Discussion
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The first point to be noted is that out of 51 males of an infertile couple investigated 23 males (45.8%) showed abnormal sperm count. Thus 45.8% males of infertile union is our study showed abnormal semen as a cause of infertility. Data from investigation of the female by co-investigator shows that in our study of 23 couples in 12 couples (23.52%) some depot was also found in the female partner. So a male cause only was found in 11 (21.56%) of all the cases.

These data can be compared with data available from other places. The W.H.O. study of the standardised investigation of the infertile couple has done comprehensive study and collected data from all over the world about the factor involved in infertility.

In developed countries the male factor is found in 33% case; A male factor only was found in 22% cases while both partners of a couple were found have a defect in, 21% of couple. Data from Mumbai & Kashmir the male factor in 18% & 22% respectively.

Thus in our study abnormality was found in comparitively in high% of case 45.8% campared to 33%, 18% and 22% in developed country, Mumbai and Kashmir respectively. Also in our study both Partner were involved in 23.5% cases with is relatively equal in 21%, 26% in developed country and Mumbai respectively. Our figure resemblence most closely to data from Carolina (Africa) which found both partner involved in 50% of cases.

The reason for this differnce can lie basically in the quality of population studies, Bundelkhand region is specially recognised as a very backword region both economicaly and socialy and
this is projected in high prevalence of infection diseases specialy T.B. etc. The nutrition in this region is also very poor as is reflected by a greater incidence of malnutrition and anemia. Thus infection and malnutrition mean that larger portion of population is diseased, which is reflected in our data by large proportion of person of both sexes affected by disease. Where age parameter are analysed it is seen that maximum number of patients seeked medical attention in the age group of 26-30 and between 2-7 years of marriage, the age group of 26-30 years is also the age of maximum reproductive capacity it is also seen that mean age of person entering our study was 28.6±3.2 years which seems rather high when it is taken into account the trend of marriage at early age in India specialy in poor clears; (must of our cases were from low S.E. status). This may reflect the tendency of couples specialy males to prevent themselves for investigation quite late because of hesitation taboos and a misconception that female is mostly responsible for infertility.

The majority of patients were of primary infertility (80%), WHO has give this figure as 84% for Asia and 79.3% for developed country. Thses data differ a lot from Africa. Where primary infertility forms only 40.8% of total infertile couples.

The mean age of primary infertility patients was much younger at 27.5±2.7 years when compared to mean age of patient with secondary infertility which was 34.11±.84 years. Thus couple with seconary infertility tried to have a baby for longer time before presenting themself for investigation.

The how incidence of secondary infertility in our study may not reflect its actual incidence. Couples with one child who subsequently because secondary infertile are were to adopt to this situation with not seek medical attention then a couple who
never has concived. A survey report by WHO showed that only
11% of seconary infertile women were intrested in therapy
compared to 40% of primary group. For further discussion we
will put all the investigation in a proper propective.

In our semen analysis the major abnormality was
algooastheozoosperma in 35.29% of our case farming 78.2% of
all semen abnormalities. Next in line was azoospermia in 9.8% of
our case and responsible for 21.73% of all semen abnormalities.

In our study we have found that low count an poor motility	ravel almost in parallel. Thus count and motality may be
reflecter of estimates of the same thing.

Divergence of these 2 parameters may indicate a specefic
defect. In our study it was maturation arrest of spernatids in the
testis in two case showing divergent trands and one case show
testicular atrophy.

In our study we did'nt find any patient siginificently abnormal
sperm morphology. WHO in its world wild study gave this figure
to about 0.6% we could not find any case of abnormal sperm
morpology probably because of our small study size. This
incidence of abnormal sperm morphology is much less frequently
diagnosed then in the past this is because of much better
understanding of normal variation in sperm morphology. Gordon
et al 1965 have shown that normal spermotozo vary typical for
most biological data.

A very high incidence of pus cells were found in semen in
our study (25.4%) which may reflect the increased prevenlenece of
genital infection in this region, pus cells is good indicator of
genital infection as a cause of infertility as expressed by the
fact that patient with singnificant puse cell in semen had
abnormal semen quality.
A very important aspect of male investigation is finding out of the etiology of infertility which is different from just finding out that a particular infertile male has abnormal semen quality. As mentioned by us in material methods the WHO has laid standard guidelines for the standerized evolution of the infertile couple with recommended that our infertile male may be provided with 2 diagnosis (I) the disruptive diagnosis which has been death with carlier. (II) etiological diagnosis.

We have found idopathic testicular failure (maturation arrest) in 2 out of 51 cases. (3.98%) It is similar to world wild data provided by WHO, which published a figure of 10% for developed countries 7% of Africa and 8% for Mumbai city in India.

Our study also provided for evolution of obstructive azoospermia. We have estimated that at least 2 out of 5 cases. WHO has given a 1% for world average with Africa 4.2%.

However our results in this aspect have to be interpreted with caution. The diagnosis of obstructive azoosperima is not easy. Conhaire et. al. state that if testicular volume and FHS is normal testicular Biopsy is needed to reach a confirmed diagnosis. Testicular Biopsy was not done in all our cases to confirm the diagnosis and only an estimate was done, but Joel C.A. has also state that if an patient with complete azoospermia the cause is an obstruction somewher in deferent ductules. It is rarely but our find completely normal spermatogenesis in biopsy. The much more specific methods for detecting obsetuction in male genital tract like differento vesiculography, transrectal ultrasonography were not performed in our study.

However, all our cases suspected of having obstruction had low to low normal semen volume. To (4%) of our study satisfy the WHO criteria for diagnosis of male aessary gland infection.
One case was estimated to be having obstructed pathology also. So this group was not exclusive confirmed study was found in (4%) cases and our other case had significant history of suggestive study. Since these group are overlapping it would be important to consider total S.T.D. related causes which would includes cofirem S.T.D. Many cases of obstructed azoospermia are caused by S.T.D. Thus 13 cases (25.4%) of cases in our study had S.T.D. related causes for their infertility 1 case had history of mumps in childhood and azoospermia.

Our results thus reflects a heavy load of infection as a factor for male infertility this is not surprizing as a incidence of T.B. and other infection is much higher in this region.

we could'nt find any cause of varicocele related infertility and congenital causes. Varicocele has been considered a very important cause of male factor infertility in studies worldwide ranging from 5.6% to 11% in Mumbai and developed countries respectively. The resion for these difference could be either a true lower incidence of varicocele in our countary or lower index of suspicion for their easily treatable cause of infertility and small size of study.

Lastly the most prepelexing part of etiology of male infertility were cases in which new etiological factor whatsoever could be indentified in the presence of definite semen abnormality, the 43.3% (10 out of 23) of cases no itological factors were indentified and were put into the category of "No demostarable cause" of the WHO. This figure in WHO study was 49% from the developed countary; 46% from Africa 53% from Mumbai. A lower % in our study could'nt necessarily meant that we were able to indentify etological factor in a comparatively higher % of patients. The difference were more likely possible because our
criteria were much less strict and final diagnosis was made in lesser number of cases; e.g. in many of our cases the diagnosis was presumptive, but this does reflect the fact that world over male infertility is much less understand problem and in a majority of cases the exact etiology remains unclear even after comprehensive testing.

We didn't find antisperm antibodies in the serum or semen of any of our subjects this was because we have used a much less sensitive biological method. Newer method like immunobead test, act to detect secretory antispermtozool Ig A; SPM test, SCMC test, various investigators have found antisperm antibodies in fair number of people.

Coming to infertile couple in which male showed a normal semen examination, many aspects emerged. In general the second semen sample was better than first. FH comhaires et al working on the behalf of the WHO task force has found similar results.

We think that one reason for this was probably patient education it has already been mentioned that abstience increase sperm count also we observe that the patient were much confident in giving sample for the second time than the first. Thus the second sample probably was collected in more efficient way, without loss of much semen and with volume. All these factors could have caused these difference in semen quality.

Another very important aspect we want to high light is the importance of evolving atleast 2 and if need arise more than 2 semen samples. This is stressed by the fact 20% of our patient at same time gave a sample which was categereased when compared to any other sample given by him. WHO also recommends at least 2 semen samples to be examined.

In our study the average sperm count of persons whose semen was categorized as normal was $72.6 \pm 6.2$ millions/ml.
This low sperm of tight langots and dhotis, high incidence of smoking and tobacco chewing habits, general malnutrition and high incidence of exposure to chemical pesticides in this predominately rural farming society.

However it was not possible to compare the incidence of these habits and exposure among persons showing semen abnormality and those not having normal sperm count because of small size of our study.

Next in line would be social factors. Although because being abstract we could not measure these factors but it was clear that male, specially the uneducated ones are very hesitant to present themselves for investigation before their wives. Only when no abnormality was found in wives would some of them volunteer for investigation. But it was also observed that education was important. After taking these males into confidence, follow up was not difficult. We even had few instances when male of an infertile union married again and only when this union was barren, he consented to be investigated, but even these case are amenable to patient education and social support.

And finally the most important aspect of a diagnostic work up of a infertile couples would be to offer them fruitful treatment. Idiopathic testicular failure 5.8% is untreatable. These would have to be offered donor insemination or adoption. Donor insemination is now popular and has reasonable pregancy rates of 10-15% per month over the first 6 months. Approximately 50% of women are pregnant by six months.

However, latest advanced techniques may be offered to these patients also. Silber S.J. has reported 4 successful pregnancies out of 38 in such patients by testicular sperm extraction and ICSI. This technique can also be offered to other types of
azoospermia Testicular atrophy by mumps also falls in the category.

Male genital tract obstructions and male accessory gland infections are potentially treatable by vasoepididomostomy, implantation of spermatoceles, microepididymal sperm aspirations, ICSI, MESA, TESE and IVF can be offered to these patients. The last 2 procedures have better success rate than the initial 2. Thus 9.8% of our infertile males are potentially treatable. Many of our cases in which no demonstratable cause was found could ultimately land in this category. However with new surgical advances repair procedures are also giving good results.

In our study all of the men seen for infertility had abnormal semen quality with etiology identifiable in a few cases only.