Incidence of ESBL Producers amongst Gram-negative Bacilli Isolated from Intra-abdominal Infections across India (Based on SMART Study, 2007 Data)

BN Chaudhuri1, C Rodrigues2, V Balaji3, R Iyer4, U Sekar5, Chand Wattal6, DS Chitnis7, TN Dhole8, Sangeeta Joshi9

Abstract

Objectives: This study was conducted in 9 centers spread over India from January 1 to December 31, 2007 to monitor in vitro susceptibility of Gram-negative bacilli to Group I carbapenem, ertapenem and other antimicrobials in intra-abdominal infections and to identify early changes in susceptibility pattern of community or hospital acquired organisms, with a focus on ESBL producers.

Material and Methods: Gram-negative bacilli isolated from intra-abdominal samples of patients with documented intra-abdominal infections were processed for identification by conventional/automated methods and antimicrobial susceptibility by Micro-Scan (Siemens) mIC panel against 12 antimicrobials (3rd and 4th generation cephalosporins, Groups I and II carbapenems, amikacin, levofloxacin, amoxicillin-clavulanic acid and piperacillin-tazobactam).

Results: A total of 588 isolates were identified, of which 351 (60%) were E. coli and 114 (19%) were Klebsiella spp. 79% of E. coli and 70% of Klebsiella spp. were ESBL producers in general. 110 of E. coli and 35 of Klebsiella isolates were from community-acquired intra-abdominal infections. 80% of E. coli and 63% of Klebsiella isolates from community-acquired infections were ESBL producers, against 79% of E. coli and 73% of Klebsiella isolates from hospital-acquired infections. Amongst the ESBL-positive isolates of E. coli, 94% were susceptible in vitro to ertapenem, 96% to imipenem and 76% to piperacillin-tazobactam. For ESBL-positive isolates of Klebsiella spp., the corresponding figures were 80%, 94% and 59% respectively.

Conclusion: The study showed a high incidence of ESBL-producers amongst Enterobacteriaceae isolates from intra-abdominal infections in both community-acquired and hospital-acquired settings across India. Ertapenem was comparable with imipenem against ESBL-positive E. coli isolates, while imipenem was more effective than ertapenem against ESBL-positive Klebsiella isolates.

Rationale

The emergence of drug-resistant organisms in both hospitals and the community is a major concern. Surveillance studies have provided important information about changes in the spectrum of microbial pathogens and trends in the antimicrobial resistance patterns in nosocomial and community-acquired infections and continued monitoring of antimicrobial resistance patterns in hospitals is essential to guide effective empirical therapy.

One of the considerations of hospital formulary committees when considering new antibiotics is the potential for altering the bacterial flora epidemiology, especially resistance rates. Large multi-centre studies are useful for tracking trends over wide geographic areas and over long periods of time. However, most of these studies are not designed to identify early changes in susceptibility patterns or to determine the importance of specific risk factors in promoting resistance. In addition, such studies are not ideally suited to monitor the effect of introduction of new antibiotic because of variable use across the participating centers.

The purpose of SMART (Study for Monitoring Antimicrobial Resistance Trends) is two-fold:

1. To monitor the susceptibility of organisms causing intra-abdominal infections. Recent data indicate that the organisms causing these infections exhibit a high degree of susceptibility to drugs as ertapenem but long term surveillance is required to monitor for any important changes in susceptibility of common pathogens;

Aims and Objectives

SMART (Study for Monitoring Antimicrobial Resistance Trends), 2007 was conducted worldwide to

1. Monitor, globally and longitudinally, the in vitro susceptibility of Gram-negative bacilli to Group I carbapenem, ertapenem and other antimicrobials in intra-abdominal infections, focusing on isolates causing intra-abdominal infections in patients admitted in all sections of the hospital;

2. Identify early changes in susceptibility patterns based on bacterial population MICs, of community or hospital acquired organisms, with a focus on ESBL producers.

The present publication focuses on the observations of SMART in India where the study was conducted in 9 centers spread all over the country from January 1 to December 31, 2007.
whether the isolate was collected within 48 hrs or after 48 hrs of hospitalization (to differentiate between community-acquired and nosocomial pathogens).

The isolates were processed for identification by conventional/automated methods and antimicrobial susceptibility tests using pre-prepared customized microdilution plates (micro-Scan, Dade, now Siemens) containing 12 antimicrobials (vide Table 1): 3rd and 4th generation cephalosporins, Groups I and II carbapenems, amikacin, levofloxacin, amoxicillin-clavulanic acid and piperacillin-tazobactam. These plates were manufactured by Dade Microscan specifically for MRL and were not available commercially. Use of these plates by participating centers for non-study purposes or to guide patient management was not allowed. A standard inoculum as specified in the methods for mIC testing of CLSI (formerly NCCLS), 2007 guidelines, was used. The susceptibility tests were performed following the manufacturers procedure outline.

Quality Control: Quality Control (QC) was performed with each batch test. Testing procedures were validated using the following 3 reference strains recommended by CLSI:

1. **Pseudomonas aeruginosa** ATCC 27853,
2. **Escherichia coli** ATCC 25922,
3. **Klebsiella pneumoniae** ATCC 700603 (ESBL + control strain)

QC results were duly recorded on QC worksheets provided with the study materials.

If the QC for any antibiotic was out of range, the QC was repeated and documented on another QC worksheet with the date the QC was repeated. If the QC was out of range for one of the ATCC strains, then only that QC was repeated.

The results of the QC testing were reviewed by MRL before data were entered into the database; data for antibiotics with QC MICs outside the range cited in the CLSI document were not included in the database.

**Statistical Analysis:** The statistical significance of the data was determined using Fisher’s Exact Test.
Results

A total of 588 isolates were identified in all the centres put together. 55% of these isolates were from male patients and 45% from female patients. The distribution of the isolates according to ward type is shown in Table 2, Fig. 2.

The different sample types/body sites from where the isolates were obtained are shown in Table 3, Fig. 3.

Table 2: Ward type: Location of Isolates

<table>
<thead>
<tr>
<th>Ward type</th>
<th>No. of Isolates</th>
<th>% of Isolates</th>
</tr>
</thead>
<tbody>
<tr>
<td>Surgical ICU</td>
<td>344</td>
<td>59</td>
</tr>
<tr>
<td>Medical ICU</td>
<td>20</td>
<td>03</td>
</tr>
<tr>
<td>Surgery General</td>
<td>68</td>
<td>12</td>
</tr>
<tr>
<td>Medicine General</td>
<td>138</td>
<td>23</td>
</tr>
<tr>
<td>Pediatric Ward</td>
<td>14</td>
<td>02</td>
</tr>
<tr>
<td>Others</td>
<td>4</td>
<td>01</td>
</tr>
</tbody>
</table>

Table 3: Sample Types

<table>
<thead>
<tr>
<th>Sample type/ Site</th>
<th>No. of Isolates</th>
<th>% of Isolates</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gastro-intestinal</td>
<td>228</td>
<td>41</td>
</tr>
<tr>
<td>Small colon</td>
<td>68</td>
<td>11</td>
</tr>
<tr>
<td>Pancreas</td>
<td>59</td>
<td>10</td>
</tr>
<tr>
<td>Liver</td>
<td>57</td>
<td>09</td>
</tr>
<tr>
<td>Body fluids</td>
<td>57</td>
<td>09</td>
</tr>
<tr>
<td>Gall bladder</td>
<td>17</td>
<td>03</td>
</tr>
<tr>
<td>Others</td>
<td>102</td>
<td>17</td>
</tr>
</tbody>
</table>

Table 4: Antibiogram of Isolates to the Panel of Drugs Tested

<table>
<thead>
<tr>
<th>Isolates</th>
<th>N</th>
<th>A/S (%S)</th>
<th>P/T (%S)</th>
<th>Ak (%S)</th>
<th>Cp (%S)</th>
<th>Lvx (%S)</th>
<th>Cfx (%S)</th>
<th>3'CPs (%S)</th>
<th>Cpe (%S)</th>
<th>Etp (%S)</th>
<th>Imp (%S)</th>
</tr>
</thead>
<tbody>
<tr>
<td>E. coli (non-ESBL)</td>
<td>72</td>
<td>60</td>
<td>97</td>
<td>99</td>
<td>61</td>
<td>63</td>
<td>&gt;90</td>
<td>&gt;90</td>
<td>100</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>E. coli (ESBL)</td>
<td>279</td>
<td>08</td>
<td>76</td>
<td>85</td>
<td>05</td>
<td>11</td>
<td>63</td>
<td>0</td>
<td>&lt;10</td>
<td>95</td>
<td>97</td>
</tr>
<tr>
<td>K. pneumonia (non-ESBL)</td>
<td>34</td>
<td>76</td>
<td>97</td>
<td>97</td>
<td>91</td>
<td>94</td>
<td>76</td>
<td>&gt;90</td>
<td>&gt;90</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>K. pneumonia (ESBL)</td>
<td>80</td>
<td>00</td>
<td>59</td>
<td>74</td>
<td>12</td>
<td>25</td>
<td>65</td>
<td>0</td>
<td>&lt;10</td>
<td>80</td>
<td>94</td>
</tr>
<tr>
<td>K. oxytoca (all ESBL)</td>
<td>15</td>
<td>07</td>
<td>87</td>
<td>87</td>
<td>13</td>
<td>27</td>
<td>60</td>
<td>0</td>
<td>&lt;10</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>Klebsiella sp.</td>
<td>1</td>
<td>00</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>Enterobacter cloacae</td>
<td>22</td>
<td>09</td>
<td>93</td>
<td>87</td>
<td>23</td>
<td>27</td>
<td>32</td>
<td>14</td>
<td>32</td>
<td>91</td>
<td>100</td>
</tr>
<tr>
<td>Enterobacter aerogenes</td>
<td>15</td>
<td>33</td>
<td>64</td>
<td>64</td>
<td>40</td>
<td>53</td>
<td>73</td>
<td>46</td>
<td>60</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>Enterobacter sp.</td>
<td>9</td>
<td>22</td>
<td>78</td>
<td>89</td>
<td>56</td>
<td>56</td>
<td>33</td>
<td>44</td>
<td>44</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>Citrobacter sp.</td>
<td>16</td>
<td>17</td>
<td>64</td>
<td>17</td>
<td>15</td>
<td>31</td>
<td>31</td>
<td>17</td>
<td>17</td>
<td>95</td>
<td>100</td>
</tr>
<tr>
<td>Edwardsiella sp.</td>
<td>1</td>
<td>00</td>
<td>100</td>
<td>00</td>
<td>00</td>
<td>00</td>
<td>100</td>
<td>00</td>
<td>00</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>Serratia marcescens</td>
<td>1</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>NA</td>
<td>100</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>S. Typhi</td>
<td>2</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>NA</td>
<td>100</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>Salmonella sp.</td>
<td>2</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>NA</td>
<td>100</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>P. mirabilis (ESBL: 33%)</td>
<td>15</td>
<td>87</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>40</td>
<td>67</td>
<td>93</td>
<td>67</td>
<td>67</td>
<td>100</td>
</tr>
<tr>
<td>P. vulgaris</td>
<td>11</td>
<td>64</td>
<td>100</td>
<td>100</td>
<td>45</td>
<td>64</td>
<td>100</td>
<td>55</td>
<td>55</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>Proteus sp.</td>
<td>1</td>
<td>00</td>
<td>100</td>
<td>100</td>
<td>00</td>
<td>00</td>
<td>100</td>
<td>00</td>
<td>00</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>M. morganii</td>
<td>6</td>
<td>33</td>
<td>83</td>
<td>100</td>
<td>67</td>
<td>67</td>
<td>83</td>
<td>67</td>
<td>83</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>Pseudomonas aeruginosa</td>
<td>49</td>
<td>NA</td>
<td>69 (IS)</td>
<td>57</td>
<td>22</td>
<td>27</td>
<td>NA</td>
<td>37</td>
<td>37</td>
<td>NA</td>
<td>57</td>
</tr>
<tr>
<td>Pseudomonas fluorescens</td>
<td>1</td>
<td>NA</td>
<td>00</td>
<td>100 (IS)</td>
<td>00</td>
<td>00</td>
<td>NA</td>
<td>00</td>
<td>00</td>
<td>00</td>
<td>NA</td>
</tr>
<tr>
<td>Pseudo. sp.</td>
<td>3</td>
<td>NA</td>
<td>100</td>
<td>100</td>
<td>00</td>
<td>100</td>
<td>NA</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>B. cepacia</td>
<td>3</td>
<td>NA</td>
<td>67</td>
<td>00</td>
<td>33</td>
<td>33</td>
<td>NA</td>
<td>33</td>
<td>33</td>
<td>NA</td>
<td>67</td>
</tr>
<tr>
<td>S. maltophilia</td>
<td>2</td>
<td>NA</td>
<td>00</td>
<td>00</td>
<td>00</td>
<td>100</td>
<td>NA</td>
<td>00</td>
<td>00</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>A. baumannii</td>
<td>27</td>
<td>19</td>
<td>11</td>
<td>22</td>
<td>07</td>
<td>27</td>
<td>NA</td>
<td>04</td>
<td>04</td>
<td>NA</td>
<td>26</td>
</tr>
<tr>
<td>A. Iwoffi</td>
<td>3</td>
<td>00</td>
<td>00</td>
<td>00</td>
<td>00</td>
<td>33</td>
<td>NA</td>
<td>00</td>
<td>00</td>
<td>NA</td>
<td>00</td>
</tr>
<tr>
<td>Acinetob. sp.</td>
<td>5</td>
<td>20</td>
<td>20</td>
<td>20</td>
<td>20</td>
<td>20</td>
<td>20</td>
<td>NA</td>
<td>20</td>
<td>20</td>
<td>NA</td>
</tr>
</tbody>
</table>

79% of E. coli and 70% of Klebsiella pneumoniae isolates were ESBL producers in general. 100% of the Klebsiella oxytoca isolates and 33% of the Proteus mirabilis isolates were also ESBL producers.

No ESBL-producing Salmonella isolates were identified in this study.

The fact that not all the ESBL producing isolates were susceptible to cefoxitin may imply that some of these strains may be solely Amp C beta-lactamase producers or concomitant Amp C beta-lactamase producers along with ESBLs. However, screening of the isolates for Amp C beta-lactamase production was not done in this study.
The list of different isolates obtained and the antibiogram of the 588 isolates to the 12 antibiotics tested in the Study are shown in Table 4.

Of the 588 isolates identified, 351 (60%) were E. coli; 130 (22%) Klebsiella spp. (K. pneumoniae: 114, K. oxytoca: 15, other sp.:1); 56 (9.5%) Enterobacter spp. (E. cloacae: 22, E. aerogenes: 15, other spp.:9); 53 (9%) Pseudomonas spp. (P. aeruginosa: 49, other spp.:4); 35 (6%) Acinetobacter spp. (A. baumannii: 27, other spp.:8); 33 (5.6%) Protea (P. mirabilis: 15, P. vulgaris: 11, M. morganii: 6, other spp.:1); 16 (2.7%) Citrobacter spp. (C. freundii: 13, other spp.:3). Vide Table 4.

Out of the drugs tested, except for carbapenems, all the others showed a statistically significant drop (p<0.0001) in sensitivity for ESBL variants as compared to the non-ESBL strains. Only carbapenems, maintained their efficacy even in the presence of ESBLs and their sensitivity patterns did not show any significant difference between the two types. In comparison, drugs like amikacin and piperacillin-tazobactam showed statistically significant (p<0.005) drop in the sensitivities for ESBL variants vis-à-vis non-ESBL strains.

All the non-ESBL strains of E. coli were susceptible to Group I carbapenem, ertapenem and Group II carbapenem, imipenem (100% susceptibility to both) and their susceptibilities to amikacin and piperacillin-tazobactam were 99% and 97% respectively.

In case of ESBL-positive E. coli isolates, high susceptibilities to imipenem and ertapenem were noted (97% and 95% respectively), whereas the susceptibility to non-carbapenem antibiotics namely amikacin and piperacillin-tazobactam were reduced (85% and 76% respectively) Although this differences was not significant for the carbapenems (p>0.05).

A similar pattern was observed in case of K. pneumoniae strains where only carbapenems maintained their efficacy in the presence of ESBLs and their sensitivity patterns did not show marked difference between ESBL and non-ESBL variants. In case of non-ESBL K. pneumoniae strains, the susceptibilities towards carbapenems, amikacin and piperacillin-tazobactam were 100%, 97% and 97% respectively. For ESBL-positive K. pneumoniae strains, there was a slight difference in their susceptibilities towards imipenem (94%, p<0.05), but more pronounced with ertapenem (80%, p<0.005), while their susceptibilities towards amikacin and piperacillin-tazobactam were 74% and 59% respectively, much less than the non-ESBL strains.

Amongst the non-fermenters, 43% of P. aeruginosa and 33% of the B. cepacia isolates were imipenem resistant. On the other hand, as high as 74% of Acinetobacter baumannii isolates and 80-100% of other Acinetobacter isolates were resistant to imipenem. However, screening of the carbapenem resistant non-fermenters for metallo-beta lactamase (MBL) production and susceptibility of those isolates to aztreonam were not included in this study.

The important features observed in isolates obtained from patients within and after 48 hours of hospitalization, so as to determine the differences between “community-acquired” and hospital-acquired isolates, are shown in Table 5.

110 E. coli and 35 Klebsiella pneumoniae isolates were from “community-acquired” intra-abdominal infections. 80% of E. coli and 63% of Klebsiella pneumoniae isolates from “community-acquired” infections were ESBL producers against 79% of E. coli and 73% of Klebsiella pneumoniae isolates from hospital-acquired infections.

There were slight differences in susceptibility patterns of “community-acquired” and hospital-acquired ESBL-positive isolates of E. coli and K. pneumoniae (vide Table 5); however,
none achieved statistical significance (p>0.05 for all pairs). No difference in ertapenem or imipenem sensitivity was observed between “community-acquired” and hospital-acquired ESBL-positive isolates of *Klebsiella oxytoca* and *Proteus mirabilis* (vide Table 5).

No significant differences (p>0.05) were observed in the imipenem sensitivity for “community-acquired” and hospital-acquired isolates of *Pseudomonas* and *Acinetobacter* spp.

### Discussion and Conclusion

The study showed an alarming incidence of ESBL-producers amongst *Enterobacteriaceae* isolates from intra-abdominal infections in both “community-acquired” and hospital-acquired settings across India, especially in case of *E. coli* and *K. pneumoniae* isolates.

However, the observation of high prevalence of ESBL producers and carbapenem resistant non-fermenters in the “community” could at least partially be attributed to the definition used, i.e., infection within 48 hours and after 48 hours of hospitalization in the centers. Given the nature of the hospitals engaged as centers for the study in India, being predominantly tertiary care set-ups admitting patients already treated in some other institutions, rather than straight from the community. Hence the data may not be representative of the true community scenario.

Another limitation is that all contributing centers are urban centers located in major cities of India. Hence, the data is not representative of rural settings/ small towns

Ertapenem was comparable with imipenem against ESBL-positive *E. coli* isolates, while imipenem was more effective than ertapenem against ESBL-positive *Klebsiella pneumoniae* isolates. No difference in Group I and Group II carbapenem susceptibility was, however, observed in case of ESBL-positive *K. oxytoca* and *P. mirabilis* isolates in all settings.

A significant observation in the study was that no ESBL-producing *Salmonella* isolates were identified. In fact, all the *Salmonella* isolates were susceptible to all the antibiotics included in this study.

The study also identified a high incidence of carbapenem resistance in the case of the non-fermenting Gram-negative isolates, especially, *P. aeruginosa* and *Acinetobacter* spp.

The study did not screen isolates for Amp C beta lactamase and MBL production. These tests may be included in the future studies to get a better idea of the incidence of such strains in intra-abdominal infections.

The high incidence of ESBL producing *Enterobacteriaceae* as well as carbapenem resistant non-fermenters in the community indicates a grave situation that needs to be tackled urgently by:

- Complete eradication of infectious agents before affected patients are discharged.
- Patient contacts and hospital personnel to properly decontaminate their hands with disinfectants (or simply with soap and water) prior to leaving hospital premises.
- Hospitals to treat and decontaminate its waste, effluents, and sewage before disposal or before releasing those in public sewerage.
- The high incidence of nosocomial infection with multi-drug resistant bugs should be prevented and controlled by:
  - Continuous surveillance of hospital, especially ICUs, OT’s and wards housing high-risk patients.
  - Disinfection of medical devices and hospital environment.
  - Maintenance of strict asepsis during invasive procedures.
  - ‘Standard Precautions’ in wards and laboratories.
  - Proper disposal of hospital waste.
  - Patient isolation-/segregation or barrier nursing to prevent transmission of infection.
  - Hand hygiene with chlorhexidine or alcohol - based disinfectants before and between patient contacts: this is the most important measure.
  - Policies for antibiotic stewardship

### Note

a. The results of this study were presented in a poster session (poster no. GN 103) at the 7th International symposium on Antimicrobial agents and Resistance (ISAAR) held in Bangkok, Thailand on March 18-20, 2009

b. There was a publication in Antimicrobial agents and chemotherapy, Aug 2009, p 3280-3284 titled ‘Emergence of High Levels of Extended-Spectrum- β-Lactamase-Producing Gram-Negative Bacilli in the Asia-Pacific Region: Data from the Study for Monitoring Antimicrobial Resistance Trends (SMART) Program, 2007’

### Acknowledgement

This study was sponsored by Merck & Co., Inc. USA. We thank Dr. Mahua Ganguly and Dr. Ankur Gupta of MSD, India for their scientific and editorial support.

### References


THE AWARD OF FELLOWSHIP OF INDIAN COLLEGE OF PHYSICIANS

Nominations are invited for the award of Fellowship of Indian College of Physicians

Full format is available on API and JAPI websites: www.apiindia.org / www.japi.org

Last date to receive nomination for FICP : 31st May 2011

Dr. Milindy Y. Nadkar
Hon. General Secretary

Dr. B. R. Bansode
Joint Secretary

DR. VITHALRAO NADGOUDA BEST ALL INDIA ANNUAL THESIS AWARD

The Association of Physicians of India
Indian College of Physicians

DR. VITHALRAO NADGOUDA BEST ALL INDIA ANNUAL THESIS AWARD

1. The award is open to the physicians from various medical institutions / hospitals from India within one year of passing the MD / DNB examination in Medicine / General Medicine / Internal Medicine as on the last date for submission of the application for the above award is 31st May, 2011.

2. There shall be two awards: the first award shall comprise of Rs. 15,000/- along with a certificate and the second award shall comprise of Rs. 10,000/- along with a certificate.

Full format is available on API and JAPI websites: www.apiindia.org / www.japi.org

Dr. Milindy Y. Nadkar
Hon. General Secretary

Dr. B. R. Bansode
Joint Secretary

DOCTOR 2010 - Medical Software

COMPATIBLE WITH WINDOWS 7, VISTA, XP, DESKTOP, LAPTOP AND NETBOOK

CLINICAL: Case sheets, speciality sheets, Inpatient, ICU, Lab, PDR, Auto Casesummary, Certificates, letters, USS, X-ray, Pathology, Endoscopy, Echo, Proc. reports, very little typing needed. Prescription Autodose, Allergy, disease-contraindication, interaction alert, Fonts option (Hindi Tamil etc) Overdose treatment, Ther. level, dose in organ failures Store Recall at a single click.


Store/Link photos, X-ray, ECG, Videos; Change Header/Footer; Diet advisor-autocalory calculator


Address : MEDISOFT, Achutha Warrier Lane, Cochin-682035.
E-mail : medisoftindia@gmail.com

http://www.medisoftindia.com

Ph.: 09847294414 © IPR. All Rights Reserved