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Pectin in the reversal of renal failure/uremia.

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Abstract

The author found that pectin with a purity (the content of Gal-A (Galacturonic Acid)) greater than 98% has a certain therapeutic effect on patients with chronic kidney disease, especially those with renal failure and uremia. We will show the relevant results that can support the above findings through animal experiments and clinical trials respectively. We are surprised that not only can it rapidly reduce and maintain serum creatinine levels for a period of time, allowing patients to stop dialysis and stay away from dialysis, but also has amazing performance in reversing glomerular filtration rate and improving kidney size. This technology provides a new method to replace dialysis or kidney replacement therapy for patients with renal failure/uremia in the past.

Keywords: Pectin, Chronic kidney disease, Glomerular filtration rate, Kidney size, Creatinine, Reversible.

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Introduction

Kidney disease has become an important disease affecting human health worldwide. According to the 2020 WHO report, Chronic Kidney Disease (CKD) has become the top 10 cause of death [1]. In developed countries such as the United States and Norway, for example, the prevalence of CKD in the adult population ranges from 10.2% to 13.0%, and the prevalence of CKD in China has also reached 10.8% [2]. Moreover, CKD and its resulting End-Stage Renal Disease (ESRD) patients are increasing year by year, and it has become a worldwide public health problem that seriously jeopardizes human health [3].

Pectin, a class of heteropolysaccharides based on polygalacturonic acid, is a safe and non-toxic natural food additive recommended by the Joint FAO/WHO Committee on Food Additives with no daily additive limit [4]. Pectin is mainly obtained from apple pomace, citrus peels and sugar beets, and currently, commercial pectin contains ≥ 65% Gal-A (Galacturonic Acid), and the authors were able to achieve more than 98% Gal-A after optimizing the pectin extraction technique.

Among the patients who tried to take the pectin, the authors unexpectedly found that the pectin had a significant effect on the rapid reduction of blood creatinine concentration in patients with chronic kidney disease. So, the authors followed up the physical examination indexes of chronic kidney disease patients who took the pectin, and surprisingly found that the pectin not only can make the patients rapidly reduce and stabilize the blood creatinine concentration in a period of time, but also has a surprising performance in the reversal of glomerular filtration rate and improvement of the size of the kidney and so on.

In the following, we will show the relevant results that can support the above findings through animal experiments and clinical trials respectively.

Animal experiments

Experimental animals: 6-8 weeks old Wistar male rats, body weight range: 200 ± 20 g, SPF grade, provided by Spivey (Beijing) Biotechnology Co.

Materials and Methods

Experimental methods

Subject administration route, period, frequency

The first step of modeling: Adenine was administered by oral gavage for 4 weeks, and the frequency of administration was once a day. Carboxymethylcellulose solution containing 300 mg/kg adenine was administered by gavage in a volume of 10 mL/kg every day for 2 weeks, and then changed to every other day for 2 weeks.

The second step of drug administration: After successful modeling and administration of pectin for 1 week, three rats were taken from each group and put to death, blood and urine were collected to test the corresponding indexes, and kidney tissues were removed for histopathological examination to evaluate the therapeutic effect of the raw materials on CKD renal fibrosis.

Reasons for the design of subject dose

Human (standard body weight 60 kg) was administered three times a day, 100 mg each time, *i.e*., the commonly used clinical human dose was 300 mg/kgBW·d, and the dosage for rats (mg/kg)=the commonly used clinical human dose (mg/kg) × the equivalent area coefficient for rats (6.25). As a result of this formula, the high dose was 1875 mg/kgBW·d, the medium dose was 937.5 mg/kg and the low dose was 468.7 mg/kg.

Animal grouping

In this experimental study, there were five experimental groups, ranging from 4-6 animals per group, *i.e*., healthy group (4 animals), healthy donor group (4 animals), model group (6 animals), high-dose group (6 animals), medium-dose group (6 animals), low-dose group (6 animals). The operation was carried out according to the experimental protocol and the subject preparations were mixed well with continuous slight shaking before and during administration.

Observation indexes and methods

Observation of clinical symptoms: Recorded once a day, including nutritional coat, mental state, appetite, head, respiration and other abnormalities.

Observations before and during the start of the experiment: Weighing and collecting blood and urine samples to monitor 24-hour urinary micro-protein (uALB), serum creatinine (CREA), and urea nitrogen (UREA) before the start of the experiment and every week during the period of the experiment, and after 4 weeks, except for the healthy group and the healthy test group, in which 1 rat was killed in each group, the remaining groups were killed by 2 rats in each group. Collection of blood and urine for uALB, CREA and UREA indicators, and the renal tissues were removed to take photographs and weighed to be subjected to histopathological examination (HE and Masson) to evaluate the modeling.

Results and Discussion

Experimental data processing and statistical analysis

The data of this experiment were expressed as mean ± standard deviation and were analyzed by t-test for independent samples using SPSS software.

Data analysis

Figure 1 shows a schematic diagram of rat kidney tissue removed from each group after the end of modeling (4 weeks later) (including 1 pair in the healthy group and 1 pair in the healthy donor group, and 2 pairs in the other groups). The third and fourth pairs in the upper row (from left to right) are the removed tissues of the healthy group and the healthy donor group, while the rest are the modeling group. Combined with the pathological examination (HE and Masson) analysis of each group of tissues shown in Figures 2 and 3, it indicates that the model construction was successful. Moreover, further testing results based on this model are as follows.

Figure 1. *Shows a schematic diagram of rat kidney tissue removed from each group after the end of modeling (4 weeks later).*

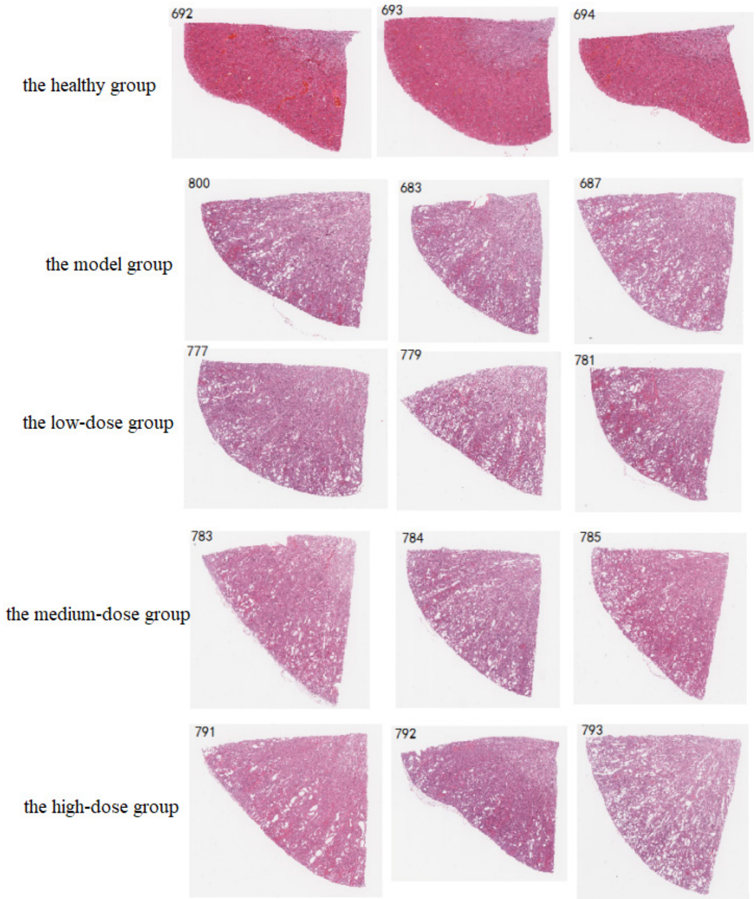

Figure 2. *Shows a comparison of Histopathological Examinations (HE) between different groups.*

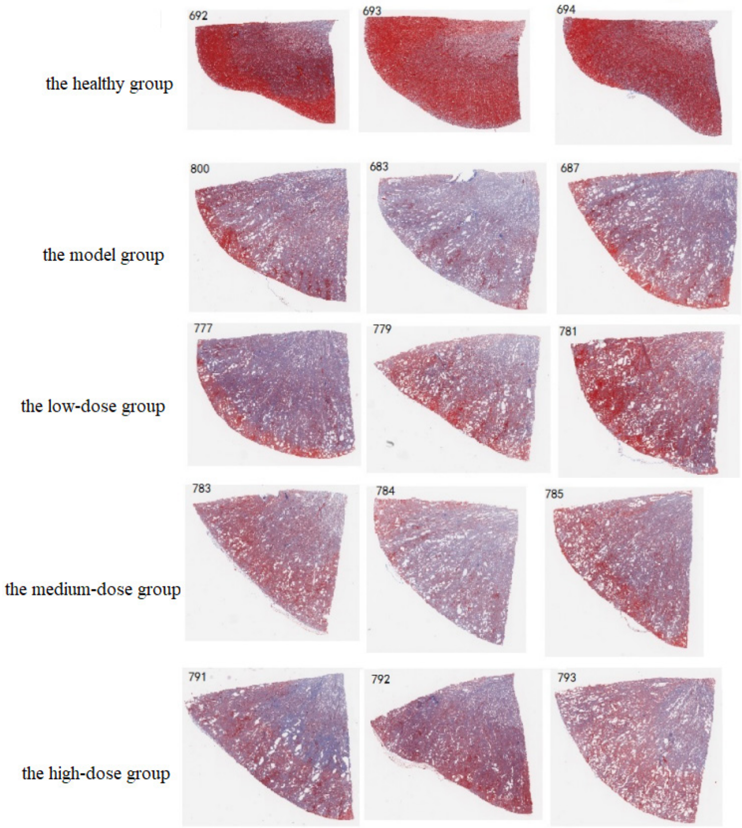

Figure 3. *Shows a comparison of histopathological examinations (Masson) between different groups.*

Changes of main indexes

As can be seen in Tables 1-3, the experiment mainly tracked the changes of the three main indicators: 24-hour urine microbial protein (uALB), serum creatinine (CREA), and urea nitrogen (UREA). Comparison of the post-feeding assay (W5) with the assay at the end of modeling (W4) revealed.

Table 1. *Statistics of urine microbial protein (uALB) data.*

Groups	NO.	uALB (mg/L)					
		D0	W1	W2	W3	W4	W5
CDK model control group	800	49.561	11.033	11.890	23.487	30.709	29.486
	683	34.916	8.115	23.233	28.754	19.124	21.070
	684	44.846	34.066	17.934	27.442	16.538	-
	685	35.372	25.968	16.721	33.731	19.718	22.934
	686	40.013	11.937	22.577	23.169	19.841	-
	687	55.502	8.821	21.718	29.690	18.284	13.367
Health - test group	688	54.403	8.562	15.958	38.103	12.689	-
	689	22.905	15.013	26.056	22.108	31.100	7.869
	690	19.815	17.979	30.137	20.026	16.355	6.879
	691	19.517	10.361	17.423	34.500	17.738	14.201
Health group	692	42.565	12.406	17.819	41.774	22.581	24.165
	693	39.793	9.216	21.718	30.303	13.198	12.787
	694	34.647	16.816	16.206	50.177	11.242	21.532
	695	53.165	9.165	29.651	32.562	12.658	-
Low-dose group	776	32.596	7.675	24.271	33.137	22.058	-
	777	24.955	17.678	23.572	27.130	23.775	19.873
	778	36.696	14.778	15.523	37.185	22.890	25.248
	779	20.696	34.680	18.227	40.481	32.048	24.085
	780	29.188	10.016	16.151	23.731	30.709	-
	781	16.982	9.659	16.635	43.381	15.119	7.969
Medium-dose group	782	26.680	19.190	15.459	43.619	16.584	-
	783	20.969	14.638	16.198	18.033	21.002	21.251
	784	66.078	10.721	13.462	40.691	22.505	16.872
	785	31.713	10.361	18.049	46.148	16.866	15.849
	786	52.820	9.090	25.712	34.657	15.445	21.251
	895	33.943	42.461	25.258	32.562	8.394	-
High-dose group	773	13.514	8.773	18.406	20.793	24.719	-
	789	42.444	30.696	24.129	40.481	18.724	21.517
	790	28.228	8.202	39.500	28.754	25.262	-
	791	39.683	11.937	24.214	25.941	10.039	11.951
	792	26.742	8.136	34.150	32.421	24.111	12.881
	793	33.943	10.158	22.532	30.428	11.507	9.451

Table 2. *Serum creatinine (CREA) data statistics.*

Groups	NO.	CREA (umol/L)					
		D0	W1	W2	W3	W4	W5
CDK model control group	800	43.359	69.745	97.970	106.319	141.320	108.377
	683	45.551	69.465	90.599	127.664	145.950	130.364
	684	45.455	65.262	68.970	82.210	107.101	-
	685	41.281	71.461	166.302	127.646	171.713	103.542
	686	43.786	64.773	94.385	99.341	171.466	-
	687	35.229	63.27	96.343	107.272	148.009	141.050
Health - test group	688	44.829	47.786	52.206	53.150	58.700	-
	689	28.383	57.289	50.505	49.341	52.150	57.598
	690	42.435	49.197	49.641	55.764	56.318	51.754
	691	44.012	59.563	48.823	54.831	56.684	-
Health group	692	51.731	49.987	50.465	49.989	59.973	54.051
	693	45.029	45.904	52.662	55.806	51.731	51.137
	694	44.781	47.491	48.506	59.712	52.608	56.304
	695	41.936	52.024	53.971	55.076	62.880	-
Low-dose group	776	49.118	64.413	74.120	187.384	301.247	-
	777	42.911	59.853	197.706	88.211	163.127	127.304
	778	42.465	58.693	86.641	76.761	111.295	93.101
	779	38.718	63.133	82.690	80.662	187.622	121.410
	780	44.93	59.396	78.223	85.811	153.441	-
	781	45.477	65.453	80.197	77.496	126.586	97.356
Medium-dose group	782	40.819	60.786	87.728	110.630	216.039	-
	783	43.294	64.577	130.189	148.513	215.556	136.876
	784	40.301	67.51	97.727	94.201	154.811	109.669
	785	47.301	68.374	96.150	87.933	198.571	164.463
	786	38.303	67.436	103.982	97.004	222.462	209.690
	895	39.781	72.955	94.150	102.090	174.551	-
High-dose group	773	41.941	54.576	77.950	83.003	141.840	-
	789	50.435	65.336	105.717	87.078	116.790	92.839
	790	38.776	60.389	84.811	86.099	145.926	-
	791	40.288	65.227	97.869	90.422	160.827	163.142
	792	46.547	69.501	89.578	80.448	137.452	115.515
	793	44.999	70.201	131.893	156.806	248.958	353.067

Table 3. *Statistics of urea nitrogen (UREA) data.*

Groups	NO.	UREA (mmol/L)					
		D0	W1	W2	W3	W4	W5
CDK model control group	800	5.005	13.274	21.673	21.643	29.197	15.432
	683	4.862	12.251	18.475	29.449	28.555	20.607
	684	5.221	10.697	10.308	14.530	19.599	-
	685	6.088	12.428	29.432	30.510	37.534	19.556
	686	5.474	9.896	22.829	21.282	44.218	-
	687	5.511	9.775	18.764	21.839	29.602	22.794
Health - test group	688	5.022	5.913	4.648	4.941	5.776	-
	689	3.825	5.329	5.718	4.503	5.165	3.510
	690	4.804	4.918	4.403	4.635	5.943	5.360
	691	5.102	5.444	5.422	5.047	6.030	-
Health group	692	5.118	4.98	4.970	5.428	5.231	4.363
	693	5.347	3.594	3.799	3.989	4.450	3.971
	694	5.357	4.029	5.296	5.329	4.908	7.558
	695	4.295	4.764	3.923	4.491	4.290	-
Low-dose group	776	5.552	9.534	14.296	39.252	56.349	-
	777	5.377	11.613	32.718	25.477	34.283	22.211
	778	5.35	10.482	17.915	15.453	24.145	16.563
	779	4.437	13.538	16.085	20.538	37.361	24.370
	780	5.494	8.807	18.629	19.753	32.481	-
	781	5.607	9.334	16.368	20.497	27.396	16.381
Medium-dose group	782	4.908	9.994	21.108	32.931	39.387	-
	783	5.417	12.01	25.459	22.560	40.081	23.406
	784	4.263	11.808	21.356	21.377	28.197	20.956
	785	4.364	13.805	20.702	24.591	35.996	27.314
	786	4.542	11.516	20.836	28.291	45.252	45.001
	895	4.768	12.075	16.191	23.555	30.690	-
High-dose group	773	4.75	8.306	16.314	20.225	28.511	-
	789	4.773	9.319	18.528	20.697	21.598	18.734
	790	4.839	9.267	16.870	20.444	23.405	-
	791	5.874	10.324	18.378	19.985	27.523	25.075
	792	6.386	13.173	15.944	18.451	23.810	17.128
	793	5.608	12.048	22.830	37.290	50.228	59.613

In the urine micro-protein data in the low to high dose group, there appeared five rats with elevated indicators after drug administration, with the percentage of elevation ranging from 1-30%, which accounted for 27.8% of the experimental rats. In the remaining rats, urinary micro-protein decreased with an effective rate of more than 70%, and the rate of decrease was between 18-52%; in the serum creatinine data, there were two rats with elevated creatinine values, and the percentage of elevation ranged from 1.6-2.5% (and they were all in the high-dose group), accounting for 11.1% of the experimental rats, and the rest of the rats had creatinine decreases of between 16-35%, with an effective rate of 80%-90%; In the urea nitrogen data, there was an increase in the urea nitrogen value of one rat, with an increase ratio of 1.9%, accounting for 5.6% of the experimental rats, and the decrease of urea nitrogen index of the rest of the rats ranged from 10.2-35%, with an effective rate of more than 90%. From the above three sets of data, it can be reasonably predicted that pectin has a significant effect on the rapid reduction of blood creatinine concentration in patients with chronic kidney disease.

Histopathological findings

The authors further compared the histopathological examinations (HE and Masson) of the healthy group (Nos. 692, 693, 694), the model group (Nos. 800, 683, 687) with the low-dose (Nos. 777, 779, 781), medium-dose (Nos. 783, 784, 785) and high-dose (Nos. 791, 792, 793) groups as shown in Figure 1-2 shown. From the comparison of the pictures of each group in Figure 1 (HE) and Figure 2 (Masson), it can be seen that compared with the model group, the degree of fibrous tissue proliferation in the low-dose group to the high-dose group was reversed to varying degrees, which can support the conclusion that pectin with a purity of >98% has a certain degree of therapeutic effect in patients with chronic kidney disease, especially in patients with renal failure and uremia.

Clinical experiments

Reversal of glomerular filtration rate

Glomerular Filtration Rate (GFR) is an indicator of kidney function. A decrease in glomerular filtration rate represents that the function of the kidneys to excrete metabolic wastes is affected.

As to whether the decline in glomerular filtration rate is reversible or not, the general knowledge of professional clinicians is that it depends on whether the patient's renal impairment is acute or chronic. If it is caused by acute renal failure, patients can be given hormones and other drugs for treatment. Usually, with active treatment, the symptoms of acute renal failure can be effectively improved, and then the decrease in glomerular filtration rate can be reversed. However, if it is caused by chronic renal impairment, the patients' glomerular filtration rate decline is irreversible. Because this stage means that a lot of the patient's renal tissues have been destroyed, and a large number of glomerulosclerosis and tubular atrophy and fibrosis occur [5].

The authors found such a patient (hereinafter referred to as "Patient 1"), Mr Li, age 32 years old, weight 77 Kg, with a history of uremia, since June 1, 2014 began hemodialysis, three times a week, dialysis to date has been 8 years, dialysis with levocanidin, erythropoietin, no other medication. On June 4, 2021, he started to take pectin (each capsule contains 0.6 g of pectin powder), 2 times/day, 5 capsules/times; from December 19, 2021 to the present, the dosage has been adjusted to 2 times/day, 7 capsules/times, and during the time of taking pectin, he only takes allicin and no other medication.

During the course of taking pectin, when comparing the patient's renal dynamic imaging reports at 6 and 10 months of taking the pectin, it was strikingly found that the decrease in glomerular filtration rate due to chronic renal impairment had been significantly reversed, *i.e.*, the glomerular filtration rate had increased from 0.438 ml/min to 2.963 ml/min over the course of the four months, of which, the left kidney had increased from 0.194 ml/min to 1.706 ml/min and the right kidney from 0.243 ml/min to 1.257 ml/min, respectively. We hypothesized that at this rate of recovery, glomerular filtration rate would probable recover to more than 15% within the next two years.

Improvement of kidney size

The pathological basis of renal atrophy is usually glomerulosclerosis, tubular atrophy, interstitial fibrosis, etc., which means that the kidneys are in a state of fibrosis. The size of kidneys will be reduced after fibrosis occurs. The most common cause of renal atrophy is chronic renal insufficiency, and it is a common knowledge in the medical field that once chronic renal insufficiency occurs, resulting in renal atrophy, the size of the kidneys generally cannot be restored. In this case, all that can be done is to target the choice of treatment program to prevent further damage to renal function and slow down the rate of deterioration of renal function.

However, patient 1's atrophied kidneys were also enriched during the ten months of taking pectin. When comparing the ultrasound reports of 6 months and 10 months of taking pectin, it was found that the size of the left kidney was enriched from 5.4 × 1.9 x 2.0 cm to 7.5 x 3.1 × 5.2 cm, and that of the right kidney from 8.0 × 3.0 × 2.9 cm to 8.9 × 4.2 cm in the past four months to 8.9 × 4.4 × 5.6 cm, with the kidney size filling close to 30%, respectively.

We hypothesized that this may be due to the improved blood supply to the atrophied kidneys, *i.e.*, the pectin molecules unclogged the glomerular arterioles and improved the microcirculation of the kidneys, which allowed the atrophied kidneys to be reversed and re-filled to a larger size.

Rapid reduction and stabilization of blood creatinine concentration

Creatinine (Cre) is the end product of creatine and phosphocreatine metabolism, and the source of creatinine in blood includes both exogenous and endogenous parts. Almost all of the blood creatinine enters the primary urine through glomerular filtration and is not reabsorbed by renal tubules, so the measurement of the blood creatinine concentration can reflect the filtration function of the glomerulus. The general knowledge of the medical profession is that chronic renal failure has a long course, and a large amount of renal tissue is destroyed in patients. Since regeneration of the already lost renal tissue is impossible, the creatinine of patients cannot be decreased, and the disease can only be controlled by some therapies such as medications for palliation.

The authors found such a patient (hereinafter referred to as "Patient 2") among the patients taking pectin. Mr Xie, age 40, body 75 Kg, diagnosed with uremia in February 2021, hospitalized for hemodialysis treatment, discharged on February 20, blood creatinine concentration of 918 umol/L, with type 2 diabetes mellitus, hypertension, history of heart failure. 20 February 20, 2021 began to take pectin (pectin powder contained in each capsule of 0.6 g), 3 times/day, 10 capsules/times, during the period of pectin consumption. taking erythropoietin, antihypertensive drugs and no other medications.

During the course of taking pectin, the authors were surprised to find that the patient's blood test data, including blood creatinine concentration (labeled as creatinine in the test report), urea, cystatin C, and glomerular filtration rate, improved significantly over the nine-month period.

Specifically, in just ten days, patient 2's creatinine value dropped from 918 umol/L at discharge to 776 umol/L; at one month of administration, patient 2 moved from the uremic stage to the renal failure stage; at the next continuation of two and a half months of administration (*i.e.*, at three and a half months of administration), the creatinine value dropped rapidly to 265 umol/L, and patient 2 entered the renal dysfunction stage, and in the following five and a half months to maintain and stabilize the gradual repair of the kidneys in the stage of renal failure (refer to the staging method of chronic renal failure [6]). The relevant indicators in the test report form are shown in Tables 4.1 and 4.2, where "-" indicates that the indicator was not detected at the time of testing.

Table 4.1. *Summary of relevant indicators in the test report form.*

No.	Indicator name	Unit	Reference value range	Test reports						
				2021/3/1	2021/3/19	2021/3/31	2021/4/14	2021/4/28	2021/5/12	2021/6/2
1	Glutamine aminotransferase	U/L	9-50	10.0	-	10.0	10.0	10.0	9.0	12.0
2	Glutamate aminotransferase	U/L	15-40	6.0	-	8.0	12.0	10.0	7.0	10.0
3	Triglycerides	mmol/L	0.30-1.92	1.76	-	2.02	2.35	2.08	1.42	1.71
4	Cholesterol	mmol/L	2.32-5.62	5.20	-	6.12	6.67	4.48	5.71	5.59
5	Urea	mmol/L	3.1-8.0	50.01	26.7	23.2	23.0	23.2	25.0	17.6
6	Creatinine	umol/L	31-132	776	550	503	450	302	383	265
7	Cystatin C	mg/L	0.3-1.2	-	-	-	4.52	3.00	-	-
8	Uric acid	umol/L	89.2-416	602	455	530	549	420	552	410
9	Glucose	mmol/L	3.90-6.16	4.81	4.8	4.67	5.05	4.67	5.05	4.82
10	Potassium	mmol/L	3.5-5.3	4.6	4.0	3.94	3.96	3.88	-	4.08
11	Sodium	mmol/L	137-147	139	141	141.7	142.3	141.9	-	143.2
12	Chlorine	mmol/L	99-110	100	98	102.3	104.1	103.1	-	104.7
13	Phosphorus	mmol/L	0.85-1.51	2.57	-	1.19	-	-	-	-
14	Glomerular filtration rate (calculated value)	ml/ min/1.73	-	6.78	10.28	11.45	13.10	21.22	15.92	24.85
Duration instructions				Ten days	one month	one and a half months	two months	two and a half months	three months	three and a half months

Table 4.2. Summary of relevant indicators in the test report form.

No.	Indicator name	Unit	Reference value range	Test reports						
				2021/6/23	2021/7/13	2021/8/4	2021/9/8	2021/10/7	2021/11/10	2021/12/8
1	Glutamine aminotransferase	U/L	9-50	12.0	-	10.0	9.0	11.0	-	12
2	Glutamate aminotransferase	U/L	15-40	11.0	-	10.0	11.0	10.0	-	10
3	Triglycerides	mmol/L	0.30-1.92	2.15	-	1.55	1.76	-	-	1.2
4	Cholesterol	mmol/L	2.32-5.62	5.83	-	4.81	4.91	-	-	4.95
5	Urea	mmol/L	3.1-8.0	17.3	16.56	16.63	17.9	23.13	17.85	15.96
6	Creatinine	umol/L	31-132	244	262	244	245	228	202	211
7	Cystatin C	mg/L	0.3-1.2	2.63	-	3.16	-	2.79	2.6	2.46
8	Uric acid	umol/L	89.2-416	376	-	501	364	399	-	428
9	Glucose	mmol/L	3.90-6.16	4.66	-	-	4.26	-	-	5.72
10	Potassium	mmol/L	3.5-5.3	3.99	4.25	-	4.91	-	-	-
11	Sodium	mmol/L	137-147	142.6	141.7	-	141.2	-	-	-
12	Chlorine	mmol/L	99-110	102.5	106.6	-	106.8	-	-	-
13	Phosphorus	mmol/L	0.85-1.51		-	-	-	-	-	1.13
14	Glomerular Filtration Rate (calculated value)	ml/min/1.73	-	27.46	25.20	27.46	27.32	29.81	34.51	32.73
Duration instructions				four months	four and a half months	five months	Six months	seven months	eight months	nine months

Interestingly, a reversal of glomerular filtration rate was also observed in Patient 2, whose glomerular filtration rate increased from an initial 6.78 ml/min/1.73 to 34.51 ml/min/1.73 after eight months of administration, which supports the findings under 1) above. The GFR (Glomerular Filtration Rate) (CKD-EPI) was calculated as follows.

$$eGFR = a \times \left(Scr/b\right)^{c} \times \left(0.993\right)^{\text{年龄}}$$

Based on the effect of patient 2 administration, it was found that for patients with renal failure/uremia, the rise in creatinine due to chronic renal insufficiency can also be decreased, not only can it be decreased, but also can be rapidly decreased in a relatively short period of time and stabilized in its state after it has been decreased to a certain degree [7].

Conclusion

In summary, the authors found that pectin with purity >98% has certain therapeutic effect on chronic kidney disease patients, especially renal failure and uremia patients, not only can make patients rapidly reduce and stabilize blood creatinine concentration in a period of time, so that they can stop dialysis and stay away from dialysis, but also has amazing performance in reversing glomerular filtration rate and improving the size of kidneys. As to how the pectin realized the above effects, its pathopharmacology and pharmacology research will be explored in detail in the next step, but this at least provides a feasible solution for the treatment of patients with renal failure and uremia.

Data Availability Statement

All data generated or analysed during this study are included in this published article.

Animal ethics statement

-The study is reported in accordance with ARRIVE guidelines.

-All experimental protocols were approved by Ethics Committee of Beijing Experimental Animal Research Center.

-All experimental protocols follow the GB/T 35892-2018 Guidelines for Ethical Review of Laboratory Animal Welfare and DB11/T 1734-2020 Technical Specifications for Ethical Review of Laboratory Animal Welfare.

Statement on the method of killing mice

The mice were killed using carbon dioxide anesthesia followed by bloodletting.

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