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Research Article

CURATIVE EFFECT OF ABHRAK BHASMA ON LIVER AND KIDNEY FUNCTIONS IN CARBON TETRACHLORIDE INTOXICATED ALBINO RATS

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Abstract: In Ayurveda, many drugs are mentioned to treat liver and kidney diseases. Abhrak bhasma is commonly used Ayurvedic medicine against many diseases including hepatitis. This study was planned to investigate the curative effect of abhrak bhasma in liver and kidney functions in CCl_4 induced hepatotoxicity. Various doses of abhrak bhasma (10, 20, 30 and 40 mg/ kg body wt) were given in curative experimental schedule in male albino rat. Administration of CCl_4 increased serum Aspartate Transaminase (AST), Alanine Transaminase (ALT) and Alkaline Phosphatase (ALP) activities reflected intoxication that produced cellular degeneration or destruction. With increasing doses of abhrak bhasma given to CCl_4 treated rats in 7 days hepatocure schedule normalized the elevated activities of AST, ALT and ALP. Similarly CCl_4 mediated increased contents of conjugated, unconjugated and total bilirubin level was reduced with increasing doses of abhrak bhasma suggesting dose dependent bilirubin clearance efficiency. Abhrak bhasma also mediated urea and creatinine clearance indicating renal curative potency. It was found that abhrak bhasma has more curative effects than SiO_2 doses. The present findings concluded that abhrak bhasma possess dose dependent curative effects against CCl_4 intoxicated liver and kidneys functions in albino rat.

Keywords: Abhrak bhasma, hepatotoxicity, Cellular degeneration, SiO_2, Hepatocure.

INTRODUCTION

Environmental pollution as well as use of antibiotics and other drugs to treat infections causes numerous toxicological alterations and other complications [1,2]. Exposure to hazardous or toxic substances can affect the body in many ways. These substances absorb and travel through the various body systems and affect target organs. It continues to increase the stress on liver and kidney. Since liver and kidney play an essential role in drug metabolisms, detoxification and other biotransformations; its healthy maintenance is of immense importance for health and survival. The elimination of toxic substances is just one of the many functions of the liver and kidneys.

In Ayurveda, many drugs are mentioned to protect or cure liver and kidneys. Evaluation of claims of these drugs, their toxicity if any, and determination of effective doses and duration for treatment are needed to work out so that these drugs can be used by practitioners of other disciplines and can be used in integrated therapy. They can be made available to the common people in reasonable prices. In India number of plants and composite drugs prepared from them are being used for the treatment of hepatitis in traditional medicine and Ayurveda [3-6]. Number of chemicals and drugs have been tried to protect or recover the liver from carbon tetrachloride (CCl_4) induced injury. Liquid paraffin [7], nicotinic acid and ascorbic acid [8], archidonic acid [9], cycloheximide [10] has shown to protect cellular necrosis induced by CCl_4. While liquid paraffin is known to cause toxicity [11]; cyclohexamide is known protein inhibitor [12,13]. Liver produces the metabolic wastes urea, which is taken through the blood to the kidney for filtration. Similarly, some of the products of drug metabolism also carried through the blood for excretion. This may affect the kidney adversely. Kidney is a vital organ that removes the waste from the body and hence must be studied for renal toxicity caused by CCl_4.

In present work abhrak bhasma is used to treat CCl_4 induced hepatotoxicity in curative experimental schedule in male albino rat. Among all bhasmas, abhrak bhasma is considered as Rasayan and Yogawahi, can be used to remodeling of tissues and retardation of aging and can be used against many diseases. Since Abhrak bhasma is a drug to be given to cure hepatic damage. Liver and kidney functional tests were analyzed to detect hepatic and renal dysfunction. The results obtained with treatments of various doses of abhrak bhasma were compared with the silicon dioxide (SiO_2) treated rat, since abhrak bhasma is derived from mica-ore, which is rich in silicates.

MATERIAL AND METHODS

Animal:

Male albino rats, *Rattus norvegicus* (Wistar strain) originally derived from National Institute of Virology, Pune, were bred and maintained in the animal house of

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the department (Reg. No. 233/CPCSEA). The rats were fed with standard pellet diet (Amrit feeds, Sangli, Maharashtra, India). The rats, which are 90 days old and weighed 110-120 g were used for experiments.

Preparation of abhrak bhasma:

Abhrak bhasma was prepared in the laboratory as described in Rasa Ratna Sammucchaya [14]. To study the detailed intermediary effects of abhrak bhasma on liver and kidney different doses viz., 10, 20, 30 and 40 mg/kg body wt were used. The abhrak bhasma was administered 10, 20, 30 and 40 mg/kg body weight with honey. To check this criticism the quartz powder (SiO_2) i. e. naturally occurring silica dioxide was also tested for its curative efficacy to compare it with abhrak bhasma.

Experimental design for hepatocurative activity:

Male albino rats were randomly assigned into several groups of six each as per various treatments given as follows:

Group I: Normal rats without any treatment.

Group II: Acute hepatotoxicity induced by CCl_4 (3.0 ml/ kg body weight/ day for 7 days *subcutaneously*).

Group III: CCl_4 subcutaneous 3.0 ml/ kg body wt/ day for 7 days followed by 10 mg abhrak bhasma/ kg body wt/ day for next 7 days given orally.

Group IV: CCl_4 subcutaneous 3.0 ml/ kg body wt/ day for 7 days followed by 20 mg abhrak bhasma/ kg body wt/ day for next 7 days given orally.

Group V: CCl_4 subcutaneous 3.0 ml/ kg body wt/ day for 7 days followed by 30 mg abhrak bhasma/ kg body wt/ day for next 7 days given orally.

Group VI: CCl_4 subcutaneous 3.0 ml/ kg body wt/ day for 7 days followed by 40 mg abhrak bhasma/ kg body wt/ day for next 7 days given orally.

Group VII: CCl_4 subcutaneous 3.0 ml/ kg body wt/ day for 7 days followed by 10 mg SiO_2/ kg body wt/ day for next 7 days given orally.

Group VIII: CCl_4 subcutaneous 3.0 ml/ kg body wt/ day for 7 days followed by 20 mg SiO_2/ kg body wt/ day for next 7 days given orally.

Group IX: CCl_4 subcutaneous 3.0 ml/ kg body wt/ day for 7 days followed by 30 mg SiO_2/ kg body wt/ day for next 7 days given orally.

Group X: CCl_4 subcutaneous 3.0 ml/ kg body wt/ day for 7 days followed by 40 mg SiO_2/ kg body wt/ day for next 7 days given orally.

Determination of Liver and kidney toxicity:

Collection of serum: On completion of the experimental schedule, animals were killed by giving deep ether anaesthesia. The blood samples were aspirated from the pinna with the disposable syringes and were allowed to clot at room temperature. On clotting, serum samples were obtained by centrifuging the clots using table top centrifuge.

Determination of Hepatocurative activity: To test the curative activity of abhrak bhasma, liver and kidney function tests were determined. Diagnosis of liver function is performed by measuring serum GOT/ AST (Aspartate aminotransferase), GPT/ ALT (Alanine aminotransferase), Alkaline phosphatase (ALP) and bilirubin (conjugated and nonconjugated). Kidney functions were determined by measuring serum urea and creatinine. All these biochemical tests were performed by Auto analyzer (Model No. ECOM-6124F; Manufactured by EPPENDORF).

Statistical Analysis:

All the results obtained were analyzed statistically. The statistical calculations were carried out with the help of XLSTAT 7.5 computer programme. Statistical significance between groups was analyzed by using I-way ANOVA; fooled by student't' test. The values of $p<0.05$, $p<0.01$, $p<0.001$ were considered as significant.

RESULTS

Abhrak bhasma mediated alterations in serum AST, ALT and ALP levels in CCl_4 intoxicated male albino rats:

For the assessment of liver functioning serum AST, ALT and ALP activities were determined. The alterations are given in Table 1.

Table 1: Abhrak bhasma mediated alterations in serum AST, ALT and ALP levels in CCl_4 intoxicated male albino rats during 7 days curative schedule. (Values expressed as units/ml serum).

Sr. No.	Group	AST	ALT	ALP
1	*Normal*	21.95 ± 1.35	19.57 ± 1.2	27.64 ± 1.61
2	*CCl_4 (3.0 ml/kg body wt) sc*	38.29 ± 1.95^{c}	27.53 ± 1.56^{b}	40.23 ± 2.08^{c}
3	*CCl_4 + AB (10 mg/kg body wt) po*	$28.43 \pm 1.75^{a,y}$	22.29 ± 1.28^{x}	32.44 ± 1.85^{x}
4	*CCl_4+ AB (20 mg/kg body wt) po*	26.38 ± 1.56^{y}	$23.58 \pm 1.34^{a,x}$	29.37 ± 1.68^{y}
5	*CCl_4 + AB (30 mg/kg body wt) po*	25.19 ± 1.45^{z}	20.12 ± 1.14^{y}	28.83 ± 1.66^{y}
6	*CCl_4 + AB (40 mg/kg body wt) po*	22.07 ± 1.20^{z}	18.98 ± 1.16^{y}	24.15 ± 1.4^{z}
7	*CCl_4 + SiO_2 (10 mg/kg body wt) po*	$27.15 \pm 1.62^{a,y}$	23.85 ± 1.46^{a}	34.56 ± 1.89^{a}
8	*CCl_4 + SiO_2 (20 mg/kg body wt) po*	26.21 ± 1.48^{z}	25.19 ± 1.55^{a}	32.82 ± 1.79^{x}
9	*CCl_4+ SiO_2 (30 mg/kg body wt) po*	$30.98 \pm 1.89^{b,x}$	27.54 ± 1.68^{b}	30.75 ± 1.77^{y}
10	*CCl_4 + SiO_2 (40 mg/kg body wt) po*	33.15 ± 2.23^{b}	24.66 ± 1.45^{c}	31.27 ± 1.62^{y}

Values are mean ± SE of 6 animals.

p-values – $a<0.05$, $b<0.01$ and $c<0.001$ vs. normal rat; $x<0.05$, $y<0.01$ and $z<0.001$ vs.CCl_4 treated rat

The AST activity in normal rat serum was 21.95 ± 1.35 units/ml. This enzyme activity was elevated by 1.74 fold after CCl_4 administration to the rats. After administration of 10, 20, 30 and 40 mg abhrak bhasma doses, AST activities were increased by 1.3, 1.2, 1.15 and 1.01 folds as compared with normal rat respectively. However, activities showed 25.75, 31.1, 34.21 and 42.36% declines when compared with CCl_4 treated rat enzyme activity. The treatments of 10, 20, 30 and 40 mg SiO_2 to the rats showed 1.24, 1.19, 1.41 and 1.51 folds increases in AST activities respectively, when compared with normal value. In contrast, activities showed 29.09, 31.55, 19.09 and 13.42 % falls after comparison with CCl_4 treated rat enzyme activity.

Serum of normal rat showed 19.57 ± 1.20 units of ALT activity/ml serum. It was increased by 1.41 fold after CCl_4 administration. Increase of 1.12, 1.20 and 1.03 folds were noted after 10, 20 and 30 mg doses of abhrak bhasma respectively, while 40 mg dose did not alter activity after comparison with normal rat enzyme activity. When compared with CCl_4 treated rat, ALT level was decreased by 19.03, 14.35, 26.92 and 31.06 % respectively after 10, 20, 30 and 40 mg abhrak bhasma administrations. Enzyme activities were elevated by 1.22, 1.29, 1.41 and 1.26 folds after comparison with normal rat ALT levels upon 10, 20, 30 and 40 mg doses of SiO_2. Comparison with CCl_4 treated rat ALT activity, there were 13.37, 8.5 and 10.42 % decrease noted after 10, 20 and 40 mg doses, but 30 mg dose did not alter ALT activity.

In normal rat serum, ALP activity was 27.64 ± 1.61 units/ml serum. The activity was increased by 1.46 fold after administration of CCl_4 to normal rat. The rises of 1.17, 1.06, 1.04 folds were observed due to 10, 20 and 30 mg abhrak bhasma administrations, but 40 mg dose showed 12.62% decrease on comparison with normal rat ALP activity. In contrast, decreases of 19.36, 26.99, 28.34 and 39.97 % were observed after 10, 20, 30 and 40 mg abhrak bhasma, when compared with CCl_4 treated rat serum ALP level. Rises of 1.25, 1.19, 1.11 and 1.13 folds were noted over that of normal rat enzyme activity after 10, 20, 30 and 40 mg doses SiO_2 respectively. Upon comparison with CCl_4 treated rat activity declines of 14.09, 18.42, 23.56 and 22.27 % were noted after 10, 20, 30 and 40 mg SiO_2 respectively.

Abhrak bhasma mediated changes in serum bilirubin levels in CCl_4 intoxicated male albino rats:

For the assessment of liver functioning serum conjugated, unconjugated and total bilirubin content were estimated. Alterations are given in Table 2.

Table 2: Abhrak bhasma mediated changes in serum bilirubin levels in CCl_4 intoxicated male albino rats During 7 days curative schedule. (Values are expressed as mg/dl serum).

Sr. No.	Group	Conjugated	Unconjugated	Total
1	Normal	0.15 ± 0.009	0.04 ± 0.002	0.19 ± 0.01
2	*CCl_4 (3.0 ml/kg body wt) sc*	0.24 ± 0.015^{c}	0.06 ± 0.004^{b}	0.30 ± 0.02^{c}
3	*CCl_4 + AB (10 mg/kg body wt) po*	0.22 ± 0.013^{b}	0.05 ± 0.003^{a}	0.27 ± 0.02^{b}
4	*CCl_4 + AB (20 mg/kg body wt) po*	$0.19 \pm 0.011^{a,x}$	0.05 ± 0.003^{a}	0.24 ± 0.02^{a}
5	*CCl_4 + AB (30 mg/kg body wt) po*	0.15 ± 0.010^{z}	0.04 ± 0.002^{y}	0.19 ± 0.01^{z}
6	*CCl_4 + AB (40 mg/kg body wt) po*	$0.12 \pm 0.009^{a,z}$	$0.03 \pm 0.001^{b,z}$	$0.15 \pm 0.01^{a,z}$
7	*CCl_4 + SiO_2 (10 mg/kg body wt) po*	$0.20 \pm 0.011^{b,x}$	0.05 ± 0.003^{a}	0.25 ± 0.03
8	*CCl_4 + SiO_2 (20 mg/kg body wt) po*	0.17 ± 0.008^{y}	0.07 ± 0.005^{c}	0.24 ± 0.02^{a}
9	*CCl_4 + SiO_2 (30 mg/kg body wt) po*	0.14 ± 0.007^{z}	0.07 ± 0.005^{c}	0.21 ± 0.02^{y}
10	*CCl_4 + SiO_2 (40 mg/kg body wt) po*	$0.11 \pm 0.006^{b,z}$	0.08 ± 0.006^{y}	0.19 ± 0.01^{z}

Values are mean ± SE of 6 animals.
p-values – a<0.05, b<0.01 and c<0.001 vs. normal rat
x<0.05, y<0.01 and z<0.001 vs.CCl_4 treated rat

Conjugated bilirubin level in normal rat serum was 0.15 ± 0.009 mg/dl. After CCl_4 administration it was increased by 1.6 fold. Increases of 1.47 and 1.27 folds were noted after 10 and 20 mg abhrak bhasma doses, but the content remained unaltered after 30 mg dose while 40 mg dose caused 20.0 % decrease when compared to normal value. Upon comparison with CCl_4 treated rat conjugated bilirubin content there were 8.33, 20.83, 37.5 and 50.0 % decreases noted after 10, 20, 30 and 40 mg abhrak bhasma treatments respectively. After comparison with normal rat conjugated bilirubin, there were 1.33 and 1.13 folds increases noted after 10 and 20 mg doses of SiO_2, while they were decreased by 6.67 and 26.67 % after 30 and 40 mg doses. Conjugated bilirubin of SiO_2 treated rats when compared with CCl_4 treated rat there were 16.67, 29.17, 41.67 and 54.17 % decreases due to 10, 20, 30 and 40 mg SiO_2 treatments respectively.

Normal rat exhibited 0.04 ± 0.002 mg/dl serum unconjugated bilirubin. It was increased by 1.5 fold after intoxication with CCl_4. Elevations by 1.25 folds were noted after both 10 and 20 mg doses of abhrak bhasma, while 30 mg dose did not alter unconjugated bilirubin content and 40 mg dose resulted in 25 % decrease after comparison with that normal rat serum.

When compared with CCl_4 treated rat 10 and 20 mg doses showed same loss of 16.67 %, while 30 and 40 mg doses showed 33.33 and 50.0 % declines. Unconjugated bilirubin contents were increased by 1.25, 1.75, 1.75 and 2.0 folds after 10, 20, 30 and 40 mg doses of SiO_2 respectively upon comparison with normal rat content. When compared with CCl_4 treated rat contents they were decreased by 16.67 % after 10 mg dose, but increased by 1.17, 1.17 and 1.33 folds after 20, 30 and 40 mg SiO_2 treatments.

Total bilirubin content of normal rat was 0.19 ± 0.01-mg/dl serum. 1.58 fold increased it after CCl_4 intoxication. Increases of 1.42 and 1.26 folds were noted after 10 and 20 mg abhrak bhasma treatments, but it remained same after 30 mg dose, while decreased by 21.05 % after 40 mg dose on comparison with that of normal rat. Decreases of 10.0, 20.0, 36.67 and 50.0 % were noted after 10, 20, 30 and 40 mg abhrak bhasma treatments respectively, when compared with CCl_4 treated rat content. After SiO_2 administrations, unconjugated bilirubin contents were increased by 1.32, 1.26, 1.11 folds after 10, 20 and 30 mg doses, while after 40 mg doses content remained same when compared with normal rat content but they were decreased by 16.67, 20.0, 30.0 and 36.67 % after comparison with 10, 20, 30 and 40 mg SiO_2 treatments respectively.

Abhrak bhasma mediated alterations in serum Urea and Creatinine content in CCl_4 intoxicated male albino rats:

For the assessment of kidney functioning serum urea and creatinine content were estimated. Alterations in serum urea and creatinine contents are given in Table 3.

Table 3: Abhrak bhasma mediated alterations in serum Urea and Creatinine content in CCl_4 intoxicated male albino rats during curative schedule. (Values expressed as mg/dl serum)

Sr. No.	Group	Urea	Creatinine
1	Normal	24.41 ± 1.45	3.02 ± 0.18
2	*CCl_4 (3.0 ml/kg body wt) sc*	38.54 ± 2.19^{c}	4.81 ± 0.27^{c}
3	*CCl_4 + Abhrak bhasma (10 mg/kg body wt) po*	27.31 ± 1.55^{y}	4.10 ± 0.24^{b}
4	*CCl_4 + Abhrak bhasma (20 mg/kg body wt) po*	22.45 ± 1.37^{z}	3.28 ± 0.19^{y}
5	*CCl_4 + Abhrak bhasma (30 mg/kg body wt) po*	21.98 ± 1.27^{z}	3.44 ± 0.21^{a,y}
6	*CCl_4 + Abhrak bhasma (40 mg/kg body wt) po*	25.03 ± 1.42^{z}	3.17 ± 0.19^{z}
7	*CCl_4 + SiO_2 (10 mg/kg body wt) po*	26.13 ± 1.48^{z}	4.55 ± 0.27^{c}
8	*CCl_4 + SiO_2 (20 mg/kg body wt) po*	31.02 ± 1.76^{a,x}	4.39 ± 0.25^{b}
9	*CCl_4+ SiO_2 (30 mg/kg body wt) po*	28.22 ± 1.61^{y}	3.68 ± 0.21^{a,y}
10	*CCl_4 + SiO_2 (40 mg/kg body wt) po*	27.91 ± 1.58^{y}	3.94 ± 0.22^{b,x}

Values are mean ± SE of 6 animals.
p-values – a<0.05, b<0.01 and c<0.001 vs. normal rat
x<0.05, y<0.01 and z<0.001 vs.CCl_4 treated rat

There was 24.41 ± 1.45 mg urea/dl serum in normal rat, which was raised by 1.58 fold after CCl_4 administration to normal rat. Increases of 1.12 and 1.03 folds were noted after 10 and 40 mg dose of abhrak bhasma while decreases of 8.03 and 9.95 % were observed after 20 and 30 mg doses after comparison with normal rat urea content. While 29.14, 41.75, 42.97 and 35.05 % decreases were noted after administrations of 10, 20, 30 and 40 mg Abhrak bhasma after comparison with CCl_4 treated rat urea content. Treatments of abhrak bhasma resulted in 1.07, 1.27, 1.16 and 1.14 folds increases after 10, 20, 30 and 40 mg doses respectively after comparison with urea content in normal rat. On comparison with CCl_4 treated rat enzyme activity they showed 32.2, 19.51, 26.78 and 27.58 % decrease after administration of 10, 20, 30 and 40 mg SiO_2 doses respectively.

Serum of normal rat showed 3.02 ± 0.18 mg/dl creatinine content. Increase of 1.59 fold was noted after administration of CCl_4. Abhrak bhasma treatments resulted in 1.36, 1.09, 1.14 and 1.05 folds increases after 10, 20, 30 and 40 mg doses respectively when compared with normal rat creatinine content.

But decreases of 14.76, 31.81, 28.48 and 34.09 % were noted after 10, 20, 30 and 40 mg abhrak bhasma doses after CCl_4 treated rat content. SiO_2 treated rats serum showed 1.51, 1.45, 1.22 and 1.30 folds increases after comparison with normal rat serum after 10, 20, 30 and 40 mg doses respectively. In contrast there were 5.41, 8.73, 23.49 and 18.09 % decreases estimated after 10, 20, 30 and 40 mg SiO_2 when compared with CCl_4 treated rat creatinine content.

DISCUSSION

The present study was performed to analyze hepatocurative effects of abhrak bhasma in male albino rat. CCl_4 induced hepatotoxicity model is used to study the influence of abhrak bhasma on liver and kidney functions.

After 7 days of administration CCl_4 caused severe liver injuries, that was recognized by production of significantly high activities of the above diagnostic

enzymes indicating no cure of liver functioning with naturally cure activities of the body. This increase in serum AST, ALT and ALP activities reflect intoxication was able to reach the liver and produced cellular degeneration or destruction. It could potentially be attributed to more production of these enzymes and their releasing from the cytoplasm into the blood circulation after rupture of the plasma membrane and cellular damage [15]. It is mediated by free radical metabolites such as $CCl_3^{\cdot}$ and CCl_3COO^- generated by CCl_4, that interact with unsaturated lipid membrane to produce lipid peroxidation and other cellular macromolecules leading to cell damage [16,17]. By increasing doses of abhrak bhasma given to CCl_4 treated rats in 7 days hepatocure schedule normalized the elevated activities of AST, ALT and ALP. ALP activities remained below normal levels indicating release of normal ALP mediated nutritional stress relieved off by abhrak bhasma or may be ALP mediated activities are under stress by highest doses of abhrak bhasma. All doses of SiO_2 given to CCl_4 treated in 7-day hepatocure schedule showed decrease in activities initially but any of the doses had not normalized the activities of AST, ALT and ALP. Thus, abhrak bhasma mediated change in liver function tests in 7 days hepatocure schedule were normalized. These results are in agreement with the results of earlier work in induced acute hepatotoxicity and hepato-protective study [18,19]. The similar effect was observed in protective schedule as our previous work but it showed that the curative effect of abhrak bhasma is more effective than its protective effects against CCl_4 induced liver toxicity [20]. It has been attributed to antioxidant and membrane stabilizing activities of the toxicant agents. It seems to preserve the structural integrity of the hepatocellular membrane as evident from the significant reduction in the CCl_4 induced rise in the serum enzymes. The decreased serum enzymes towards the respective normal value is may be due to the curative effect on the intracellular enzymes by its membrane stabilizing activity as well as repair of hepatic tissue damages caused by CCl_4.

In the present study it has also been observed that CCl_4 mediated increased contents of conjugated, unconjugated and total bilirubin was reduced but the levels were not normalized indicating the intrinsic nature activities that the bilirubin clearance through bile though set in no treatment period. They seemed to be very slow, are not able to clear the arrested bilirubin in serum and remained high. But abhrak bhasma mediated alteration indicated the dose dependent changes in conjugated and unconjugated levels of bilirubin and total bilirubin indicated in 7 day cure schedule the bilirubin clearance efficiency was normalized may be helping through the set in abhrak bhasma specific processes for the stability of biliary dysfunction in rat liver during chronic hepatic injury [21]. SiO_2 doses in similar conditions have shown the initial drop in the bilirubin levels but this was not preceded further in any of the hepatocure schedule. The results indicated that some basic stimulation might have been given by SiO_2 mediated activities towards the bilirubin clearance in 7 days hepatocure schedule. But further SiO_2 mediated activities seem to fail in bilirubin clearance i.e. SiO_2 specific some of the metabolisms may not be utilizing the favorable path of liver function normalizing and seem to interfere with initial setting in of the improvement in liver function tests.

Abhrak bhasma mediated creatinine-controlling urea and creatinine clearance may be of such activities or may be natural pathways hurried by the abhrak bhasma mediated area and creatinine clearance. Administration of CCl_4 induced a significant increase in creatinine concentration. Treatments of increasing abhrak bhasma doses reduced this elevated level, indicating renal curative potency of abhrak bhasma. But SiO_2 though basically showed this dependency with all the form doses was not able to maintain it with all the four doses and therefore it is not equally potent as abhrak bhasma in renal function cure.

It has been reported that CCl_4 induced damage in liver and kidneys is mediated mainly through free radical generation. Therefore substances with antioxidative and free radical scavenging potency are promising approach in protecting liver and kidney toxicity [22,23]. The present results show that abhrak bhasma reduces the toxicity to the rat liver and kidney induced by CCl_4.

From the results, it is concluded that abhrak bhasma exert dose dependent hepatocurative effects with greater effects at higher doses in CCl_4 induced toxicity in rat. However, further studies are needed to better evaluate these activities and the proper treatment for the liver diseases.

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