

Serum Aluminium Levels in Stage 4, 5 & 5D Chronic Kidney Disease

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ABSTRACT

Objectives: Use of non aluminum containing phosphate binders and improvement in feed water quality for hemodialysis (HD) have resulted in low prevalence of aluminum toxicity. The study was undertaken to detect the prevalence of high serum Al levels in chronic kidney disease (CKD) patients & controls.

Settings & Design: Cross sectional study carried out at a tertiary care teaching hospital.

Methods: We analyzed the serum aluminum levels in 32 CKD patients & 32 controls. It was one time sample check. The levels were correlated with anemia, Serum PTH levels, neurologic features if any. Iron status was noted in all.

Results: All 32 CKD patients were anemic with Hb levels between 7 to 10 gm%. None were on any medication containing aluminum. Al level in our institute tap water & R.O. Water was < 5µg/l. Six volunteers showed Serum Al level range between 5 to 15 µg/l while it was undetectable in remaining 26 volunteers. All 32 CKD patients had Serum Al > 5µg/l: 23 with 5 to 40 µg/l, 8 with 40 to 200 µg/l & one > 200 µg/l. Of 23 pts [in range 5 to 40 µg/l], 19 were on Maintenance HD while 4 were pre -HD. All 8 patients [in range 40 to 200 µg/l] & the one with S.Al > 200 µg/l were on HD. The difference in serum aluminium levels in two groups [Control Vs CKD] was significant (p< 0.001). The co-relation between

serum Al level and Calcium (p- 0.21), as well as iPTH (p- 0.42) were not significant. None had any neurologic features.

Conclusions: Serum aluminum was detectable in only 23% of controls but 100% in CKD patients. Though detectable, the levels were not significant in controls. However, 28% of CKD had S.Al >40 µg/l. Though it is a small study group, it highlights significantly higher levels, the source of which needs to be looked into.

Key words: Serum Aluminium, Chronic Kidney Disease, Hemodialysis.

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INTRODUCTION

Aluminum is a trivalent cation found in its ionic form in most kinds of animal and plant tissues and in natural waters everywhere. It is the most abundant metal in the earth's crust.¹ Aluminum is absorbed from the GI tract in the form of oral phosphate-binding agents (aluminum hydroxide), parenterally via immunizations, via dialysate on patients on dialysis or total parenteral nutrition (TPN) contamination, via the urinary mucosa through bladder irrigation, and transdermally in antiperspirants. Lactate, citrate, and ascorbate all facilitate GI absorption.¹

Approximately 95% of an aluminum load becomes bound to transferrin and albumin intravascularly and is then eliminated renally. In healthy subjects, only 0.3% of orally administered aluminum is absorbed via the gastrointestinal (GI) tract, and the kidneys effectively eliminate aluminum from the human body. Only when the GI barrier is bypassed, such as by intravenous infusion or in the presence of advanced renal dysfunction, does aluminum have the potential to accumulate.² If a significant aluminum load exceeds the body's excretory capacity, the excess is deposited in

various tissues, including bone, brain, liver, heart, spleen, and muscle. This accumulation causes morbidity and mortality through various mechanisms.³

Aluminum toxicity occurs in dialysis patients or Chronic Kidney Disease (CKD) patients with GFR <30 mL/min/1.73 m² (CKD Stages 4 and 5) because aluminum that is absorbed from the gut or that enters the body from dialysate or another parenteral route is not excreted, or is inadequately excreted by the diseased kidneys.^{4,5}

When aluminum accumulates in dialysis patients, it is only slowly removed by dialysis because 90% of aluminum is bound to serum proteins (primarily transferrin).^{6,7} The aluminum entering the body accumulates in various tissues, including bone, brain, parathyroid glands, and other organs.^{8,9} Such accumulation of aluminum can produce toxicity with several distinct syndromes, depending on the rate and magnitude of aluminum loading. Aluminum intoxication can cause dialysis encephalopathy¹⁰, a demineralizing osteodystrophy¹¹, and anemia.¹²

With non aluminium containing phosphate binders & use of reverse osmosis in dialysis centres, occurrence of aluminum intoxication in dialysis centers, with associated severe clinical manifestations, have virtually disappeared.¹³

Recently, one of our hemodialysis patient, who was resistant to erythropoietin [EPO], in spite of iron repletion, no infection, normal bone marrow and no EPO antibodies, revealed serum Al levels beyond permissible limits (unpublished data).

This pilot project was planned to assess serum Aluminium (Al) levels in CKD stage 4, 5 & 5D.

AIMS AND OBJECTIVES

- To compare the prevalence of serum Al levels in patients with CKD stage 4 & 5, 5D & with control group of healthy volunteers.
- To correlate Sr. Al levels with anemia, serum intact parathyroid hormone [iPTH], serum calcium in CKD group

MATERIALS AND METHODS

This was a population based cross sectional study carried out at a teaching hospital after due permission from the Institutional Ethics committee. Sample size was 64: 32 of CKD & 32 with normal kidney function as assessed by estimated Glomerular Filtration Rate [eGFR].The selection was as per rules of normal distribution law. Selection in both groups was independent & random.

Inclusion Criteria: Those with all the following were included.

- Age > 18 years
- CKD stages 4,5, 5D with GFR <30 ml/min for > 3 months and
- Either pathological abnormalities or markers of kidney damage i.e. abnormalities in blood, urine or imaging tests and
- Patients willing to give informed written consent.

Exclusion Criteria:

- On oral aluminium containing medications

All were evaluated with detailed history, complete systemic examination, Investigations- Hemoglobin (Hb), WBC, Blood Urea Nitrogen (BUN), Serum Creatinine, Serum Electrolytes including calcium, Serum Proteins, Lipid profile, Serum Aluminium, Serum intact PTH assay, Urine routine, Ultrasonography- Kidney, Urter & Bladder (USG-KUB), Electrocardiography (ECG), Two dimensional Echocardiography (2D Echo) and X ray chest.

Serum aluminium levels were measured by spectrophotometry. Levels were done in 32 CKD & 32 controls. Aluminum levels in municipal tap water & water post Reverse Osmosis treatment in our centre were measured.

Those with higher (> 60 µg/l) serum Al level were planned for DFO test &/or intense hemodialysis.

According to K-DOQI guidelines CKD was defined as:

- 1) Kidney damage for > 3 months with structural or functional abnormalities of the kidney, with or without decreased GFR, manifested by either pathological abnormalities or markers of kidney damage i.e. abnormalities in blood or urine investigation or imaging
- 2) Glomerular filtration rate of < 60 ml/min/1.73 m² for > 3months with or without kidney damage

GFR was estimated using MDRD equation:

MDRD equation: 186 X (sr Creat)^{-1.154} x age^{-0.203} x 0.742 (if female) X 1.210 (if black)

Stages CKD were defined according to Kidney Disease Outcomes Quality Initiatives (K-DOQI) guidelines:

- Stage 1: GFR ≥90ml/min
- Stage 2 : GFR 60-89 ml/min
- Stage 3 : GFR 30-59ml/min
- Stage 4 : GFR 15-29 ml/min
- Stage 5 : GFR <15ml/min (or dialysis : Stage 5 D)

Statistical Analysis

Data was analyzed using Mean, Standard Deviation and Test of Significance for quantitative data. P value <0.05 was considered to be significant.

Table 1: Clinical demography

	CKD Patients	Control
No of subjects	32	32
Age in years	18-60	18-60
Sex : M : F	2 : 1	2 : 1
eGFR	<15ml /min n= 27(CKD VD) n=01 (CKD V)	>90 ml/min
	15 to 29 ml/min n=04 (CKD IV)	
Sr. Al µg /l [Mean ± SD]	46.89 ± 83.62	2.8 ± 3.31
Sr. Al µg /l [Range]	6.6 -455.3	0.19-12.24

CKD: chronic kidney disease; eGFR: estimated glomerular filtration rate; Sr. Al: Serum aluminium

RESULTS

Clinical Demography

Sample size was 64:32 CKD patients & 32 controls. Both the subgroups were comparable [Table 1] as far as age & sex distribution concerned. Estimated GFR [eGFR] by Modification of Diet in Renal Disease [MDRD] formula in CKD group was < 30 ml/min: 27 patients were on maintenance hemodialysis (CKD

stage V D),1 was with eGFR < 15 ml/min but not on dialysis (CKD stage V), while 4 were in CKD stage IV [eGFR 15 to 29 ml/min].

None of the CKD patients were on maintenance hemodialysis at our centre. None had any neurologic features.

Serum Aluminum Levels

Aluminium levels in tap water & water post reverse osmosis treatment were < 5 µg/l.

Mean Serum Al levels in control group was $2.8 \pm 3.31 \mu\text{g/l}$ [range: 0.19-12.24] while in CKD patients the levels were significantly higher: $46.89 \pm 83.62 \mu\text{g/l}$ [range : 6.6-455.3] ($p < 0.001$) [Table 1]. Twenty six of the controls had serum aluminium levels less than 5 $\mu\text{g/l}$, six showed levels between 5 to 15 $\mu\text{g/l}$ while none had level higher than 15 $\mu\text{g/l}$. On the other hand, none of the CKD group subjects had serum aluminium level less than 5 $\mu\text{g/l}$, twenty three

had levels ranging between 5 to 40 $\mu\text{g/l}$, eight with levels 40 to 200 $\mu\text{g/l}$ while one showed level higher than 200 $\mu\text{g/l}$. [Figure 1] Of 23 from CKD group with levels between 5 to 40 $\mu\text{g/l}$, 19 were on MHD, while four were not on dialysis support. All 9 CKD patients with levels more than 40 $\mu\text{g/l}$ were on MHD. [Figure 1] Those with serum Al more than 60 $\mu\text{g/l}$ were advised intensive hemodialysis as they all declined desferrioxamine test.

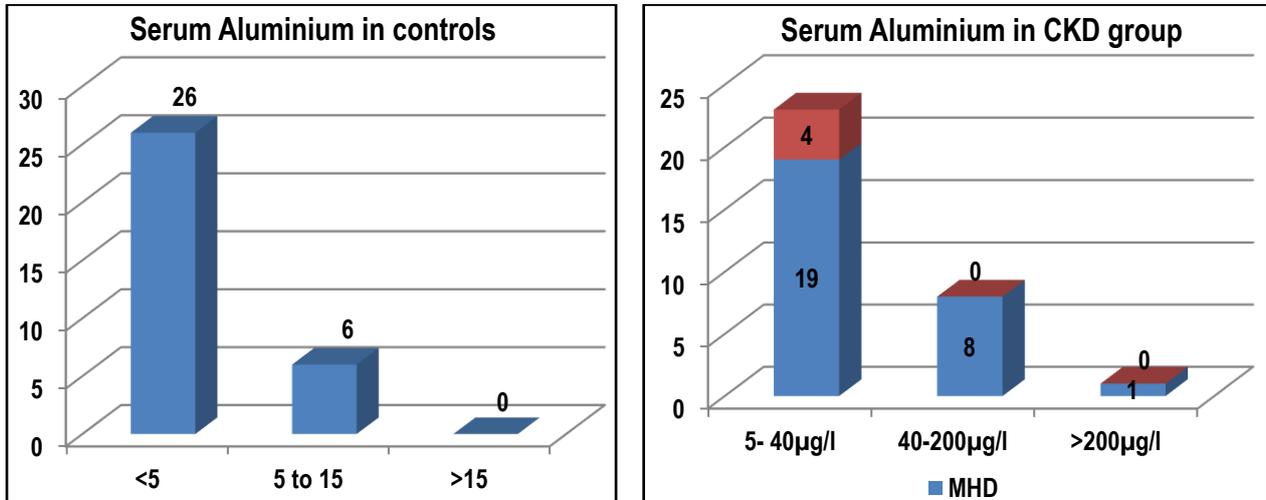


Figure 1: Serum aluminium levels in controls Vs CKD group. Most of the control group had serum aluminium level less than 5 $\mu\text{g/l}$ ($n=26$) while none of the CKD group had levels $<5 \mu\text{g/l}$. Of 23 from CKD group with levels between 5 to 40 $\mu\text{g/l}$, 19 were on MHD. All 9 CKD patients with levels more than 40 $\mu\text{g/l}$ were on MHD. The difference between two groups [control & CKD] was significant [$p < 0.001$]

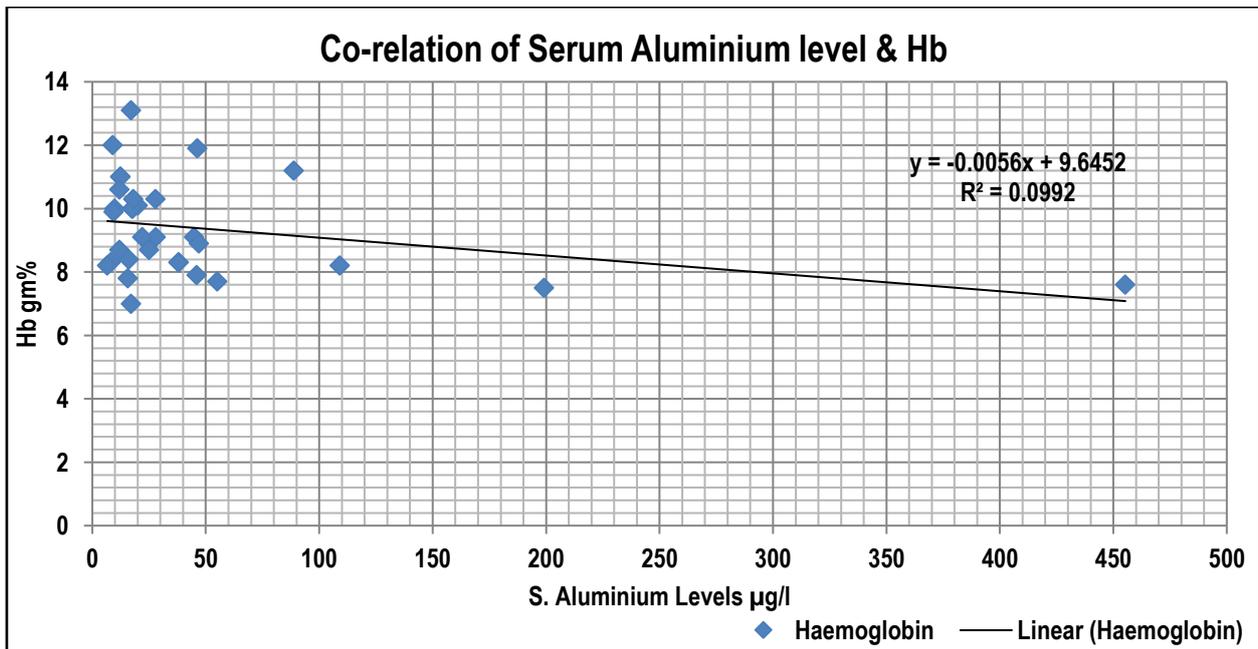


Figure 2: Significant co-relation between serum aluminium levels & Hemoglobin. ($r -0.32$ & $p-0.04$)

Anaemia & Serum Aluminium Levels

Mean Serum hemoglobin [Hb] levels in CKD groups with serum aluminium 5 to 40 $\mu\text{g/l}$ [$n=23$], 41 to 200 $\mu\text{g/l}$ [$n=8$] & $>200 \mu\text{g/l}$ [$n=1$] were 9.57 gm% [range: 7 to 13], 9.05 gm% [range: 8 to 12], & 7.6 gm% respectively. The co-relation between serum Al level and Hb is significant ($r -0.32$ & $p-0.04$). [Figure 2] Majority, 87.5% revealed normochromic normocytic anemia [Figure 3], while 3.13% showed microcytic hypochromic anemia. Approximately 21.82% had iron deficiency anemia while 78.12% had normal iron study. [Table 2]

Serum Calcium, Parathyroid Hormone Co-Relation With Serum Aluminium Levels

Mean Serum intact Parathyroid hormone [iPTH] levels in CKD groups with serum aluminium 5 to 40 $\mu\text{g/l}$ [$n=23$], 41 to 200 $\mu\text{g/l}$ [$n=8$] & $>200 \mu\text{g/l}$ [$n=1$] were 293.17 [range 13 to 1218] pg/ml, 384.7 [range 10 to 1173] pg/ml, 372 pg/ml respectively. The respective serum calcium values in the same groups were 7.96 mg% [range 6-9.3], 8.8 mg% [range 6-3 to 11], 7.7mg%. [Table 3] The co-relation between serum Al level and Calcium ($p- 0.21$), as well as iPTH ($p- 0.42$) were not significant.

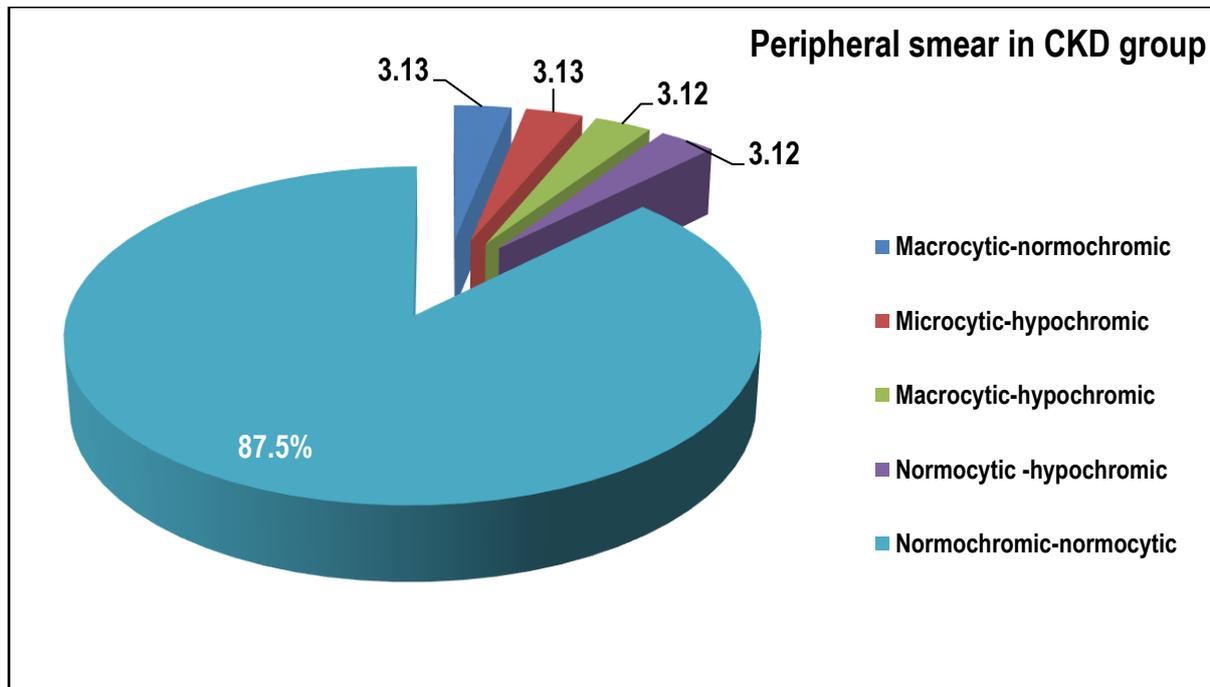


Figure 3: Peripheral smear in CKD group; Microcytic hypochromic anemia seen in 3.13 %

Table 2: Iron studies in CKD group

Variable		S. Aluminium (µg/l)		
		5-40 (n-23)	41-200(n-8)	>200 (n-1)
S. Ferritin (ng/ml)	Mean	866.43	1069.1	571
	Range	65 to 6037	138 to 4701	
S. Iron (µg/dl)	Mean	104.48	133.5	38
	Range	32 to 302	42 to 332	
TIBC (µg/dl)	Mean	228.13	265.0	216
	Range	109 to 484	170 to 396	
Transferrin Saturation%	Mean	45.26	47.25	18
	Range	17 to 99	17 to 87	

TIBC: Total iron binding capacity

Table 3: Co-relation between serum Al level and Calcium as well as iPTH

Variable		S. Aluminium (µg/l)		
		5-40 (n-23)	41-200(n-8)	>200 (n-1)
iPTH (pg/ml) [intact PTH]	Mean	293.17	384.7	372
	Range	13 to 1218	10 to 1173	
Serum Calcium (mg /dL)	Mean	7.96	8.8	7.7
	Range	6 to 9.3	6.3 to 11	

DISCUSSION

Serum levels in healthy individuals range from 1 to 3 µg/L.¹⁴ None of subjects from control group showed significantly higher level of serum aluminium (2.8 ± 3.31 µg/l). The level was less than 15 µg/L in all. However, the levels in CKD group [stage 4,5,5D] were significantly higher, 46.89 ± 83.62 µg/l [range: 6.6-455.3] (p< 0.001) [Table 1 & Figure 1].

As per KDOQI Clinical Practice Guidelines for Bone Metabolism and Disease with Chronic Kidney Disease: baseline levels of serum aluminum should be <20 µg/L & in all patients with

baseline serum aluminum levels >60 µg/L, a positive DFO test, or clinical symptoms consistent with aluminum toxicity, the source of aluminum should be identified and eliminated.¹⁵ Mean serum aluminum level in our CKD group was well above permissible level in CKD patients. Serum Al levels > 40 µg/l were seen only on those on maintenance hemodialysis.

Being a public health institute with massive workload of acute kidney injury, maintenance hemodialysis [MHD] is not our policy. All these 32 CKD patients were on MHD at outside centers, following up monthly in our Nephrology outpatient department. All

these centers had water treatment plant in their dialysis units. Though, we do not have chemical analysis of water of these individual centers, municipal water supply was common to all including our centre [Municipal corporation of Greater Mumbai] & serum aluminium level of our municipal water source was 0.30 µg/L. The dialysate Al level in our dialysate was 0.50 µg/L.

Aluminium intoxication was known from early 1960 to late 1980s. The source of intoxication was aluminium concentration in dialysate water as alum was used for municipal water purification¹⁶⁻²² or aluminium containing medications.^{23,24} With reverse osmosis membranes in water treatment plant, published guidelines of Association for Advancement of Medical Instrumentation for accepted aluminium levels in dialysate water²⁵ & use of non aluminium containing phosphate binders, aluminium toxicity is almost a non-entity. Thus, in spite of water treatment plant [with feed from municipal water] in all centers and none of the CKD group patients on aluminium containing phosphate binders, significantly high serum aluminium levels were observed. Various factors need to be considered to explain this high serum aluminium level.

1. Sources within the distribution system of water for dialysis in dialysis centre
2. Sources outside dialysis
3. Inadequate dialysis

Aluminium contamination due to materials in water treatment plant & dialysate delivery system like aluminium pump, water pipes can be a source but with stainless steel pipes routinely used in all centers & guidelines for hemodialysis unit by Indian Society of Nephrology followed by most of the centers, use of reverse osmosis and/or deionizers regular chemical analysis of R.O. water, this source appears less likely.²⁶

It is important to note that aluminium is now added to or used in almost everything we eat, drink, inject or absorb. Apart from being the most abundant in the Earth's crust & naturally absorbed from the soil by plants and foodstuffs, it is present in food additives, vaccines, antiperspirants, sunscreens.²⁷ This assumes importance as renal function decreases significantly, ie eGFR < 30ml/min.

Increasing workload of end stage kidney disease in India²⁸, have forced many centers to offer MHD for 8 hrs instead of 12 hours per week. This may further add up to high serum aluminium level due to inadequate HD.

Thus, high levels of serum aluminium in our CKD group appears to be multifactorial.

As per NKF KDOQI guidelines, in children with CKD Stage 5, to assess aluminum exposure and the risk of aluminum toxicity, serum aluminum levels should be measured at least yearly and every 3 months in those receiving aluminum-containing medications.²⁹ Serum aluminium levels, in today's practice are not done regularly. With the finding of significantly high levels of serum aluminium in our pilot study, it may be prudent to check levels yearly, more so if erythropoietin resistant anemia, unexplained neurologic features or a dynamic bone disease are evident.

In our study group, co-relation between anemia & serum aluminium was significant. However, only 3.13% showed microcytic anemia, while aluminium induced anemia is iron resistant microcytic anemia.³⁰ This may be because, majority of the CKD patients had less than toxic level {< 60}, though significantly high.

Association of aluminium levels with serum calcium & iPTH was not significant. Also, none of the patient had neurologic features.

LIMITATIONS

It is pilot study with small sample size. Ideally, R.O. water analysis of every centre should have been known.

CONCLUSION

Significantly high serum aluminium levels, in CKD stage 4,5,5D in spite of these limitations, cannot be ignored. Aluminium toxicity as cause of EPO resistant anemia is not considered in today's scenario of R.O water & non aluminium containing phosphate binders. Regular serum aluminum levels yearly should be considered in stage IV & V, VD CKD. Source of aluminium leading to significant high levels needs to be looked into.

SOURCE OF SUPPORT

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Conflict of Interest: None Declared.

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