identified through RT-PCR since it is very likely to have potential under-reporting issues (Inbaraj, George, and Chandrasingh 2021).

Furthermore, to tackle this issue, the concerned authorities suggested that more research should be done on population-based sero-epidemiological surveys to generate more data that would help design the most effective control strategies ('World Health Organization. Coordinated global research roadmap: 2019 novel coronavirus; March 2020. Geneva: World Health Organization,' 2020). In addition, sero-epidemiological studies can help estimate the percentage of the population still susceptible to SARS-CoV-2. It is also important to investigate how Anti-SARS-CoV could provide immunity to the virus (Inbaraj, George, and Chandrasingh 2021). Most of early serosurveys, including the nationwide serosurvey that considered 21 Indian states by the Indian Council of Medical Research (ICMR), estimated the seroprevalence of COVID-19 in India to be 0.73% between May-June 2020 (Inbaraj, George, and Chandrasingh 2021; Murhekar et al. 2020). Subsequently (i.e., around August-September 2020), most serosurveys estimated the seroprevalence of SARS-CoV-2 in India to be 7-10% (George et al. 2021; Inbaraj, George, and Chandrasingh 2021; Khan et al. 2020; Murhekar, Bhatnagar, Selvaraju, et al. 2021; Murhekar, Bhatnagar, Thangaraj, et al. 2021). In their recent serosurvey study, Murhekar et al. estimated the overall seroprevalence of SARS-CoV-2 in India to be more than 24% (Murhekar, Bhatnagar, Thangaraj, et al. 2021).

The epidemiological implications of SARS-CoV-2 seroprevalence are that it helps identify whether herd immunity has been reached (due to vaccination or previous exposure to

the virus). It also helps to identify more cases, especially asymptotically infected individuals who do not usually show COVID-19 symptoms, to design the most effective control strategies to mitigate the epidemics. High estimation of seroprevalence of COVID-19 would indicate that most of the population have been infected, and the herd immunity threshold will likely be reached (George et al. 2021; Kwok et al. 2021). Hence, the control efforts should be emphasized on recovering for the damages caused by this deadly virus, which includes suspension of control and intervention programmes, immunization and primary health care services for other infectious diseases; as well as rebuilding for better socio-economic development.

Our study has some limitations. We employed serological data that used multiple and mixing testing machines (Abbott and non-Abbott, e.g. Roche) to reconstruct an IAR and estimate the seroprevalence of COVID-19 in large cities in India. The true situation is much complicated than what we have considered. This could likely affect the actual estimation of seroprevalence in these cities. Further, we considered only 12 Indian cities; thus, interpretation for the overall scenarios has to be done with caution since new epidemiological features of SAR-CoV-2 is being discovered every day. We rely on the confidence on the COVID-19 death reporting.

Conclusion

The seroprevalence of COVID-19 in India did not usually alter by sex or age group. This paper reconstructed the attack rate and estimated the seroprevalence of COVID-19 deaths in 12 large Indian cities. We found a reasonable seropositivity rate to SARS-CoV-2 in most cities, where Abbott was likely used. We recommend that further sero-epidemiological studies and improved COVID mortality reporting couple with different existing control strategies can effectively help in suppressing the COVID-19 morbidity and mortality in India

and beyond. Although we did not generate new data, we provide a way to connect data from different sources (COVID-19 deaths and serological survey) which were treated separately in previous studies. Hence the overall confidence on these data is enhanced and potential inconsistency can be revealed.

Declarations

-Ethics approval and consent to participate

Not applicable.

-Consent for publication

Not applicable.

-Availability of data and materials

Serological data used in this work were taken from the following references, Maine et al, 2020, Muecksch et al 2020, Eberhardt et al 2020, Kahre et al, 2020 Harris et al, 2020. Velumani et al. 2021. The COVID-19 deaths data for Indian cities were obtained from <https://api.covid19india.org>.

-Competing interests

The authors declare that they have no competing interests.

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-Authors' contributions (This statement must exactly match on Editorial submission system and in the manuscript)

All authors conceived the study, carried out the analysis, wrote the draft, revised the manuscript critically, and approved it for publishing.

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None.

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Figures

Figure 1. A comparison of sensitivity of Abbott vs Roche over time. Data from (Muecksch et al. 2021). The sensitivity of Abbott drops faster over time.

Figure 2 Abbott sensitivity as a function of time after symptom onset for a group of PCR positive patients (panel a). We assume the date of symptom onset and the date of RT-PCR confirmation are close. We fit a model (see main text) to the observed data (panel b).

Figure 3. Simulated attack rate (red curve) and seroprevalence (blue dashed curve) over time compared with serological surveys (black curve with circles) in 12 Indian cities (Velumani et al. 2021). The attack rate (AR) is a monotone-increase function of time given its definition. The seroprevalence is lower than the AR due to sensitivity which is less than 100% and decays rapidly over time. Since we do not know the infection fatality ratio, we simply align the reconstructed seroprevalence and serological survey to be equal on Oct 15, 2020 to yield an IFR which is applied to both the reconstructed seroprevalence curve and the AR curve.

Figures

Figure 1

A comparison of sensitivity of Abbott vs Roche over time. Data from (Muecksch et al. 2021). The sensitivity of Abbott drops faster over time.

Figure 2

Abbott sensitivity as a function of time after symptom onset for a group of PCR positive patients (panel a). We assume the date of symptom onset and the date of RT-PCR confirmation are close. We fit a model (see main text) to the observed data (panel b).

Figure 3

Simulated attack rate (red curve) and seroprevalence (blue dashed curve) over time compared with serological surveys (black curve with circles) in 12 Indian cities (Velumani et al. 2021). The attack rate (AR) is a monotone-increase function of time given its definition. The seroprevalence is lower than the AR due to sensitivity which is less than 100% and decays rapidly over time. Since we do not know the infection fatality ratio, we simply align the reconstructed seroprevalence and serological survey to be equal on Oct 15, 2020 to yield an IFR which is applied to both the reconstructed seroprevalence curve and the AR curve.