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Research Article

Hospital acquired acute renal failure

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ABSTRACT

Background: Acute renal failure has continued to attract interest and stimulate investigators .This is in part, a reflection of many clinical entities that can result in an acute renal failure. HAARF is an important cause of morbidity and mortality and is associated with the ten fold increase in the risk of death during the hospitalization.[14] Despite advances in diagnosis and management it still carries a high mortality. HAARF is associated with grave consequences. Some of the clinical setting leading to HAARF can be limited by monitoring of renal function, better control of infection, avoiding nephrotoxic drugs and initiation of therapy at the earliest. Present study is proposed to evaluate the incidence, etiological factors and measures to reduce the incidence of HAARF.
Methods: All patients were admitted to RPGMC from Dec. 2010 to APRIL 2014. They were screened for the development of the ARF during their hospital stay. The parameters of Prakash et al. were taken for the diagnosis of HAARF.
Results: In our present study HAARF was diagnosed in 88 patients of 56904 admission during 40 month period, representing 0.15% of the admission. Predisposing factors were present in 64 % of the patients. They were elderly age >60 years (22.72%), DM (13.64%), pre-existing renal disease (13.64%) and HTN (4.54%). It was due to nephrotoxic dugs in 45.45%, decreased renal perfusion in 22.72%, infections in 13.64%, hepatorenal syndrome 9.09% surgery in 4.54% and Weil's disease in 4.54 %. Among the nephrotoxic drugs antibiotics were the most common and NSAIDs were the second most common etiological factors. Decrease renal perfusion secondary to volume depletion and heart failure were equally responsible for HAARF in 9.09% each. Septicemia was responsible for 4.54% of cases. Oliguric renal failure was seen in the 31.82% where as nonoliguric renal failure was seen in 50% of cases. Great majority of non oliguric renal failure was due to nephrotoxic drugs. Oliguric patients have longer duration of hospital stay and high mortality as compared to the non oliguric renal failure. A high s. creatinine and high urea levels at the time of admission were associated with the earlier development of HAARF. Oliguria and anuric patients had a longer duration of hospital stay. Overall mortality of HAARF was 18.18% and nephrotoxic drugs responsible for one half of the total mortality.
Conclusion: High risk group patients for HAARF needs meticulous monitoring during hospital stay. Hospitalized patients on nephrotoxic drugs should have frequent renal function tests. Proper fluid and electrolyte balance in hospitalized patients needs special emphasis to avoid HAARF.

Keywords: Acute renal failure

INTRODUCTION

Acute renal failure by definition is deterioration of renal functions over a period of hours to days, resulting in failure of kidneys to excrete nitrogenous waste products and to maintain fluid and electrolyte homeostasis. Acute renal failure developing during the hospital stay is termed as Hospital Acquired Acute Renal Failure (HAARF)[2] and

the one acquired outside the hospital is termed as community acquired ARF.[3] The frequency of ARF among patients is 1 % at the time of admission to the hospital and 2-5% during hospitalization. ARF can result from decrease renal perfusion with out cellular injury; and ischemic toxic and obstructive insult to the renal tubule; a tubulointerstitial process with inflammation and edama; or primary reduction in the filtering capacity of the glomerulus .Accordingly ARF has been classified as prerenal, renal and post renal. In case of prerenal azotemia, the renal clearance is limited by renal perfusion while renal tubular and glomerular functions are intact. Renal dysfunction related to obstruction of urinary out flow tract is termed as post renal azotemia and ARF due to primary intra renal cause is called as renal azotemia. Prerenal azotemia accounts for 70% of community acquired[3] and 40% of hospital acquired cause of ARF.[4] Among hospitalize patient prerenal azotemia is often due to cardiac failure liver dysfunction or septic shock.[1] Sustained prerenal azotemia predisposes to ischemic necrosis, and is the most frequent cause of hospital acquired ARF.

The common etiological factors for HAARF are, use of nephrotoxic drugs, decreased renal perfusion, surgery and septicaemia. They are responsible for 79-80% of HAARF.[6] Post-operative ARF[4] is second most common cause of HAARF and the drug induced renal insufficiency is a major cause of iatrogenic renal insufficiency. The nephrotoxic drugs[7] are incremented in some 23.9% of cases of hospital acquired ARF. ARF in hospitalized patents is often, due to more than one cause. The frequent combination of causes includes exposure to aminoglycoside with sepsis,[8] administration of radio contrast agents in patients receiving ace inhibitors[9] and NSAIDS[10] in the presence of congestive heart failure. ARF, according to urine output has been classified as oliguric (uo <400 ml) per day and non oliguric (uo ≥600 ml/day).[11] Although oliguria was considered the cardinal feature of ARF, non oiguric ARF has been rising since 1943 and accounts for 20-30% of ARF. Non oliguria ARF occurs more frequently, than it generally is recognised and causes lees morbidity and mortality than oliguria ARF. The clinical picture of ARF in hospitalized patients[12] is slightly different from the community acquired ARF, hospitalized patients are on various drugs like dopamine, diuretics and nephrotoxic drugs. They all contribute to the development of non oliguric renal failure and it obscures the substantial underlying ARF.

Advanced age, DM, HTN, pre-existing kidney and liver disease have been recognised as the important risk factor in hospital acquired ARF. Volume depletion, use of aminoglycosides, congestive heart failure. Radio contrast agent exposure and septic shock are otter important factor for development of the HAARF.[14] The poor prognostic indicators of HAARF,[4] are oliguria, active urinary sediments and a serum creatinine of 3 mg/dl or more.

METHODS

All patients were admitted to RPGMC from Dec. 2010 to APRIL 2014. They were screened for the development of the ARF during their hospital stay. The parameters of Prakash et al. were taken for the diagnosis of HAARF. These are: an increase in s. Cr. level above 2 mg/dl in whom the base line serum cr. less than 1.5 mg/dl.

Exclusion criteria

1. Patients having ARF at the admission
2. Patients taking nephrotoxic drugs with in three days prior to admission
3. Patient not willing to participate in the study

The patients fulfilling the above criteria were included in the study. First value serum creatinine was taken as base line value.

These patient were explained in detail about the purpose of the study and informed consent was taken.A detailed history physical examination and lab. Investigation were made as the performa. All these patients were followed daily until discharged death or return of renal function to the base line levels. A serial record of urine output and S. cr. were maintained. Each patient was assessed for the complication of ARF like hyperkalemia neurological abnormalities pulmonary. edema, GI bleeding, pericarditis and infection. HD and or PD were instituted as when required. Renal failure was incriminated as the cause of death if patient exhibited evidence of severe uremia, hyperkalemia or volume overload secondary to oliguria. Patients were classified as oliguric (uo, 400 ml/dl) or non oliguric uo >400 ml/day during the azotemic phase. To establish the cause of HAARF clinical criteria of Hou et al.[4] were used. The hospital charts of the case subjects were reviewed to identify demographic data admission and discharge diagnosis. Risk factors were identified during a specific assessment period. This was defined as period of time from the baseline S. cr. determination until the date when the serum cr. rose to the levels qualifying for inclusion in the study. The risk factors studied were intravascular volume depletion, CHF, shock due to sepsis, myocardial failure, radiocontrast agents exposure, aminoglycosides use, NSAID use, intraoperative hypotension, pre-existing renal and liver disease, HTN and DM. Length of the study and discharged status were recorded as outcome variable. The influence of factors such as presence or absence of oliguria S. cr. at the time of admission, age of the patient, and the presence or absence of multiorgan failure as the cause and prognosis of the ARF was analysed.

RESULTS

The present study was conducted in dept. of medicine at RPGMCH. A total of 58666 patients were admitted in RPGMCH in more than 3 years and were screened prospectively through out there stay in the hospital for the development of ARF. Between Dec. 2010 and April 2014 88 patients satisfied the criteria for inclusion in the study and thus HAARF representing 0.15% of all hospital admission. Out of 88 patients of 54 were males and 32 were females. Age of HAARF patients varied from 22 years to 90 years with a mean of 44.5 ± 19 years. 32 patients (36.36%) were between 21 years to 30 years of age, 20 (22.73%) patients were more than 60 years of age, 16 (18.81%) patients were between 51 to 60 years, 12 patients (13.64%) were between 31 and 40 years of age and 8 patient (9.09%) were from 41 and 50 years of age.in male patients age ranged from 22 to 90 years with a mean of 49.8 years. In female patients' age ranged from 23 years to 59 years with a mean of 35.1 years of age.

At the time of admission the clinical diagnosis of these patients was, DM 12 patients (13.64%), renal calculus 12 patients (13.64%), ALD 8 patients (9.09%), diarrhoea in 8 patients (9.09%), septic abortion in 8 patients (9.09%), hypertension, fulminant hepatic failure, chronic duodenal ulcer, Ca stomach, chronic obstructive air way disease, septicemia, disseminated tuberculosis, tachyarrhythmia, fracture Lt trochanter and unknown poisoning in 4 patients (4.54%) each. Out of the 12 patients of DM, 4 patients had T2 DM with mononeuritis multiplex with autonomic neuropathy, 4 had T2DM with neuropathy and nephropathy and 4 had T2DM with neuropathy with CVA (right sided hemiplegia) and aspiration pneumonia. 8 patients of renal calculus had uretic stone 4 had calculus in renal parenchyma. 4 patients of ALD had cirrhosis and 4 patients had hepatitis with prehepatic coma.

4 patients of diarrhoea had chronic diarrhoea and 4 had acute diarrhoea. The patients of chronic duodenal ulcer had GOO. Patients of fulminant hepatic failure had upper GI bleed. COPD patients had bronchiectasis. The patients of Ca stomach had secondries in the liver. Patients with tachyarrhythmia had paroxysmal SVT with aberrant conduction and patient of fracture trochanter was operated. Patients 60 (68.18%) were from medicine and allied, 20 patients (22.72%) were from surgery and allied while 8 patients (9.09%) were from gyne and obstructive. Out of 60 patients 48 patients (63.63%) were from general medicine and 4 patients (4.54%) were from cardiology. Out of 20 patients 12 patients were from general surgery (13.63%), 4 patients (4.54%) from orthopaedics and 4 patients (4.54%) from urology. The duration of hospital stay for the development of HAARF (time duration between admission and development of HAARF) varied between 3 days to 50 days with a mean of 12.95 ± 11.75 days. 36 patients (40.96%) developed it in first 20 days of the admission, 16 patients (18.18%) developed it between 6^{th} to 10^{th} day and 16 (18.18%) between 11^{th} to 25^{th} day. 4 patients each (4.54%) developed it between 16^{th} to 20^{th}, 21^{st} to 25^{th} and 26^{th} to 30^{th} day of admission. 58 patients (77.26%) developed acute renal failure in first 15 days of hospital admission. 8 patients (9.09%) developed ARF in more than 30 days of hospital stay. 4 on 3^{rd} day and 4 on 15^{th} day of admission.

Etiological factors for HAARF

Out of 88 patients of HAARF, 20 patients (22.72%) had decreased renal perfusion. Out of which 8 patients (40%) had decreased renal perfusion due to volume depletion secondary to dehydration, 8 patients (40%) had decreased renal perfusion due to heart failure, 4 patients (20%) had decreased renal perfusion due to shock of unknown poisoning. In 88 patients of HAARF, 76 (83.36%) were on nephrotoxic drugs, 40 patients (45.45%) were taking single nephrotoxic drug while 36 were (46.90%) on more than one nephrotoxic drug.

DISCUSSION

This prospective study was aimed at evaluating HAARF with special reference to the incidence, causative factors, clinical course, morbidity, mortality and it impact on the patient management.

In the present study, hospital acquired ARF complicated the course of 88 patients of 56904 admissions (0.15%). Hou et al.[4] reported an incidence of 4.9% of it from the New England Medical Centre, Boston. Shusterman et al.[14] reported it as 2% from Pennsylvania (USA) whereas Prakash et al.[1] reported it as 0.7% from BHU Varanasi and Jha et al.[6] as 0.64% from PGIMER Chandigarh. The lower incidence in our study is multifactorial. During the study period (Dec. 2010 to April 2014) no open heart surgery was done and it is an imp cause of HAARF. All admitted patients did not have their RFT done routinely so a few pts might have been over. In the present study the male to female ratio was 1.7:1, and is comparable with 1.2:1 ratio of Prakash et al. Age range of these pts varied from 21 t0 90 years with mean age of 44.5 plus minus 19 years. It was 43.6 years in the study of Prakash et al.[13] and 42.9 years in the study of Jha et al.[6] and is comparable. In this study 68.18% of the pts were from medicine and allied specialities, 22.72% were from surgery and allied specialities and 9.09% were from gyne. Shusterman et al.[14] noted that 52.9% pts were from the medical 44.1% from the surgical and 2.9% from the gyne side. The largest no. of pts in present study was from medicine and allied specialities and is comparable to the study of Shusterman et al. However the percentage figure differs because in our institution open heart and pancreatic surgery were not done during the study period.

Predisposing factors for the development of HAARF in these pts were: Elderly (age >60 years) - 20 pts (22.72%), - 12 pts (13.64%), pre-existing renal disease - 12 pts (13.64%), pre-existing liver disease - 8 pts (9.09%) and

HT - 4 pts (4.54%). Thus 64% pts had one or another predisposing risk factor whereas these factors were noted in 54% of pts.[13]

The duration of hospital stay for the development of HAARF in this study varied between 3 to 50 days with a mean of 12.95 plus minus 11.75 days. The available studies[4,6,7,13,14] are silent on this aspect. It is further discussed with biochemical parameter. The etiological factors of it were, nephrotoxic drugs in 76 pts (86.36%), decreased renal perfusion in 20 pts (22.72%), surgery in 12 pts (13.63%), infection in 12 pts (13.64%), hepatorenal syndrome in 8 pts (9.09%) and unconfirmed Weil's disease in 4 pts (4.54%). Among the nephrotoxic drug group, in 40 pts (45.45%) it was the direct effect of nephrotoxic drugs where as in the remaining 36 pts (40.90%) nephrotoxic drugs were contributory factors.in 32 pts (36.36%) antibiotics were responsible for it either alone or in combination. In 8 pts (9.09%) NSAIDS's were responsible in combination with antibiotics. There was no pt. of contrast induced HAARF. The nephrotoxic drugs constituted the largest group and antibiotics dominated it and NSAIDS closely followed it. It is comparable with other studies.[6,13]

Decreased renal perfusion accounted for HAART in 20 pts (22.72%). It was due to volume contraction in 8 pts (40%), cardiac dysfunction including heart failure in 8 pts (40%) and shock in 4 pts (20%). Jha et al. reported HAARF due to renal hypoperfusion in 27% and Prakash et al. reported it in 30.5% and is comparable. However, a slightly higher figure of 42% was reported by Hou et al. from Boston. Post-operative renal insufficiency was observed in 12 pts (13.63%). In 4 pt surgery was the major culprit whereas in the remaining 8 surgery was a contributing factor. Jha et al observed it as 18%, Prakash et al. as 13.4% and Hou et al. as 10.8%. The lower incidence in this study is because of not doing open heart and pancreatic surgery, which is most likely to develop HAARF postoperatively. In the present study infections accounted for HAARF in 12 pts (13.63%). In 4 pts it was due to septicaemia, in another 4 it was due to septic abortion and in the rest 4 it was due to aspiration pneumonia. Jha et al. noted septicaemia in 17%, Prakash et al. in 14.65 and Prashar et al. in 21.95%. A higher incidence of it due to infection in above studies is probably due to the admission of more pt with infections as is evident from the fact, that there were only 4 pts of septicaemia in our study whereas this no. varied from 18 to 33 in the above studies. Hepatorenal syndrome accounted for 9.09% patients in our study and is comparable with other studies.[4,6]

One of our patients had clinical profile compatible with Weil's disease but confirmatory tests could not be undertaken. There was no patients of Weil's disease in the available studies.[4,6,13,20] However Weil's disease is a known cause of acute renal failure and it occurs in 44.67% of all cases 47. In true sense renal insufficiency was a part of the spectrum of the diseases, but according to our definition it falls in the category of HAARF. At the time of admission 64 (63.63%) patients were with normal renal function (S. cr. 1.5%) in our study and is comparable time of admission with other studies 6 the present study showed the temporal relation between S. cr. value at the time of admission and the day of development of HAARF. Out of 88 patients 32 patients (36.4%) had a serum creatinine of more than 2 mg% at the time of admission and they developed the HAARF with in first 10 days of admission whereas out of 56 patients who had S. cr. value less than 1.5 mg% at the time of admission, 40 patients developed HAARF in more than 10 days of admission to hospital. It is statistically significant (P value <0.05).thus higher the S. cr. level at the time of admission shorter the duration of development of HAARF.

The serum creatinine value at the time of admission was compared to with the duration of hospital stay of HAARF. It was evident from the study that a low value of serum creatinine at the time of admission was associated with shorter duration of hospital staying HAARF, though it was not statistically significant. Serum cr. value at the time of admission was compared to the urinary output at the time of detection of HAARF (table).[20] 12 out of 32 patients who had admission cr. value of more than 2 mg% had anuric renal failure, 12 had oliguric renal failure and 8 had non oliguric renal failure. Out of 56 patients who had serum cr. less than 1.5 mg% at the time of admission, 4 had anuric renal failure 16 had oliguric renal failure 36 had non oliguric renal failure. It is statistically significant (P value <0.05). Thus a high serum cr. value at the time of admission was more likely to have oliguric and anuric renal failure.

Serum cr. value at the time of detection of HAARF was compared with duration of hospital stay (table).[22] 8 patients had serum cr. value less than 2 mg% at the time of detection of HAARF and in both these patients hospital stay was less than 5 days. 32 patients had had serum creatinine value between 2 and 3 mg% at the time of admission. Hospital stay was less than 5 days in 8, and more than 5 days in 24. 48 patients serum cr. value more than 3 mg%, 28 had a hospital stay of less than 5 days and 20 had hospital stay of more than 5 days. Though study showed temporal relation between serum creatinine value at the time of detection of HAARF with duration of hospital with duration of hospital stay, but not statistically significant (P value <0.05). The serum creatinine value at the detection of HAARF was compared with the mortality. Out of 48 patients who had serum creatinine value of more than 3 mg% at the time of detection of HAARF, 8 died whereas out of 40 patients of serum creatinine value of less than 3 mg% only 8 patients died. It was statistically significant (P value <0.05). In this study of 88 patients of HAARF showed a temporal relation of blood urea level with development of HAARF and is depicted in table 25. All the 40 patients having blood urea of more than 40 mg% developed HAARF in the first 10 days. Out of the remaining 48 patients with

urea level of less than 40 mg% only 8 patients developed in first 10 days .though it was not statistically significant.

The blood urea level on admission was compared with urine output of HAARF (table).[23] There were 48 patients with blood urea level of <40 mg%, 36 out of these developed non oliguric renal failure ,8 developed oliguric renal failure and 4 developed anuric renal failure. In the remaining 40 patients with a blood urea level of more than 40 mg% at the time of admission, 8 patients had non oliguric renal failure, 20 had nonoliguric renal failure and 12 had anuric renal failure. It was statistically significant (P value <0.05) thus high blood urea level at the time of admission was more commonly associated with oliguric or anuric renal failure. In the present study 44 patients (50%) were non oliguric, 28 patient (31.82%) were oliguric and 16 patients (18.18%) were anuric, were as Jha et al. noted that 52% were non oliguric and 42% were oliguric, Prakash et al. observed 47.6% non oliguric and 52.4% oliguric and Prasshar et al. noted 39.2% of non oliguric which are comparable whereas Hou et al. reported 80% non oliguric and 20% oliguric. The difference in percentage figure is due to probably that majority of our patients it was due to nephrotoxic drugs which are known to produce non oliguric renal failure. Hospital stay was compared with urine output in oliguric and non oliguric groups. In the oliguric group there were 28 patients .duration of hospital stay was less than 10 days in 12 patients and more than 10 days in 12 patients. In non oliguric group here were 44 patients, 32 patients had hospital stay of less than 10 days and 12 had a stay of more than 10 days. In the anuric group there were 12 patients and all had a stay of less than 10 days. Apparently it was suggested that oliguric patient has longer duration of stay as compared to non oliguric patients but statistically it was not significant (P <0.05).

Mortality of oliguric and non oliguric group was compared. Oliguria was seen in 28 patients and 8 out of 7 died (30%). Non oliguic renal failure was present in 44 patients and 4 out of it died (9%). Anuria was detected in 16 patients and 4 out of it died (25%). Oliguic renal failure had a mortality more than non oliguic renal failure and is comparable with Hou et al. of 52% and 17% respectively and Jha et al. of 60% and 24% respectively. However the percent figure differ due to less number of patients in our study. The electrolyte abnormality was corrected during the hospital stay and they did not influence the outcome of HAARF. In none of the 16 patients of HAARF, who died in the hospital , the cause of death was attributable to electrolyte abnormality and none of our patients required dialysis 52 (59.09%) were of normal kidney size on ultrasonography while 12 patients (13.63%) were of large kidney (>11 cm) and 8 patients (9.09%) were of small kidney (<9 cm). 8 patients had large kidney on USG 4 had pregnancy and 4 had Weil's disease. 4 patients with small kidney had chronic renal failure whereas 4 had had septic abortion. The available studies[4,6,13,20] did not comment upon kidney size in HAARF. Overall mortality of HAARF in this study was 18.18%. 8 were from the nephrotoxic drug group, 4 from the hypovolemic group and 4 from the hepatorenal group. This is comparable with the study of Prakash et al. Other studies[6,20] have noted a higher mortality ranging from 32 to 41%. The lower mortality figures in this study were due to the absence of high mortality HAARF e.g. cardiac surgery, pancreatic surgery etc. Overall mortality of HAARF was 18.18% and nephrotoxic drugs responsible for one half of total mortality.

High risk group patients for HAARF needs a meticulous monitoring during hospital stay.

Hospitalized patients on nephrotoxic drugs should have frequent renal function tests. Proper fluid and electrolyte balance in hospitalized patients needs special emphasis to avoid HAARF.

CONCLUSION

In our present study HAARF was diagnosed in 88 patients of 56904 admission during 40 month period, representing 0.15% of the admission. Predisposing factors were present in 64 % of the patients. They were elderly age >60 years (22.72%), DM (13.64%), pre-existing renal disease (13.64%) and HTN (4.54%). It was due to nephrotoxic dugs in 45.45%, decreased renal perfusion in 22.72%, infections in 13.64%, hepatorenal syndrome 9.09% surgery in 4.54% and Weil's disease in 4.54 %. Among the nephrotoxic drugs antibiotics were the most common and NSAIDs were the second most common etiological factors. Decrease renal perfusion secondary to volume depletion and heart failure were equally responsible for HAARF in 9.09% each. Septicemia was responsible for 4.54% of cases. Oliguric renal failure was seen in the 31.82% whereas nonoliguric renal failure was seen in 50% of cases.

Great majority of non oliguric renal failure was due to nephrotoxic drugs. Oliguric patients have longer duration of hospital stay and high mortality as compared to the non oliguric renal failure. A high s. creatinine and high urea levels at the time of admission were associated with the earlier development of HAARF. Oliguria and anuric patients had a longer duration of hospital stay. Overall mortality of HAARF was 18.18% and nephrotoxic drugs responsible for one half of the total mortality.

High risk group patients for HAARF needs meticulous monitoring during hospital stay. Hospitalized patients on nephrotoxic drugs should have frequent renal function tests. Proper fluid and electrolyte balance in hospitalized patients needs special emphasis to avoid HAARF.

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