A Retrospective Analysis Of Nephrogenic Systemic Fibrosis In A Population Undergoing Renal Magnetic Resonance Angiography Stratified By eGFR

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Introduction:

Nephrogenic Systemic Fibrosis (NSF) is a rare, debilitating condition that associates with the administration of gadolinium-based contrast agents (GBCA) used for magnetic resonance angiography (MRA). Only individuals with renal impairment develop NSF. The incidence in patients with end stage renal failure exposed to the highest risk GBCA (gadodiamide) is 3-7%. European Guidelines contraindicate the use of high risk GBCAs in AKI and CKD < 30ml/min and advise caution for those with eGFR < 60ml/min. American guidelines suggest a class ban on GBCAs. We considered a population with varying eGFR exposed to renal artery MRA to determine the distribution of NSF with reference to renal function.

Methods:

A list of all patients who underwent renal MRA in Glasgow was obtained from radiology records. Those with renal unit electronic patient records (EPR), follow-up for at least 90 days post imaging, an eGFR measurement within 90 days of MRA and documented follow up within 90 days of MRA were included. Information on diagnosis, in patient and out patient follow ups, outcome and the presence of NSF was determined. NSF was diagnosed after review of diagnosis, clinical history, timeline and correspondence screens within the EPR with cross reference to the pathology database.

Results:

1551 people under went renal MRA with 30 ml gadodiamide between the start of 1998 and the end of 2005. Of these 481 met the inclusion criteria. The spread of eGFR is shown below.

CKD stage	eGFR(ml/min)	number	%
5	<15	69	14.6
4	15-29.9	168	35.5
3b	30-44.9	144	30.4
3a	45-59.9	52	11.0
1/2	60+	40	8.5

Eight were identified as having acute kidney injury at the time of imaging and not included in CKD staging. 38.1% had renovascular disease and 2.1% were potential live kidney donors. The median time from MRA to follow up was 20 days with a median of 3.7 years follow up. The median number of out patient follow ups with correspondence was 12. At the end of the study period 35.6% were dead, 24.9% discharged or transferred, 33.3% continued as out patients and 6.2% were receiving renal replacement therapy. 3 patients were identified as having NSF. 2 had AKI at the time of imaging and NSF developed following renal MRA. The third had deteriorating CKD with an eGFR of 16.1ml/min at the time of renal MRA but developed NSF three years later following a second MRA with 30 ml gadodiamide. Peritoneal dialysis was established for 16 months prior to the second scan. All three were alive at the end of the study period. All cases of gododiamide-associated NSF from our unit (n=16) were reviewed with respect to eGFR at the time of MRA preceding diagnosis. 13 were established on dialysis, 2 had AKI and one had an eGFR of 8.3 ml/min.

Conclusions:

These findings suggest that individuals at greatest risk of developing NSF have AKI or stage 5 CKD at the time of gadodiamide exposure. We identified no NSF cases linked to GBCA use with an eGFR >15 ml/min despite looking at a population with a wide range of eGFR.