Effect of methanol leaf extract of anogeissus leiocarpus on gentamicin induced biochemical derangement in rats

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In traditional medicine, different parts of Anogeissus leiocarpus are highly valued for the treatment of array of human diseases including cardiotonic, diuretic, hepatoprotective, antimicrobial and anti-inflammatory activity. In the present study, single oral dose (100-400 mg/kg/day for 7 days) of methanol leaf extract of A. leiocarpus leaves were studied for its hepatoprotective and nephroprotective effect on albino rats. In the study of gentamicin induced abnormalities in metabolic biochemical parameters in albino rats, thirty six wistar albino rats were evenly divided into six groups of six animals each. The treated groups received gentamicin and doses of (100, 200 and 400 mg/kg) body weight per day of leaf extract respectively and were compared with the control group. On the 8th day, the blood samples of all the groups were taken for biochemical examination on different parameters that is, serum levels of urea, protein, creatinine, aspartate transaminase (AST), alanine transaminase (ALT), alkaline phosphatase, total bilirubin and direct bilirubin, as well as quantitative ionic estimation of some ions like sodium, potassium, chloride and bicarbonate. In the study of gentamicin induced abnormalities in rats treated with extract (100-400 mg/kg) significantly (p<0.05) decreased the levels of aspartate transaminase (AST) from 182.72±12.43(U/L) to 94.41±3.25 (U/L) , alanine transaminase (ALT) from 68.03±3.51 (U/L) to 35.14±8.88 (U/L), alkaline phosphatase (ALP) from 278.12±4.67 (U/L) to 245.39±12.02 (U/L), serum creatinine from 115.77±2.75(mmol/L) to 62.77±5.23 (mmol/L), acid phosphatase from 336.00±33.27 (U/L) to 240.62±34.28 (U/L) and serum urea from 8.39±0.95 (mmol/L) to 2.94±0.32 (mmol/L) when group 2 is compared with group 6 however, the plant extract reversed the effect of gentamicin in potassium ion from 12.64±0.68 (mmol/L) to 13.32 ± 0.86(mmol/L), chloride ion from 80.09+1.99(mmol/L) to 87.13±1.95 (mmol/L) and bicarbonate ion from 25.00±0.49 (mmol/L) to 25.21±0.37 (mmol/L) when group 2 was compared with group 6. The results suggest that Anogeissus leiocarpus is a potent hepatoprotective and nephroprotective agent which is capable of normalizing biochemical abnormalities associated with liver and kidney damage.

Key words: Anogeissus leiocarpus, gentamicin, aspartate transaminase (AST), alanine transaminase (ALT) and alkaline phosphatase.

INTRODUCTION

Herbal medicine or phytomedicine is recognized as the most common form of alternative medicine (Ogbonnia et al., 2011). The World Health Organization (WHO,1991) estimates that 80% of the world’s population relies on these “alternative” plant-based medicines as their primary medical intervention especially in the developed countries where modern medicines are predominantly used (Rickert et al., 1999;Ogbonnia et al., 2008). Over the years, the use of herbs in the treatment of illness has been very successful and its historic usage has been useful in drug discovery development. Medicinal plants have formed the basis of health care throughout the world since the earliest days of humanity and have remained relevant in both developing and the developed nations of the world for various chemotherapeutic purposes. From a health regulatory stand point, the
The greatest and most valued attribute of medicinal plants however appears to lie in their protective and multipurpose remedy (Abdul, 2000). Herbal prescriptions and natural remedies are commonly employed in developing countries for the treatment of various diseases, this practice being an alternative way to compensate for perceived deficiencies in orthodox pharmacotherapy (Sofowora, 1989; Zhu et al., 2002). The popularity and availability of the traditional remedies have generated concerns regarding the safety, efficacy and responsibility of practitioners using traditional remedies (Chan, 1995). Herbal remedies are considered safer and less damaging to the human body than synthetic drugs (Alam et al., 2011).

The plant, *Anogeissus leiocarpus* is of the family combataceae. It grows widely in Nigeria and it is used as folk medicine in Central and West Africa, (Burkill, 1985). The medicinal uses of *A. leiocarpus* can never be over-emphasized in Nigeria and other places. It is well known for its haemostatic and wound healing properties. The plant have considerable attention for their medicinal properties and find application in folk medicine, as well as in contemporary medicine (Adigun et al., 2000). The leaves and bark of *A. leiocarpus* are bitter tonic, astringent, analgesic and carminative. Ethnopharmacologically, it is used for the treatment of diarrhea, vomiting, earache, burns, abscesses, diabetic ulcers, insect bites and fever (Burkill, 1985; Ibrahim et al., 1997; Onyeyili, 2000), the juice from the fresh leaves are used to treat pneumonia, cough, asthma, gonorrhoea, headache, convulsion and general debility (Mann et al., 2010). Leaves powder is used externally as a decoction in the eastern part of Nigeria for the treatment of skin diseases and itch of psoriasis.

In South-Eastern Nigeria, the herb is used as an antimicrobial agent against bacteria infections (Dweek, 1996). It is largely used in folk medicines for treatment of schistosomiasis and leprosy (Burkill, 1995). In traditional medicine, the leaves of the plant also have been used for antifungal (Akande and Hayashi, 1998), anti-caries and anti-periopathic (Akande and Hayashi, 1998).

The plant proved to be useful in vitiated conditions, epilepsy, piles (Atawodi et al., 2003), haemorrhoids, cuts and wounds, discolorations of the skin, boils, eczema, scald, corn (Agaie et al., 2007). *A. leiocarpus* can reduce fever and does provide anti-inflammatory and muscle relaxant effect (Adeleye et al., 2003).

The medicinal uses of *A. leiocarpus* can never be over-emphasized in Nigeria and other places. It is well known for its haemostatic and wound healing properties. The plant have considerable attention for their medicinal properties and find application in folk medicine, as well as in contemporary medicine (Adigun et al., 2000).

The study provide detail information of the plant, exploring its uses and phytochemical studies on *A. leiocarpus* and also pinpoint unexplored potentials of the plant.

**MATERIALS AND METHODS**

**Plant collection and identification**

Fresh leaves of *Anogeissus leiocarpus* were collected along Numan-Yola road Adamawa state, Nigeria. The plant was taken to the plant science (Botany) department and was taxonomically identified and authenticated by a taxonomist in the department.

**Preparation of extract**

The leaves of the plant were shade – dried and crushed manually in mortar. The dried leaves were reduced to coarse powder and 250 g of the powdered sample was soaked in 1000 ml of methanol (70% v/v) for 48 h with occasional shaking. The resulting mixture was filtered using whatman No. 1 filter paper and the resulting methanol extract was evaporated to dryness using a rotary evaporator.

**Reagents:** All the reagents and solvents used were of analytical grade.

**Animals**

The grown albino rats with an average weight of 100–20 g were obtained from the animal breeding department of the College of Medicine University of Jos. The albino rats were kept under light in well ventilated cages with comfortable accessibility to adequate water and feed ad-libitum to enhance rapid growth and development.

**Determination of body weight**

The body weights of each rat before and after the experimental period were obtained using a weighing balance (Ohaus, UK). The average weight of each group was taken and recorded carefully.

**Experimental design**

After acclimatization, 36 animals were randomly divided to six groups of six animals each (6 rats/each group). Injections of gentamicin (80 mg/kg) was done intraperitoneally to induced toxicity and the plant extracts of different doses were administered orally for a period of seven days as given below:

**Animal Sacrifice**

Animals were sacrificed under chloroform anaesthesia and the blood were collected through cardiac puncture and allowed to clot and centrifuged to obtain the serum for assay of biochemical parameters.
### Table 1. Experimental design.

<table>
<thead>
<tr>
<th>Group</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1 normal control</td>
<td>1 ml of distilled water daily</td>
</tr>
<tr>
<td>Group 2 negative control</td>
<td>80 mg/kg of gentamicin</td>
</tr>
<tr>
<td>Group 3 positive control</td>
<td>80 mg/kg gentamicin + Silymarin</td>
</tr>
<tr>
<td>Group 4 treated with extract</td>
<td>80 mg/kg gentamicin + 100 mg/kg <em>Anogeissus leiocarpus</em></td>
</tr>
<tr>
<td>Group 5 treated with extract</td>
<td>80 mg/kg gentamicin + 200 mg/kg/day <em>Anogeissus leiocarpus</em></td>
</tr>
<tr>
<td>Group 6 treated with extract</td>
<td>80 mg/kg gentamicin + 400 mg/kg/day <em>Anogeissus leiocarpus</em></td>
</tr>
</tbody>
</table>

### Biochemical studies

Portion of the blood was dispensed into plain bottles, allowed to clot and centrifuged at 3500 rpm at 10 min and the clear sera aspirated off for biochemical evaluation. Alanine Aminotransferase (ALT) and Aspartate Aminotransferase (AST) were determined by monitoring the concentration of pyruvate hydrazone from with 2,4, dinitrophenyl hydrazone and oxaloacetate hydrazone formed with 2,4-dinitrophenyl hydrazone respectively using Randox Diagnostic kits (Randox laboratories Ltd., Antrim, UK) (Reitman and Frankel, 1957). Alkaline phosphatase was determined using EC. 3.13.1 commercial kit from Randox Laboratories (Grant et al., 1987). Acid phosphatase was determined using Randox kit (Kornberg, 1955; Lohr and Waller, 1974). Acid phosphatase was determined using Randox kit (Kornberg, 1955; Lohr and Waller, 1974), Bilirubin activity was determined using colorimetric method described by Jendrassik and Grof; (1938). Total protein was assayed using described by Tietz, (1995). Albumin levels were determined by dye-bonding reaction with bromocresol green using colorimetric estimation (Grant, et al., 1987). Serum Creatinine and urea were assayed using standard procedures as described by Varley and Alan, 1984, Electrolyte estimation were done by the method described by (Henry et al., 1995).

### Statistical analysis

Results were expressed as mean+ SEM. Differences between the groups were determined by performing one way analysis of variance (ANOVA) and significance difference (LSD) to compare the mean at 0.05 by using the statistical package for social sciences (SPSS) software for windows (version 16.0).

### RESULTS

**Effects of *Anogeissus leiocarpus* on body weight of gentamicin induced rats**

Table 2 shows the effect of single oral dose of 100-400 mg/kg/day of *A. leiocarpus* on the body weight on gentamicin (80 mg/kg) induced rats for 7 days. As indicated in the (Table 1), i.p injection with gentamicin induced significant (p<0.05) weight loss of whole body in the treated rats. However, body weight was significantly (p<0.05) increased by *A. leiocarpus* treatment in a dose related manner.

**Effect of graded doses of *Anogeissus leiocarpus* and gentamicin on liver marker enzymes**

Gentamicin elevated the level of biochemical parameters; AST, ALT, ALP, TB, DB, TP, ALB, and APH in the serum as compared to that of the control group (Table 3). Treatment of *A. leiocarpus* significantly (p<0.05) reversed the serum level of AST, ALT, ALP, TB, DB, TP, ALB and APH close to normal levels when compared with the gentamicin treated group 2 (Table 3).

**Effect of graded oral doses of *Anogeissus leiocarpus* on serum creatinine, total protein, serum urea and plasma electrolyte concentrations in gentamicin induced rats**

Table 4 indicated that the serum creatinine, and serum urea levels were increased significantly (p < 0.05) in rats treated with only gentamicin, whereas treatment with the *A. leiocarpus* 100-400 mg/kg significantly lowered their levels in the treated animals in a dose dependent manner.

### Table 2. Effect of graded oral doses of *A. leiocarpus* on average body weight in gentamicin induced rats.

<table>
<thead>
<tr>
<th>Group</th>
<th>Day 0</th>
<th>Day 7</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control group</td>
<td>110.21±2.34</td>
<td>117.22±0.90</td>
</tr>
<tr>
<td>Gentamicin (80 mg)</td>
<td>105.98±3.06</td>
<td>94.15±2.35</td>
</tr>
<tr>
<td>Gentamicin+Silymarin</td>
<td>106.58±3.50</td>
<td>109.53±2.40</td>
</tr>
<tr>
<td>Gentamicin+100 mg</td>
<td>105.19±2.97</td>
<td>100.13±1.51</td>
</tr>
<tr>
<td>Gentamicin+200 mg</td>
<td>106.92±3.71</td>
<td>105.47±3.33</td>
</tr>
<tr>
<td>Gentamicin+400 mg</td>
<td>107.91±3.05</td>
<td>111.19±2.14</td>
</tr>
</tbody>
</table>

Values are expressed as mean ± S.E.M. (n = 6). P values were calculated using one way ANOVA (Analysis of Variance). Mean with different superscripts (a, b) differ significantly, p < 0.05.
3.51 9.11 35.14+
3.82 41.86+
1.04
8.88

al. (2009), the terpenoidal fractions from ability and strong reducing ability. According to Mann et
attributed to flavonoids, phenolic acid and tannins. This activity can be or post-treatment with drugs. This activity can be (Mann et al., 2009).

could be a potential source of chemotherapeutic age nts that the methanol extract of that the methanol extract of
agreement with that of Atawodi et al. (2011) who reported biochemical abnormalities associated with liver and
The results from our findings suggest that Anogeissus leiocarpus is a potent hepatoprotective and nephroprotective agent which is capable of normalizing biochemical abnormalities associated with liver and kidney damage (Tables 3 and 4), this findings is in agreement with that of Atawodi et al. (2011) who reported that the methanol extract of A. leiocarpus have strong in vivo antioxidant hepatoprotective and ameliorative activities on hepatocellular injury following pre-treatment or post-treatment with drugs. This activity can be attributed to flavonoids, phenolic acid and tannins. Victor and Grace, 2013 and Olajide, (2011) stated that methanol extracts of A. leiocarpus exhibited scavenging ability and strong reducing ability. According to Mann et al. (2009), the terpenoidal fractions from A. leiocarpus could be a potential source of chemotherapeutic agents (Mann et al., 2009).

The liver plays a central role in the metabolism of drug, xenobiotics, protein synthesis and in maintaining biological equilibrium of organisms. Due to these important roles of liver enzymes, they are used as markers in assessment of drug or plant extract safety or toxicity (Tietz, 1995). The transaminases are involved in intermediate metabolism and are thus present in high concentration in the liver (Tietz, 2000).They are rapidly released into the serum in cases of acute destruction of tissues as in myocardiac infarction or hepatocellular necrosis (Tietz, 1995).There are many enzymes found in the serum that did not originally originate from the serum. During tissue damage, some of these enzymes find their way into the serum, probably by leakage. Serum enzymes measurements are therefore a valuable tool in clinical diagnosis, providing information on the effect and nature of pathological damage to any tissue. Alanine and aspartate amino transaminases are considered to be sensitive indicators of hepatocellular damage, increase in the level of these parameters in the serum after exposure of the experimental animals to xenobiotics such as drugs indicate an injury to the liver (Finlayson et al., 1995). Interestingly, the levels of the electrolytes were brought to near normal levels by Silymarin

**DISCUSSION**

as compared to the gentamicin group. However, the changes of some ions like sodium, potassium, chloride and bicarbonate concentration were restored by the administration of the extract at a concentration-dependent dose.

Table 3. Effect of graded oral doses of A. leiocarpus and gentamicin on some Liver function parameters of rats.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Control</th>
<th>Gentamicin (80 mg/kg)</th>
<th>Gentamicin (80 mg/kg) + Silymarin</th>
<th>Gentamicin + 100 mg A. leiocarpus</th>
<th>Gentamicin + 200 mg A. leiocarpus</th>
<th>Gentamicin + 400 mg A. leiocarpus</th>
</tr>
</thead>
<tbody>
<tr>
<td>ALP (U/L)</td>
<td>176.43±11.97</td>
<td>278.12±4.67&lt;sup&gt;a&lt;/sup&gt;</td>
<td>240.68±8.30&lt;sup&gt;a&lt;/sup&gt;</td>
<td>260.18±7.19&lt;sup&gt;a&lt;/sup&gt;</td>
<td>249.41±4.63&lt;sup&gt;a&lt;/sup&gt;</td>
<td>245.39±12.02&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>AST (U/L)</td>
<td>83.30±6.12</td>
<td>182.72±12.43&lt;sup&gt;a&lt;/sup&gt;</td>
<td>97.20±3.73</td>
<td>148.60±11.24&lt;sup&gt;a&lt;/sup&gt;</td>
<td>122.03±6.92&lt;sup&gt;a&lt;/sup&gt;</td>
<td>94.41±3.25</td>
</tr>
<tr>
<td>Total bilirubin (mg/dl)</td>
<td>1.52±0.43</td>
<td>2.83±0.29&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.62±0.22</td>
<td>1.70±0.16</td>
<td>1.68±0.14</td>
<td>1.60±0.19</td>
</tr>
<tr>
<td>Conjugated Bilirubin (mg/dl)</td>
<td>0.88±0.14</td>
<td>1.17±0.48&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.93±0.27</td>
<td>1.04±0.24</td>
<td>0.98±0.11</td>
<td>0.92±0.09</td>
</tr>
<tr>
<td>Total protein (mg/dl)</td>
<td>39.38±18.73</td>
<td>27.12±7.03&lt;sup&gt;a&lt;/sup&gt;</td>
<td>33.35±15.73&lt;sup&gt;a&lt;/sup&gt;</td>
<td>31.39±3.33&lt;sup&gt;a&lt;/sup&gt;</td>
<td>27.44±4.19&lt;sup&gt;a&lt;/sup&gt;</td>
<td>29.23±2.18&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Albumin (mg/dl)</td>
<td>32.14±8.81</td>
<td>30.12±0.24</td>
<td>31.72±0.25</td>
<td>30.84±1.14</td>
<td>31.75±0.24</td>
<td>30.62±0.13</td>
</tr>
<tr>
<td>APH (U/L)</td>
<td>246.20±5.73</td>
<td>336.00±33.27&lt;sup&gt;a&lt;/sup&gt;</td>
<td>242.36±5.09</td>
<td>311.34±13.35&lt;sup&gt;a&lt;/sup&gt;</td>
<td>263.44±20.16&lt;sup&gt;a&lt;/sup&gt;</td>
<td>240.62±34.28</td>
</tr>
<tr>
<td>ALT (U/L)</td>
<td>34.2±6.86</td>
<td>68.03±3.51&lt;sup&gt;a&lt;/sup&gt;</td>
<td>36.44±3.82</td>
<td>41.86±1.04&lt;sup&gt;a&lt;/sup&gt;</td>
<td>36.14±9.11</td>
<td>35.14±8.88</td>
</tr>
</tbody>
</table>

ALP = Alkaline Phosphatase ,AST = Aspartate Aminotransferase ,APH = Acid Phosphatase, ALT = Alanine Aminotransferase .

Values are expressed as mean ± S.E.M, (n = 6). P values were calculated using one way ANOVA (Analysis of Variance). Mean with different superscript (a) differ significantly, p < 0.05.

Table 4: Effect of graded oral doses of A. leiocarpus and gentamicin on some Kidney function parameters of rats.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Control</th>
<th>Gentamicin (80mg/kg)</th>
<th>Gentamicin (80mg/kg) + Silymarin</th>
<th>Gentamicin + 100mg/kg A. leiocarpus</th>
<th>Gentamicin + 200mg/kg A. leiocarpus</th>
<th>Gentamicin + 400mg/kg A. leiocarpus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urea (mmol/L)</td>
<td>7.86±0.84</td>
<td>8.39±0.95</td>
<td>6.70±0.21&lt;sup&gt;a&lt;/sup&gt;</td>
<td>5.19±0.54&lt;sup&gt;a&lt;/sup&gt;</td>
<td>4.60±0.30&lt;sup&gt;a&lt;/sup&gt;</td>
<td>2.94±0.32&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Creatinine (mmol/L)</td>
<td>80.47±5.17</td>
<td>115.77±2.75&lt;sup&gt;a&lt;/sup&gt;</td>
<td>102.99±23.48&lt;sup&gt;a&lt;/sup&gt;</td>
<td>107.10±21.57&lt;sup&gt;a&lt;/sup&gt;</td>
<td>102.08±7.35&lt;sup&gt;a&lt;/sup&gt;</td>
<td>62.77±5.23&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Na&lt;sup&gt;+&lt;/sup&gt; (mmol/L)</td>
<td>131.41±10.73</td>
<td>140.86±22.19&lt;sup&gt;a&lt;/sup&gt;</td>
<td>130.33±24.17</td>
<td>129.33±23.57</td>
<td>121.30±13.39&lt;sup&gt;a&lt;/sup&gt;</td>
<td>120.66±13.36&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>K&lt;sup&gt;+&lt;/sup&gt; (mmol/L)</td>
<td>13.18±1.04</td>
<td>12.64±0.68</td>
<td>13.32±1.05</td>
<td>13.25±1.03</td>
<td>13.26±1.03</td>
<td>13.32±0.86</td>
</tr>
<tr>
<td>Cl (mmol/L)</td>
<td>87.56±0.17</td>
<td>80.09±1.98</td>
<td>86.43±1.69</td>
<td>81.77±1.59&lt;sup&gt;a&lt;/sup&gt;</td>
<td>82.16±4.43&lt;sup&gt;a&lt;/sup&gt;</td>
<td>87.13±1.95</td>
</tr>
<tr>
<td>HCO&lt;sub&gt;3&lt;/sub&gt;- (mmol/L)</td>
<td>27.00±0.58</td>
<td>25.00±4.93</td>
<td>27.71±0.82</td>
<td>28.84±0.83</td>
<td>29.71±0.82</td>
<td>25.21±0.37</td>
</tr>
</tbody>
</table>

Values are expressed as mean ± S.E.M, (n = 6). P values were calculated using one way ANOVA (Analysis of Variance). Mean with different superscript (a) differ significantly, p < 0.05.
urea, serum creatinine, and sodium ion were found to be significantly (p < 0.05) increased in rats treated with only gentamicin, whereas treatment with methanol extracts of leaves of *Anogeissus leiocarpus* reversed the effect of gentamicin close to normal levels indicating its therapeutic activity. The results of the biochemical studies revealed that the methanol extract of *Anogeissus leiocarpus* restored the adverse effect of gentamicin in groups 4-6 in (Table 4) indicating that the plant exerted therapeutic effects on gentamicin. The findings showed that the effects of *Anogeissus leiocarpus* on Alanine aminotransferase (ALT) in the control group was 34.20±6.86 (U/L), whereas in group 2 treated with gentamicin only at the dose of 80 mg/kg it increased to 68.03±3.51 (U/L). The post treated group 6 (400 mg/kg) showed a significant decrease in alanine aminotransferase from 68.03±3.51 (U/L) to 35.14±8.88 (U/L) as compared with the gentamicin group 2 (p < 0.05). As shown in (Table 4). Aspartate aminotransferase (AST) in the control group was 83.30±6.12(U/L), whereas in the gentamicin group (group 2) it increased to 182.72±12.43(U/L). The post treated group 6 (400mg/kg) showed a significant decrease in aspartate aminotransferase from 182.72±12.43(U/L) to 94.41±3.25(U/L) as compared with the gentamicin group 2 (p < 0.05)suggesting that the plant extract possess some hepatoprotective activities.

Alkaline phosphatase (ALP) in the control group was 176.43±11.97 (U/L), whereas in the gentamicin group (group 2) it increased to 278.12±4.67 (U/L). The post treated group 6 (400 mg/kg) showed a significant decreased in alkaline phosphatase from 278.12±4.67 (U/L) to 245.39±12.02 (U/L) as compared with the gentamicin group 2 (p < 0.05) as shown in (Table 4). Acid phosphatase (APH) in the control group was 246.20±5.73 (U/L), whereas in the gentamicin group (group 2) had increased to 336.00±33.27 (U/L). The post treated group 6 (400 mg/kg) showed a significant decreased in acid phosphatase from 336.00±33.27 (U/L) to 245.39±12.02 (U/L) as compared with the gentamicin group 2 (p < 0.05). Total bilirubin in the control group was 1.52±0.43(mg/dl), whereas in the gentamicin group (group 2) it increased to 2.83±0.29 (mg/dl). The post treated group 6 (400 mg/kg) showed a significant decreased in Total bilirubin from 2.83±0.29 (mg/dl) to 1.60±0.19 (mg/dl) as compared with the gentamicin group 2 (p < 0.05). As shown in (Table 4).

Conjugated bilirubin in the control group was 0.88±0.14 (mg/dl), whereas in the gentamicin group (group 2) it increased to 1.17±0.48 (mg/dl). The post treated group 6 (400 mg/kg) showed a significant decrease in conjugated bilirubin from 1.17±0.48 (mg/dl) to 0.92±0.09 (mg/dl) as compared with the gentamicin group 2 (p < 0.05). As shown in (Table 4). Total protein in the control group was 39.38±18.73 (mg/dl) whereas gentamicin treated group (group 2), it reduced to 27.12±7.03 (mg/dl). The post treated group 6 (400 mg/kg) showed a significant increase to restore the total protein from 27.12±7.03 (mg/dl) to 39.29±2.18 (mg/dl) as compared with the gentamicin group 2 (p < 0.05). As shown in (Table 4).

Albumin in the control group was 32.14±0.81 (mg/dl) whereas gentamicin treated group (group 2), it reduced to 30.12±0.24 (mg/dl). The post treated group 6 (400 mg/kg) showed a significant increase to restore the total protein from 30.12±0.24 (mg/dl) to 30.62±0.13 (mg/dl) as compared with the gentamicin group 2 (p < 0.05). As shown in (Table 4). Serum urea in the control group was 7.86±0.84 (mmol/L) whereas in the gentamicin group (group 2) it increased to 8.39±0.95 (mmol/L). The post treated group 6 (400 mg/kg) showed a significant decrease in Serum urea from 8.39±0.95 (mmol/L) to 2.94±0.32 (mmol/L) as compared with the gentamicin group 2 (p < 0.05). As shown in (Table 4). Since the elevated levels of urea and creatinine are markers of kidney function, (Odoula et al., 2007) it then indicates that gentamicin is nephrotoxic. In renal disease, the serum urea accumulates because the rate of serum urea production exceeds the rate of clearance (Barza et al., 1978). Serum Creatinine in the control group was 80.47±5.17 (mmol/L) whereas in the gentamicin group (group 2) it increased to 115.77±2.75 (mmol/L). The post treated group 6 (400 mg/kg) showed a significant decrease in Serum creatinine from 115.77±2.75 (mmol/L) to 62.77±5.23 (mmol/L) as compared with the gentamicin group 2 (p < 0.05) (Table 4). Elevation of urea and creatinine levels in the serum is taken as the index of nephrotoxicity (Mayne, 1994). Thus, elevations of serum concentrations of these markers are indicative of renal injury. In this study, it was observed that serum urea and creatinine concentrations were significantly increased (p<0.05) in the group 2 animals than group 1 which indicates the induction of severe nephrotoxicity.

Sodium ion in the control group was 131.41±10.73 (mmol/L) whereas in the gentamicin group (group 2) it increased to 140.86±22.19 (mmol/L). The post treated group 6 (400 mg/kg) showed a significant decreased in sodium ion from 140.86±22.19 (mmol/L) to 120.66±12.36 (mmol/L) as compared with the gentamicin group 2 (p < 0.05) (Table 4). Potassium in the control group was 13.18±1.04 (mmol/L), whereas in gentamicin treated group (group 2), it reduced to 12.64±0.68(mmol/L). The post treated group 6 (400 mg/kg) showed a significant effect to normalize the potassium ion from 12.64±0.68 (mmol/L) to 13.32±0.86 (mmol/L) as compared with the gentamicin group 2 (p < 0.05) (Table 4). Chloride ion in the control group was 87.56±2.17 (mmol/L), whereas in gentamicin treated group (group 2), it reduced to 80.09±1.99 (mmol/L). The post treated group 6 (400 mg/kg) showed a significant increase to normalize the chloride ion from 80.09±1.99 (mmol/L) to 87.13±1.95 (mmol/L) as compared with the gentamicin group 2 (p < 0.05). Bicarbonate ion in the control group was 27.00±0.58 (mmol/L), whereas in gentamicin treated group (group 2), it reduced to 25.00±0.49 (mmol/L). The
post treated group 6 (400 mg/kg) showed a significant effect to restore the bicarbonate ion from 25.00±0.49 (mmol/L) to 25.21±0.37 (mmol/L) as compared with the gentamicin group 2 (p < 0.05).

Gentamicin treated animals (Group 2) showed a decrease in body weight compared to the control rat (Group 1). There was an increase in body weight of animals treated with methanol extract of A. leiocarpus respectively, when compared with Group 2. Generally there were decrease in the level of electrolytes due to loss of electrolytes induced by gentamicin. Interestingly the levels of these electrolytes were brought to near normal levels by A. leiocarpus Plant extract in experimental animals. Therefore this study showed that there was a therapeutic change in the damage done to the liver and kidneys, since there was a significant (p<0.05) change in all the parameters assayed for in all the experimenting groups compared to the control group.

**CONCLUSION**

In this study, Alanine aminotransferase, Aspartate aminotransferase, Alkaline phosphatase, Acid phosphatase, Total bilirubin, conjugated bilirubin, serum urea, serum creatinine, and sodium ion were found to be significantly (p<0.05) increased in rats treated with only gentamicin, whereas treatment with methanol extracts of leaves of *Anogeissus leiocarpus* reversed the effect of gentamicin toxicity close to normal levels indicating its therapeutic activity. The results suggest that *A. leiocarpus* has a potent hepatoprotective and nephroprotective agent which is capable of normalizing biochemical abnormalities associated with liver and kidney damage. In the near future, *A. leiocarpus* could constitute a lead to discovering a novel drug which will be useful in the treatment of drug induced hepatic and renal injuries.

**REFERENCES**


